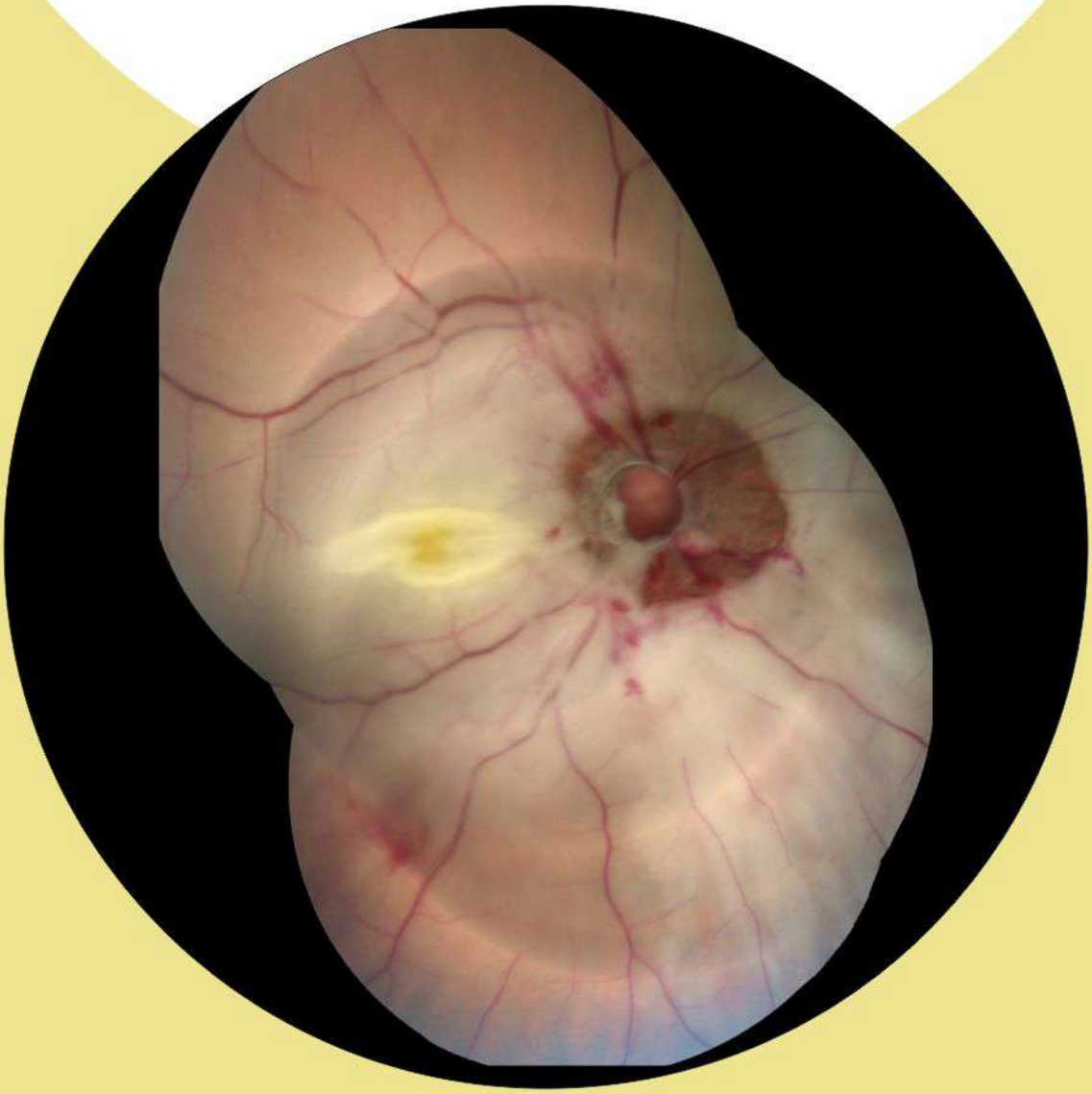
RJO 2022

RAJASTHAN JOURNAL OF OPHTHALMOLOGY



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President's Message

Dear ROS Members,

Ophthalmology is advancing very rapidly with new technology knocking at our doors every day. Cataract surgery has now become a "refractive cataract surgery" and with the advent of premium intraocular lenses, our patients are enjoying excellent visual outcomes. With advancing technology and techniques, it is sometimes difficult to decide what should be the preferred practice patterns. Rajasthan Journal of Ophthalmology (RJO) is playing a vital role in disseminating preferred practice patterns through current ophthalmology articles, case reports, clinical study articles, and practice management articles contributed by the ROS members as well as by the other prominent ophthalmologists of India. I congratulate and thanks the editor, Dr. Suresh K. Pandey, for bringing out an excellent issue of RJO 2022.

The 44th annual conference of the Rajasthan Ophthalmological society (ROSCON 2022) will be held at Kota and the organizing team has left no stone unturned to make it a grand success. The ROS executive committee and the entire local organizing committee have put in all efforts to make an excellent academic and cultural program. I congratulate all of them for their fantastic work and wish for a very successful ROSCON 2022 conference.

Dr. K. K. Kanjolia
President, Rajasthan Ophthalmological Society (ROS)

Secretary's Message

Dear ROS Members,

I take this opportunity to invite you all to the grand academic extravaganza of the 44th Annual ROS Conference (ROSCON 2022) scheduled to be held on 14"-16th October 2022 at Hotel Menaal residency, Kota. The Editorial team under the leadership of editor Dr. Suresh K. Pandey has brought out an extremely useful issue of the Rajasthan Journal of Ophthalmology (RJO 2022). You shall find an interesting amalgamation of practically useful academic articles, case reports as well as ophthalmic practice pearls. I sincerely thank all contributors for taking the time to contribute to the RJO.

Looking forward to seeing you during the ROSCON 2022.

Dr. Sandeep Vijay
Secretary, Rajasthan Ophthalmological Society (ROS)

Dr. Suresh K. Pandey
Editor Journal & Proceedings

Editor's Desk

Dear ROS Members,

I would like to express my heartfelt thanks to all of you for giving me the opportunity to serve as an Editor Journal of our prestigious Rajasthan Ophthalmological Society (ROS).

It gives me immense pleasure to present *RJO 2022*. This issue contains several articles useful in our day-to-day ophthalmology practice. A galaxy of renowned ophthalmologists have shared case reports, research studies, articles on mucormycosis, phacoemulsification in presence of anterior capsule tear, femto-second assisted phacoemulsification for management of posterior polar cataract surgery, retinal vasculitis and cysticercosis, etc. In addition to several clinical studies, several interesting case reports, tips to minimize consumer cases and secrets to become a perfect surgeon are also shared.

I sincerely thank all the esteemed authors for submitting their valuable articles for publication in RJO and also all other authors whose articles could not be added to this issue due to space constraints. I would like to encourage them to send these for the next issue.

My sincere thanks to editorial board members for their support and encouragement. The PDF version of this issue will also be made available to all members in their inboxes and also on the website of the ROS (https://rosonline.in/).

I look forward to receiving your valuable feedback and suggestions.

Wishing you a great scientific feast.

Suresh K Pandey

Dr. Suresh K. Pandey
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CURRENT OPHTHALMOLOGY

Conservative Management by Transcutaneous Amphotericin B in Rhino-Orbital-Cerebral-Mucormycosis

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Email:santosh.honavar@gmail.com

Introduction

The SARS CoV-2 pandemic led to a surge in cases of rhino-orbital-cerebral mucormycosis (ROCM) as seen over the last two years. The overwhelming increase in the number of cases required a detailed understanding of the risk-factors, staging and management strategies based on the baseline staging[1]. The management for the orbital involvement has ranged from conservative interventions like trans-cutaneous Amphotericin-B (TRAMB) to radical surgical interventions like orbital exenteration to optimize life, eye and vision salvage based on severity.[2–4]

Local irrigation of the affected area following surgical debridement has been successfully practiced by few authors.[5,6] Irrigation with intra-venous cannula, catheters, soaked cottonoid pledgets and fenestrated drains have been reported to give good outcomes. This is to provide local delivery of drug to minimize systemic side-effects.[7–10]

Local injection of Amphotericin-B (AMB) in retro-bulbar space via transcutaneous approach has also been advised with various formulations of AMB. Considering the psycho-social aspects and rehabilitation issues related to radical procedures like orbital exenteration, it is important for the treating ophthalmologists to be aware of the correct procedure and importance of conservative approaches like TRAMB.

Stages of ROCM with orbital involvement

In ROCM cases, disease progression occurs to involve structures from the orbit to intra-cranial structures via contiguous and non-contiguous routes. Accurate

staging of the disease and management is possible with the contribution of a multi-disciplinary team of experts i.e. radiologists, microbiologists, pathologists, infectious disease specialists, neurologists, critical care experts, otorhinolaryngologists , ophthalmologists as well as neuro-surgeons. Therefore, the disease extent must be identified duly by clinic-radiological assessment before planning the steps of management. Diagnostic nasal endoscopy, contrast-enhanced MRI or CT are preferred in these patients.[2] Orbital Mucormycosis cases belong to the stage 3 and stage 4.[1]

Stage 3: Involvement of orbit

In this stage, the patients present with symptoms like pain, forward protrusion of the eye, drooping of eyelids, loss of vision, double vision, decreased sensation over the face. On examination, signs include conjunctival chemosis, isolated ocular motility restriction, ptosis, proptosis, infra-orbital nerve anaesthesia, central retinal artery occlusion and superior ophthalmic vein occlusion. Features of V1, V2 anaesthesia and III, IV and VI nerve palsy might indicate the presence of orbital apex involvement/superior orbital fissure involvement.

Stage 3a: As showed in figure 1a, stage 3a includes localized orbital involvement i.e. involvement of the nasolacrimal duct and medial orbit, with unaffected vision.

Stage 3b: As shown in figure 1b, stage 3b includes diffuse orbital involvement i.e. >1 quadrant or >2 structures, and unaffected vision.

Stage 3c: Figure 1c describes stage 3c, wherein there is central retinal artery or ophthalmic artery occlusion or superior ophthalmic vein thrombosis; involvement of superior orbital fissure, inferior orbital fissure, orbital apex and loss of vision.

Stage 3d: Figure 1d showed bilateral orbital involvement in stage 3d

Stage 4: Extensive progression with central nervous system involvement.

In this stage, along with the above-mentioned symptoms and signs, the patient might present with bilateral proptosis, paralysis/hemiparesis, altered consciousness or focal seizures indicating brain invasion and infarction.

Stage 4a: Focal or partial cavernous sinus involvement and/or cribriform plate involvement

Stage 4b: Diffuse cavernous sinus involvement and/or presence of cavernous since thrombosis

Stage 4c: Involvement goes beyond the cavernous sinus with involved skull base, internal carotid artery occlusion and even possible brain infarction.

Stage 4d: This stage shows multifocal or diffuse CNS involvement

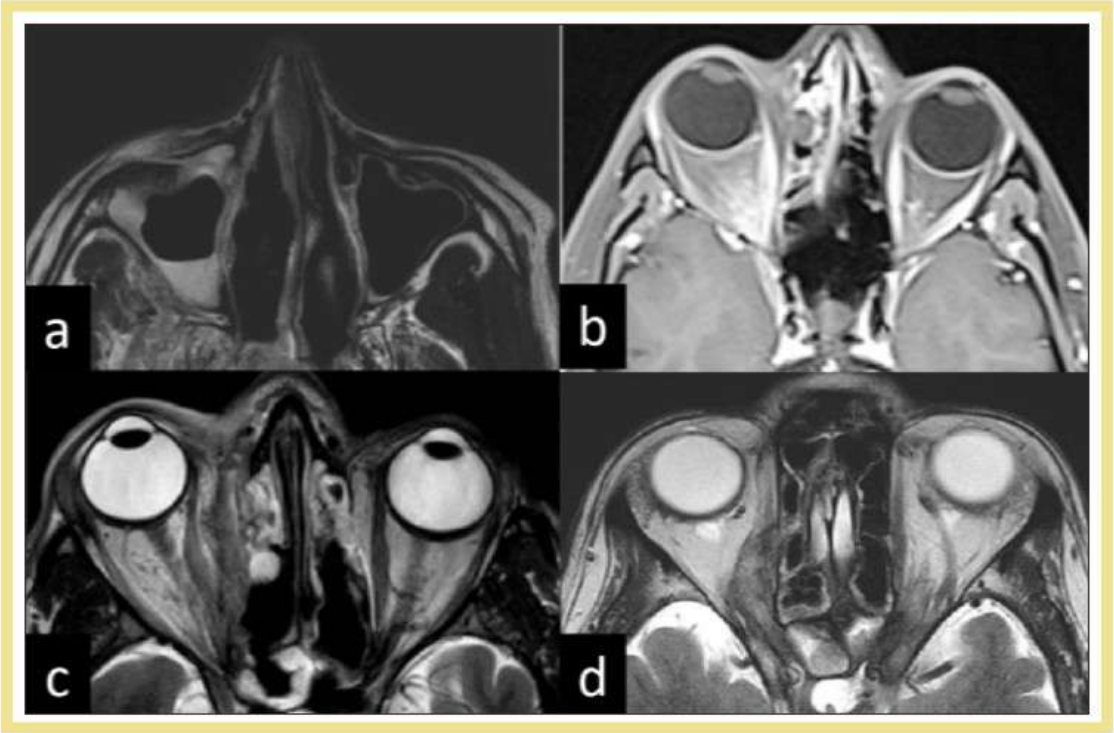

Figure 1: MRI showing various stages of ROCM. Fig 1a: Stage 3a – Axial T2 weighted MR showing involvement of the right nasolacrimal duct (red arrow), extending into the medial orbit; Fig 1b: Stage 3b - Axial contrast-enhanced MRI (T1) of the orbit, par nasal sinuses, and brain showing involvement of the medial orbit and abnormal intensity of the orbital fat in the posterior orbit along with involvement of the right ethnocide sinus; Fig 1c: Stage 3c - Axial MRI (T2) showing right ethnocide sinus and diffuse orbital involvement extending to the orbital apex; Fig 1d: Stage 3d - Axial MRI (T2) of the orbit and par nasal sinuses showing bilateral orbital apical involvement, more extensive on the right side.(*Courtesy – Sen et al*[2]).

Patients belonging to stage 3a, 3b can be safely managed with conservative approach, however, the cases in the grey zone (Fig 2) i.e. stage 3c, 3d should be assessed carefully to categorize for conservative versus radical management.[11] Stage 4 cases should preferably be attended to with radical approach only, considering the aggressive nature of the disease and possibility of intra-cranial involvement.

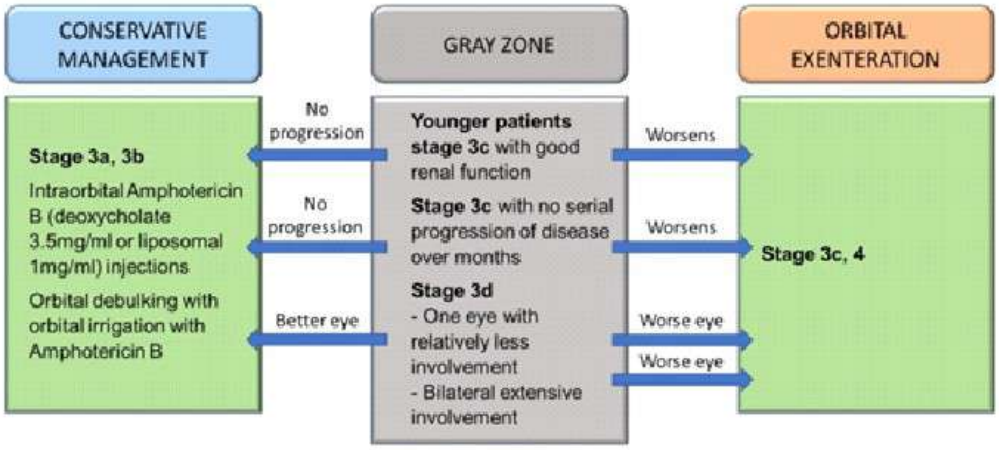

Figure 2: Stage-wise management of orbital mucormycosis (*Courtesy – Honavar et al*[11]).

Transcutaneous retro bulbar amphotericin - B injection

Over the years, amphotericin B has been used intra-venously to treat fungal infections. The usage has definitely increased with the surge in ROCM cases. However, the off-label use of Amphotericin B has been adopted in treatment of orbital components via the transcutaneous retro-orbital route.[11–14]

Drug formulations

Amphotericin B comes in three formulations –

1. L-AMB : Liposomal amphotericin B
2. ABCD: Amphotericin B colloidal dispersion
3. AMB-D: Amphotericin B deoxycholate

With the limitation that no randomized controlled trial is available for the different formulations of amphotericin B used for TRAMB, various authors have worked on L-AMB and AMB-D.[7,12,15,16]

Mechanism of action

It has been postulated that the Rhizopus spores and germ tubes damage the human endothelial cells by adhering to the cells and causing phagocytosis which leads to the injury.[17] AMB binds to ergosterol of the fungal cell membranes and forms pores that lead to electrolyte leakage, especially the monovalent ions which leads to fungal cell death. AMB also has high protein binding and large molecular weight due to which it diffuses slowly into the tissues.[18]

Dosage

To begin with, there were speculations on usage of variable dosages for TRAMB formulations ranging from 1mg/ml (Fig 3) to 3.5mg/ml. However, several authors have shown excellent results with symmetric dosing of injecting 1ml of 3.5mg/ml L-AMB or AMB-D which has provided a basis for safe concentration.[7,12] However, it must be noted that randomized controlled trials have not been carried out to be able to confirm the ideal concentration and the articles published till date do not have a significant follow-up to comment on the same.[19]

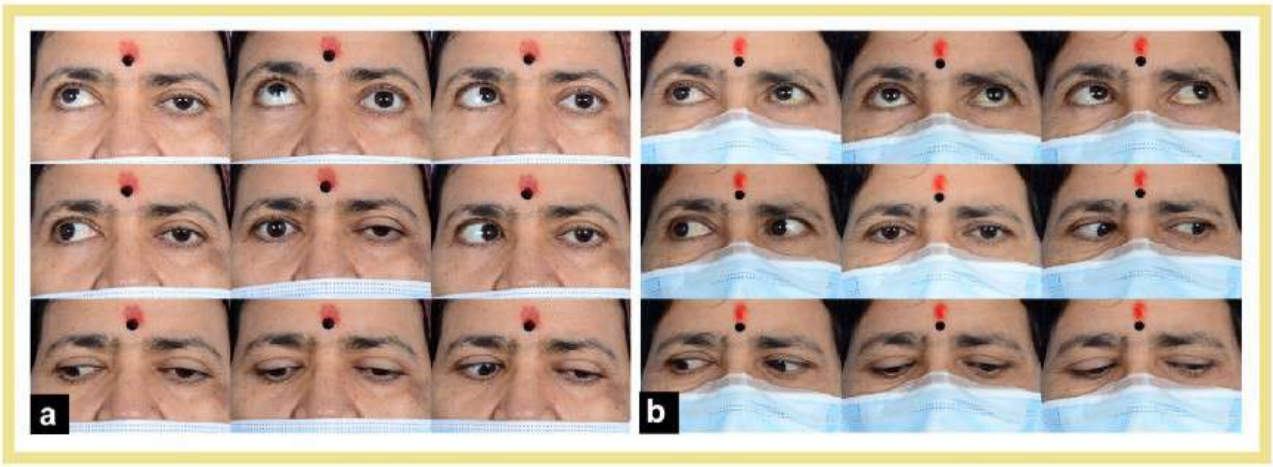

Figure 3a: A 56-year-old-lady presented to us with left eye BCVA of light perception only, ptosis of 3mm, restriction of movements in all directions with radiological evidence of involvement in the supero-medial and infero-medial aspect. She was diagnosed to have stage 3b ROCM. Figure 3b: After a series of 7 injections of trans-cutaneous retro-orbital Amphotericin B (1mg/ml), she had complete resolution of symptoms and signs along with BCVA of 20/40 at the latest follow-up 5 months and interval decrease in the medial orbital involvement on MRI. (*Courtesy – Honavar et al*[11])

Site of injection

The site of injection is chosen as per clinico-radiological assessment. The most extensively involved area is ideally chosen for TRAMB.[12,13]

Procedure

This can be performed as an out-patient procedure under aseptic precautions. The drug comes in powder form (50mg) and requires to be reconstituted by sterile water. Saline should not be used for formulation to avoid precipitation induced by sodium chloride. Lidocaine 2%, 1ml – 3ml, is injected at the ideal site. Five minutes following the anaesthesia, 1ml of 3.5mg/ml L-AMB or AMB-

D preparation is injected with a 26-gauge needle and gentle pressure is applied along with tight eye patching for 3-4 hours.

Interval and number of injections

Variable intervals have been followed by the authors who have worked on TRAMB and no definite interval has been defined. Injections have been given as per the clinical and radiological findings.[12] Bay ram et al have given a mean of 2.2+0.6 L-AMB injections in their eleven reported cases. Sharifi et al reported injections to be given for 3 doses daily or on alternate days.[20]

Adverse reactions

Considering the off-label usage of TRAMB, the patients are observed closely for the development of adverse effects and are duly informed about the same.
Mild and moderate adverse effects include eyelid edema, chemosis (yellow-tinged) and congestion which resolve spontaneously after stopping the injections within 4-6 weeks. Severe adverse effects include orbital congestion due to involvement of extraocular muscles, orbital compartment syndrome, visual deterioration which might require immediate intervention with canthotomy and cantholysis.[7,13,20]

Conclusion

The COVID-19 pandemic and its consequences affected patients psychologically as well as financially. A multi-disciplinary approach is recommended for monitoring disease progression. As per the stage-based management, an economical, effective, conservative approach involving TRAMB can be adopted for stage 3a and stage 3b. Whereas, radical approach like orbital exenteration can be reserved for stage 3c, stage 3d and stage 4 or the cases refractory to TRAMB. A well-planned and well-executed conservative approach can lead to a holistic and wise management of ROCM patients keeping in mind the psychological, social, cosmetic and economical aspects.

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CURRENT OPHTHALMOLOGY

Cysticercosis

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Introduction

This article summarizes the manifestations caused by cysticercosis which may affect multiple organ systems and hence often requires a multidisciplinary approach. Fundus visualisation of cysticercus constitutes an absolute criterion for its diagnosis hence an ophthalmic consult is mandatory in all systemic manifestations be it dermal, cardiac, lung, neurological or any other manifestations of cysticercosis. Similarly an ophthalmologist must send appropriate referrals to rule out involvement of any other organ system. Cysticercosis is a parasitic infection caused by the larval form of tapeworm of genus Taenia (Cysticercosis cellulosae).

It can cause two different types of human diseases : Taeniasis and Cysticercosis[1]. These are classified as "cyclo-zoonoses" as they require more than one vertebrate host species (but no invertebrate host) to complete their developmental cycles[2].

Background

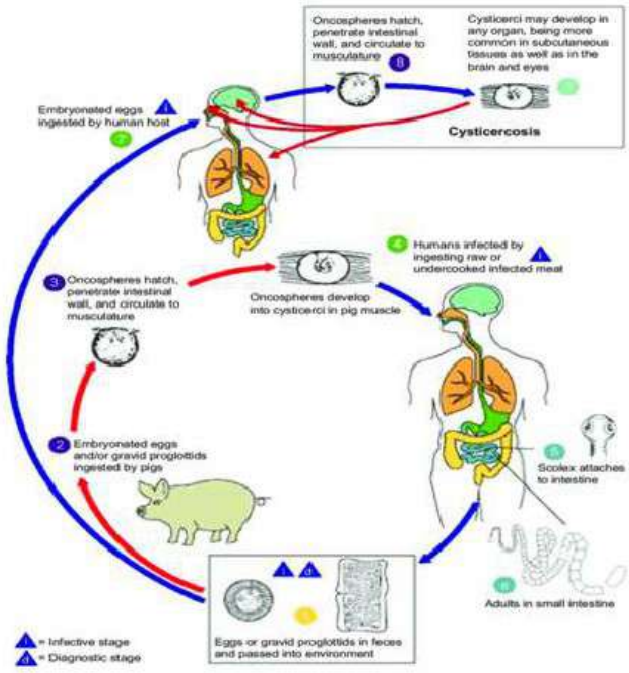
Taeniasis is an intestinal infection caused by adult forms of Taenia solium and Taenia saginata . Humans are the only definitive hosts for Taenia solium[1]. The larval stage of T. solium mainly occurs in pig but man may also be affected. Cattle is the main host for the larval stage of T. saginata. Such infections occur where sanitary conditions are poor and with the routine consumption of raw or undercooked contaminated pork and beef.

Epidemiology

This disease affects around 2.5 million people who carry T. solium and 20 million are infected with cysticerci. Endemic foci are South and Central America, sub-Saharan Africa and Southeast Asia where up to 25% are seropositive and 10-18% have CT findings suggestive of neurocysticercosis[3].

Lifecycle

consumption of raw or poorly cooked meat infested with the larval form (cysticerci) leads to taeniasis. These larvae attach to the host intestinal mucosa by their scolex and develop into adult worms (3-9m) in the intestinal lumen.. The incubation period for adult tapeworm is 8-14 weeks. Adult worms produce as many as 1000 proglottids (T. solium, 12*5 mm) which become gravid. They detach from the tapeworm and migrate to the anus. They are shed in the feces in groups of 3-5 and contain 50,000 to 100,000 viable eggs[4]. Ingestion of eggs by intermediate hosts (pigs, cattle, or humans) results in hatching of the eggs into larvae (5-10 mm, with a scolex) called oncospheres which penetrate through the intestinal wall. Autoinfection may occur in humans if proglottids pass from the intestine to the stomach via reverse peristalsis. The larvae are transmitted through the lymphatic and circulatory systems, where they invade the striated muscles of the neck, tongue and trunk, various other organs and develop into cysticerci (infectious form). Humans develop cysticercosis via ingestion of *T. solium* eggs, either from exogenous sources or from their own stools. Only larvae of *T. solium* penetrate the human intestine; *T. saginata* does not cause human cysticercosis because the larvae cannot penetrate the intestinal wall[1].



Life cycle of Taenia solium[8]

https://www.researchgate.net/figure/Life-cycle-of-Taenia-solium-cysticerci-Note-Reproduced-from-Centers-for-Disease-Control_fig1_283493022

The impact of tapeworm infection in man is difficult to quantify because in vast majority of cases, they do not lead to clinical ill health, except occasional abdominal discomfort, anorexia and chronic indigestion
Straying of proglottids may sporadically cause appendicitis or cholangitis. The most serious risk of T. solium infection is cysticercosis[2].

Categories of criteria	Criteria
Absolute	1. Histologic demonstration of the parasite from biopsy of a brain or spinal cord lesion
	2. Cystic lesions showing the scolex on CT or MRI
	3. Direct visualization of subretinal parasites by funduscopic examination
Major	1. Lesions highly suggestive of neurocysticercosis on neuroimaging studies
	2. Positive serum EITB for the detection of anticysticercal antibodies
	3. Resolution of intracranial cystic lesions after therapy with albendazole or praziquantel
	4. Spontaneous resolution of small single enhancing lesions
Minor	1. Lesions compatible with neurocysticercosis on neuroimaging studies
	2. Clinical manifestations suggestive of neurocysticercosis
	3. Positive CSF ELISA for detection of anticysticercal antibodies or cysticercal antigens
	4. Cysticercosis outside the CNS
Epidemiologic	1. Evidence of a household contact with Taenia solium infection
	2. Individuals coming from or living in an area where cysticercosis is endemic
	3. History of frequent travel to disease-endemic areas

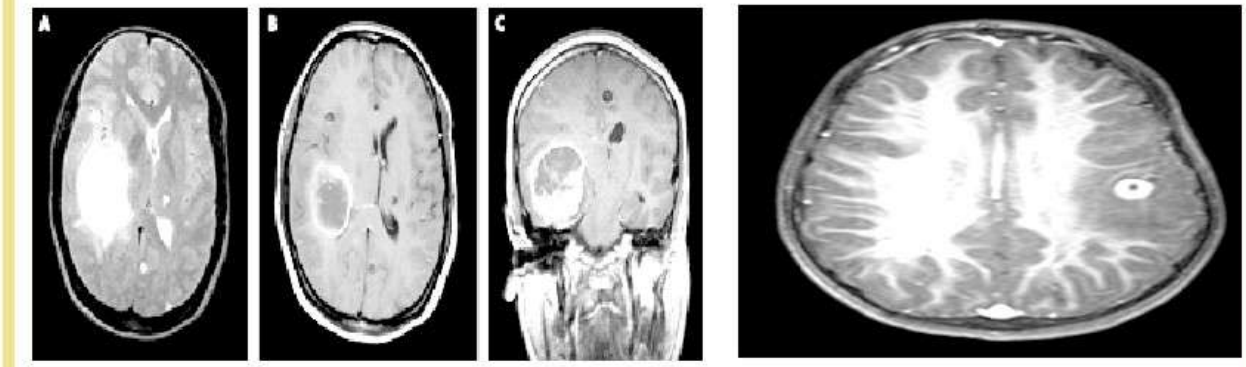
Definitive diagnosis
1. Presence of one absolute criterion
2. Presence of two major plus one minor and one epidemiologic criteria

Probable diagnosis
1. Presence of one major plus two minor criteria
2. Presence of one major plus one minor and one epidemiologic criteria
3. Presence of three minor plus one epidemiologic criteria

Diagnostic Criteria14
Harrison's principles of internal medicine; edition 17th, chapter 213 infectious diseases, page 1337

Ophthalmologists should be aware of the myriad symptoms and presenting signs of Cysticercosis which is a pleomorphic diseases[9]. Cysticerci can be found anywhere in the body but most commonly detected in the brain, cerebrospinal fluid, skeletal muscle, subcutaneous tissue and eye. A multimodal approach may be needed to detect the full extent of the disease. Ophthalmic consult with dilated fundus examination is of paramount importance when any physician encounters a possible cysticercus case scenario. However cysticercosis of the eye may be a presenting feature in a patient and in such cases the experienced ophthalmologist must rule out systemic findings suggestive of cysticercosis. Physician opinion should be taken if the ophthalmologist suspects obvious cutaneous, neurological or respiratory involvement. Opinion of neurologist is invaluable if patient is otherwise asymptomatic and only ocular involvement is suspected.

The clinical presentation depends on a complex range of interconnecting factors such as the location and extent of cysticerci as well as the extent of associated inflammatory responses or scarring. It is thought that the viable cysts initiate a complex immune evasion response, allowing them to exist undetected in the body. This may persist for a prolonged period of time, with immune-mediated symptoms sometimes being delayed for several, or as many as 10 years. Development of symptoms is generally associated with the immune system overcoming such evasion mechanisms and initiating subsequent immune responses against the degenerating cysts, leading to systematic effects and a corresponding clinical profile.[10]



Ring enhancing lesion in Neurocycticercosis[15]
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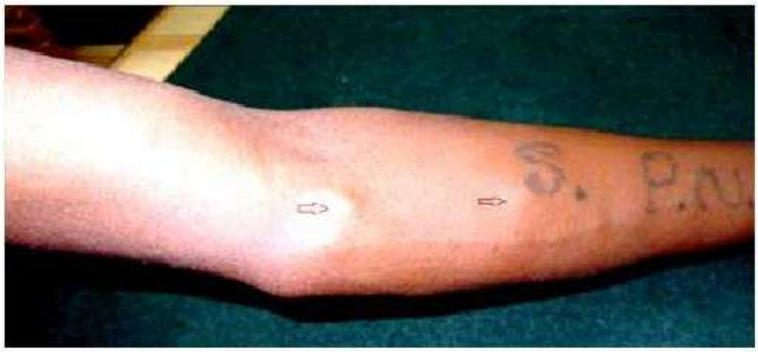

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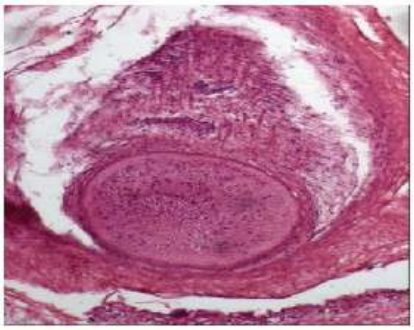
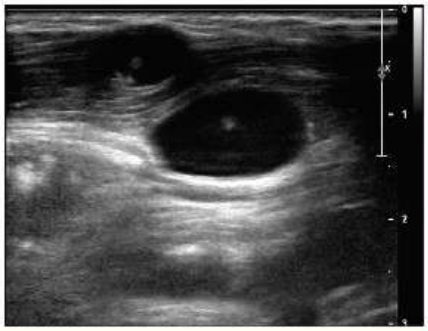


Histology[20]



USG[21]

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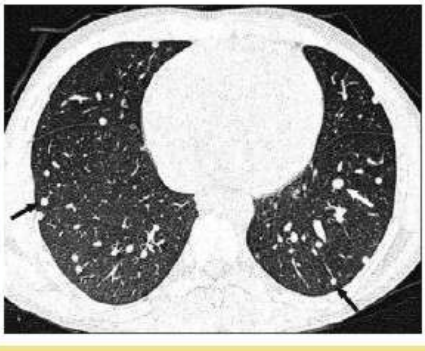

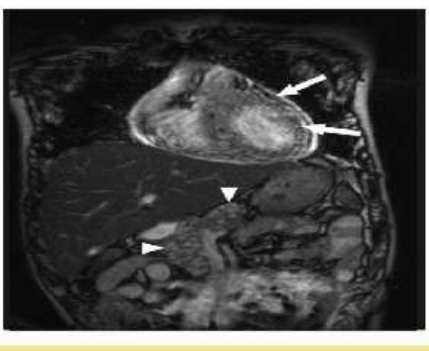

nodular lesions seen in the cardiac muscle (arrows) and pancreas (arrowheads)[27]https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3056633/

HRCT shows nodular lesions in the lungs[28]

Ophthalmological

Cysticercosis is a preventable cause of blindness endemic in India. It is a parasitic infestation caused by Cysticercus cellulosae, which is the larval form of Taenia solium. In 1829, Soemmering reported first case of a live anterior chamber cysticercosis.Ocular cysticercosis has a varied presentation depending upon the site of involvement, number of lesion and the host immune response. In contrast to Western literature, Indian studies have reported ocular adnexa as the most common site of involvement. While the most common site of localization reported in Western studies is the posterior segment, in the Indian literature the ocular adnexa is the most common site. In a study reported by Kruger-Leite et al, 35% of the cysts were found in the subretinal space, 22% in the vitreous, 22% in the subconjunctival space, 5% in the anterior segment, and only 1% in the orbit.

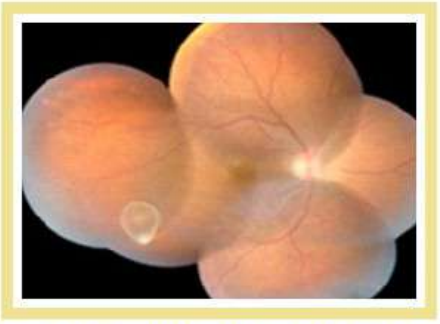

Figure 1. Intravitreal free floating cyst caught on montage.

Figure 2. Cyst with retinal detachment.

Figure 4. Multiple cysts.

Figure 3. Kidney bean shaped cyst.

Figure 6. Large cyst with protruding baby cyst.

Figure 5. Subretinal cyst.

Ocular manifestation, usually unilateral, but occasionally bilateral in disseminated cases, have been reported in 13-46% of patients, most often children and young adults [22]. Both extra ocular (orbit or extra ocular muscles) and intraocular disease has been reported. As per literature, intraocular presentations are more frequent in the western world, whereas extra-ocular presentations are more frequent in the endemic countries. Most commonly involved are the extra ocular muscles, the subconjunctival space, anterior chamber, vitreous cavity, subretinal space, eyelid, optic nerve, retro-orbital space and lacrimal gland. Orbital involvement may present with proptosis, ptosis or restriction of movement with resultant diplopia (it can also be caused by cranial nerve palsies from neurocysticercosis). In a study by Rath S et al the 3 main symptoms at presentation were periocular swelling (38%), proptosis (24%), and ptosis (14%) with a median duration of 2 (range 0-24) months. The 3 main signs at presentation included ocular motility restriction (64.3%), proptosis (44.4%), and diplopia (36.8%). The cyst locations in the decreasing order of frequency were anterior orbit (69%), subconjunctival space (24.6%), posterior orbit (5.8%), and the eyelid (0.6%). In all, 80.7% of patients had cysts in relation to an extra ocular muscle. The superior rectus (33.3%) was the most commonly involved extra ocular muscle. Contact B-scan ultrasonography was diagnostic of cysticercosis in 84.4% of patients. Orbital cysticercosis was managed medically in 158 of 166 patients.

Optic atrophy, papilledema, nystagmus, pupillary changes, orbital inflammation, periorbital pain, loss of vision, scotoma and photopsia may also be seen. Any extra ocular muscle can be involved but the least frequently involved is the inferior oblique. Muscle involvement may present as Brown syndrome or inverse Duane's retraction syndrome, and may even mimic orbital cellulitis or idiopathic orbital inflammation, especially if the muscles are diffusely enlarged (in contrast to the more common cystic or nodular swelling demonstrated on imaging)[22]. Depending on their location, some cases of extra ocular muscle involvement can indent the globe externally, simulating the appearance of retinal detachment. Intraocular disease presents as anterior chamber, intravitreal, subretinal cysts or vitritis, retina; vasculitis, macular edema, scarring, exudative retinal detachment or chorio-retinitis. Anterior chamber cysts with concomitant hypopyon, various forms of uveitis, cataract and secondary glaucoma have also been reported. The release of antigens after intraocular larval death or degeneration initiates a profound inflammatory response, which can lead to endophthalmitis, making the surgical removal of intraocular larvae or cysts mandatory[22].

Ophthalmic Cysticercosis[29]
https://image.slidesharecdn.com/taeniaspp-150420155140-conversion-gate02/95/taenia-spp-19-638.jpg?cb=1429563220

We include intraoperative pictures of intravitreal cysticerci cases operated at SMS, medical college, Jaipur. As per our experience timely intervention in these cases can preserve visual function.

A recent case report by Karthikeya et al[24] in the BMJ shows migration of cysticercus causing Full thickness macular hole. In the ocular tissues, macula has been noted to be the preferred site for the lodgement of the blood borne cysticercus possibly due to the rich blood supply. From the macular subretinal space it can enter the vitreous cavity through a break in the overlying neurosensory retina. When this migration occurs, the defect in the retina thus formed can give rise to rhegmatogenous retinal detachment, or more commonly this site heals with a scar due to the inflammation associated with the cysticercus migration and leads to an area of scarring in the retina. Sharma et al have hypothesized an alternate route of entry for cysticercus into the vitreous cavity based on their observation in four cases of intravitreal cysticercosis and no retinal lesion—through a retinal blood vessel, optic disc or ciliary body. George *et al* have also reported an intravitreal cysticercosis with an associated retinal break.[25]

Diagnosis

Diagnosis is usually made using a combination of imaging studies such as CT or MRI and Ultrasound Imaging which typically reveals cystic lesions with or without high reflective spikes (A-scan) or spots (B-scan) which suggests the presence of the scolex signifying a viable cyst as opposed to a degenerating or a calcified one, CT or MRI can also show the presence of scolices and this is considered pathognomic of cysticercosis. Serologic testing such as ELISA and the newer western blot or enzyme-linked immuno electro transfer blot assay (EITB) are reported as 80-100% sensitive in neurocysticercosis, but in isolated intraocular or extra ocular cases, or an otherwise low burden of infection, their yield is much lower. False-negative results can occur as well as false-positive results from cross-reactions with other helminthes depending on the selected cut-off point. Therefore, a positive result helps to confirm the diagnosis but a negative result does not rule out the diagnosis. Stool microscopy is done by the

demonstration of eggs or gravid proglottides in stool by direct fecal smear, Brine floatation technique and Cellophane tape technique. Acid fast stain is used to distinguish the eggs, T. saginata is acid fast positive (red) while T. solium is negative (blue)[29]. Histology shows body of the cysticercus larvae with multiple papillary infoldings and scolex with suckers and hooklets.
Cysticercus cellulosae mainly has three stages of evolution. The live or vesicular cyst is the living cyst with a well-defined scolex. It causes minimal or no inflammation in the tissue. As larva begins to die the cyst wall becomes leaky, releasing toxins and causing varying degrees of inflammation. This is the colloidal vesicular stage. Eventually, the larvae die and are either totally resorbed or calcified. This is the calcified nodular stage. Symptoms will be affected by the stage of cyst as well.

Treatment

Treatment options include albendazole or praziquantel but corticosteroids are always employed to limit host inflammatory responses from the death of the cestodes. Some patients may experience a relapse of symptoms after initial therapy with one agent either from inadequate duration or dosage or resistance, but almost always responds well to the other agent. Albendazole has been reported to have an 80% elimination rate compared with 67% for praziquantel, which is also more expensive. 10-15 mg/kg/day of Albendazole is given twice daily with a fatty meal for 7 to 14 days but a longer course (up to 28 days) is advisable at present, it can be repeated as necessary. Upton 3 months of treatment may be needed for ventricular and subarachnoid cysts. Praziquantel is given in 50 mg/kg/day in 3 divided doses for 15 days[2].
Recent reports have shown excellent preservation of vision with modern vitreoretinal surgical techniques.

Pars Plana Vitrectomy

23G pars plana vitrectomy, posterior vitreous detachment (PVD) induction, in vivo cyst lyses and aspiration, and brilliant blue G-assisted internal limiting membrane (ILM) peeling with inverted ILM flap over macular hole with 25% SF6 gas injection [30] is the standard of care now-a-days.

Extra ocular Involvement : Residual deficits such as proptosis, ptosis or motility restriction may persist despite treatment in some cases of orbital involvement. It has been suggested that this maybe from fibrosis occurring as a consequence of chronic inflammation in long-standing cases. Early surgical removal of intraocular cysts is the treatment of choice. Cysts may also be extruded spontaneously within 3 to 5 days of starting medical therapy.

For any physician treating cysticercus ,it is prudent to always exclude ocular involvement before initiating medical therapy, as the resulting inflammation may have devastating visual consequences for the patient if the dying ocular larvae are not surgically removed.

Prevention

The prevention of cysticercosis involves minimizing the opportunities for ingestion of fecally derived eggs by means of good personal hygiene, effective fecal disposal and treatment and prevention of human intestinal infections. In many countries, T. solium has been controlled by meat inspection, proper housing and feeding of pigs.[2] Thorough cooking of beef and pork is the most effective method to prevent food-borne infections. Education of the public to prevent pollution of soil, water and food with human feces and washing of hands before eating and after defecation and are important health education messages. Mass chemotherapy has been administered and newer vaccines to prevent porcine cysticercosis are under development.[4]

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CURRENT OPHTHALMOLOGY

Optical Coherence Tomography: A New Approach in Glaucoma

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Abstract

Glaucoma is a chronic progressive disease which increase pressure in eye and damage the optic nerve and it can lead to blindness. Early detection is crucial to prevent further damage. Though glaucoma is more a clinical diagnosis but new approaches like optical coherence tomography can help us diagnose suspicious cupping and early glaucoma. The newer modalities help us to prognosis the disease by seeing for progression. This article will help OCT use in glaucoma with better understanding.

Key words: Glaucoma evaluation, optical coherence tomography

Introduction

Glaucoma is a progressive disease which can damage eye optic nerve. It's often linked to a build-up of pressure inside the eye and damage optic nerve, which sends image to the brain and its lead to blindness. Early detection is crucial to prevent further damage. 30-40% of ganglion cell loss happens before it can be detected on field test.[1] Thinning of the neuro-retinal rim and RNFL loss predicts glaucoma damage.[2] To document disease progression requires both structural and functional assessment.[3] Imaging techniques are objective and allows quantitative measurement. There are different modalities to image RNFL including OCT, HRT and SLP. This article describes interpretation of the OCT. It also discusses the limitations of OCT and artifacts affecting image quality.

Interpretation of Optical Coherence Tomography

There are four types of spectral-domain optical coherence tomography (OCT) machine available commercially: Zeiss Cirrus, Heidelberg Spectralis, Optovue Avanti RTVue, and Topcon.

We describe how to interpret the Cirrus HD OCT reports; however, the same principle applies to most devices. Due to differences in the measurement protocols in different machines, OCT machine data should be compared inter changeably.

Cirrus OCT has superior image quality than time-domain OCT due to faster scanning speed and better image resolution.[4]

Steps in assessment are as follows:

1. Type of scan [Table1]

2 Assess the quality of the scan: Signal strength, centration of the disc, OCT image, and artifacts [5] [Figures 9-12]

3 Interpret the printout

a. Age: Important because an inaccurately entered age will result in comparison with the wrong normative data age group. In normal eyes, a 2μm retinal nerve fiber layer (RNFL) loss/decade and 0.2-0.5%/year (0.52μm/year) has been reported.

b. RNFL thickness map: It shows the thickness of the RNFL – it is coded from blue (thin) to white (thickest). It normally shows an hourglass pattern (butterfly). It can give you a gross idea of the RNFL thickness. The superior RNFL and inferior RNFL are the thickest where as the nasal and temporal are less thick. Small optic discs, long axial length, and older age are associated with thinner RNFL. Every 1-mm increase in the axial length is associated with an approximately 2.2μm decrease in RNFL thickness.

c. Look for artifacts in this map. Compare with the other eye [Table 2]; intraocular difference >9 μm is unusual in normal eyes and glaucoma should be ruled out (glaucoma suspect).[6]

Table: 1. Types of scans and its limitation

	Optic nerve head	RNFL analysis	Macular ganglion cell analysis
Scan protocol	Optic disc cube 200×200(4 mm× 4mm)	3.4 mm measurement circle centered on optic disc using radial line scan	Macular cube 512×128 (6 mm×6 mm)
parameter	Disc area , rim area Vertical and horizontal cup disc ratio, Cup volume, average RNFL value RNFL symmetry	Mean RNFL measurement TSNIT graph symmetry, sectoral chart Clock chart	Ganglion cell complex including RNFL, ganglion cells inner plexiform layer
Best parameter for glaucoma detection	Rim area Vertical rim thickness Vertical C:D ratio[5]	Inferior RNFL, Average RNFL[5,6]	Minimum GC-IPL (macula, ganglion cell, inner plexiform layer)[7]
Limitation	Can miss glaucoma in smaller discs	Tilted myopic disc with PPA	Confounded by macular disease
Glaucoma detection	Not well established	Good	Comparable to RNFL Better when done in combination of RNFL Does not consider RGC outside macular areas[7,8]

RNFL: Retinal nerve fiber layer, GC-IPL: Ganglion cell-inner plexiform layer, RGC: Retinal ganglion cells, TSNIT: Temporal, superior, nasal, inferior, temporal graph

Table : 2 Decoding the color code

Colour Code	RNFL analysis (Normative database matched to age)	ONH analysis (Normative data base matched to age and disc area)
Green	Between 5%-95% Within normal limit	Between 5%-95% within normal limit
Yellow	Between 1%-5% border line thinning suspect	Between 1%-5% border line thinning suspect
Red	<1% Outside normal limit(Thinnest)	<1% Outside normal limit (Thinnest for Neuro-retinal rim and largest for CD ratio)
White	>95%(Thickest) Also used when normative data base not available ex: age less than 18	>95% (Thickest for neuro-retinal Rim and smallest for C:D/ratio)
Gray	Not applicable	Used when normative database not applicable • Average or vertical CD ratio is below 0.25 • When disc area is less than 1.3 mm^2 or greater than 2.5 mm^2 rim area • Normative data off

Artifacts in OCT Imaging

IMAGING RELATED:

Poor imaging quality, device malfunction, Motion Artifact, Segmentation Errors

PATIENT RELATED:

Split bundles , shifted RNFL

Media opacities , vitreo-retinal interface problems etc.

Green Disease :

False negatives : A Green OCT that is believed to be normal but may be indicative of another disease or just green as a result good imaging quality:

1.Optic disc pit

2. Hypoplasia

3. Coloboma

4.Early glaucoma.

Red Disease :

False positive : A red OCT that is delivered to be glaucoma but may be indicative of another disease or just red as a result poor imaging quality:

1.Anatomic anomalies:Tilted disc and PPA

2.Media opacities: Reduce image quality

3.ONH masquerades: Optic neuritis, AION etc

4. High myopia:

RNFL deviation map: This compares the clusters of pixels in the test image with the normative database and color codes the areas accordingly. It is important to carefully assess this map because small localized defects may sometimes be seen only on this print-out. Look specifically for the measurement circle and motion artifacts [Figures 1-8].

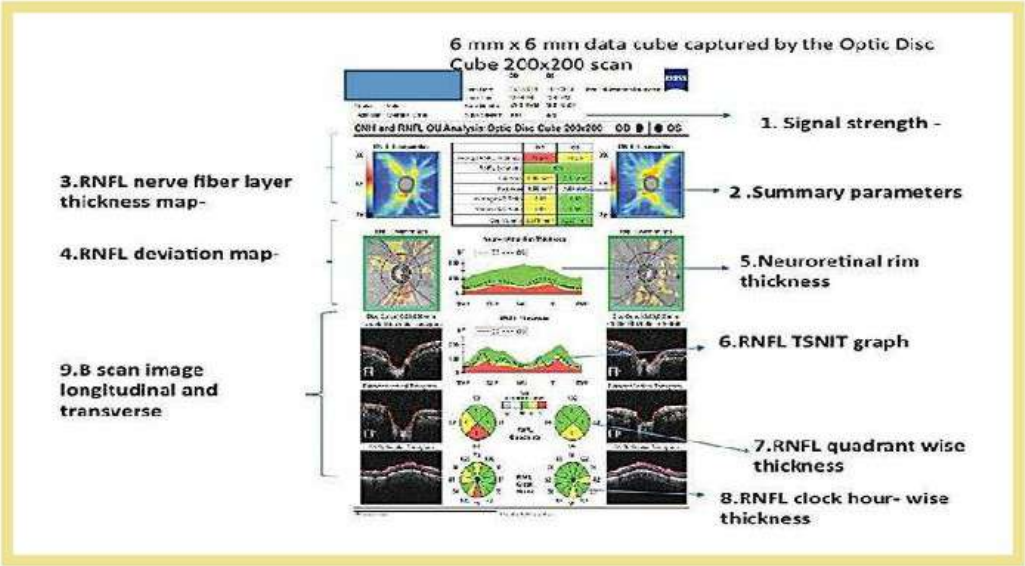

Figure 1: Optic nerve head and retinal nerve fiber layer optical coherence tomography analysis report

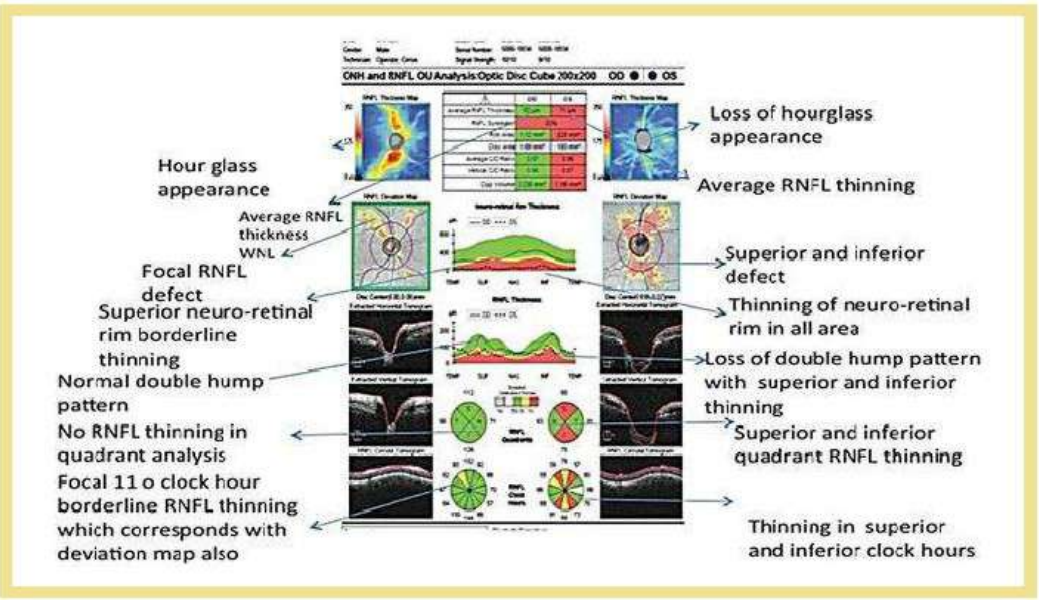

Figure 2: Example of optical coherence tomography interpretation

1. **Signal strength** -It range from 0-10. Anything above 6 is acceptable
2. **Key parameters :** compared to normative database for RNFL analysis and normative database and disc area for disc parameter. Disc area values are always shown in grey as no normative database is available. When disc area is less than 1.3mm² or greater than 2.5 mm² rim area, CD ratio and CD volume will be shown in grey colour . Two eyes have a similar profile when symmetry parameter is close to 100%,.Average RNFL thickness values should be looked for which decreased with severity of glaucoma increases.

	OD	OS
Average RNFL Thickness	103 µm	103 µm
RNFL Symmetry	98%	
Rim Area	1.17 mm²	1.32 mm²
Disc Area	1.95 mm²	2.25 mm²
Average C/D Ratio	0.64	0.63
Vertical C/D Ratio	0.61	0.58
Cup Volume	0.327 mm³	0.364 mm³

	OD	OS
Average RNFL Thickness	50 µm	74 µm
RNFL Symmetry	34%	
Rim Area	0.63 mm²	0.98 mm²
Disc Area	2.26 mm²	2.16 mm²
Average C/D Ratio	0.84	0.72
Vertical C/D Ratio	0.84	0.67
Cup Volume	0.749 mm³	0.421 mm³

	OD	OS
Average RNFL Thickness	59 µm	94 µm
RNFL Symmetry	7	5%
Rim Area	0.64 mm²	1.27 mm²
Disc Area	2.51 mm²	1.27 mm²
Average C/D Ratio	0.89	0.15
Vertical C/D Ratio	0.90	0.13
Cup Volume	1.420 mm³	0.004 mm³

Figure 3: Steps of optical coherence tomography interpretation – signal strength and key parameter. (a) Key parameters in healthy eye. (b) Key parameter in glaucoma. (c) When disc area is < 1.3 mm² or > 2.5 mm² rim area, CD ratio and CD volume are shown in gray color

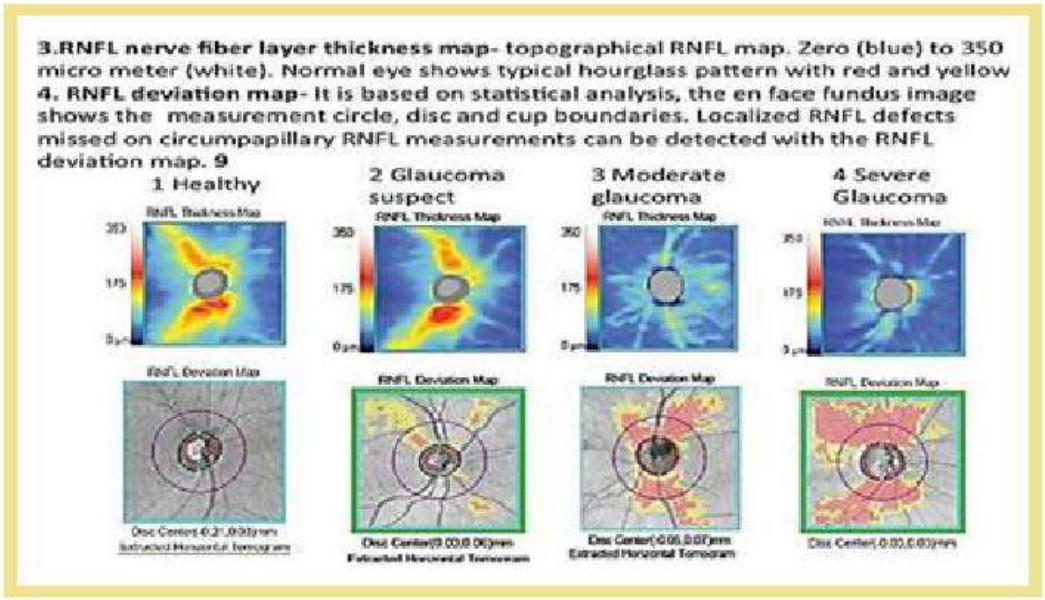

Figure 4: Steps of optical coherence tomography interpretation – retinal nerve fiber layer thickness map and retinal nerve fiber layer deviation map – Severity of glaucoma shows corresponding decrease in retinal nerve fiber layer thickness. (1) Healthy; (2) glaucoma suspect;(3) moderate glaucoma; and (4) severe glaucoma

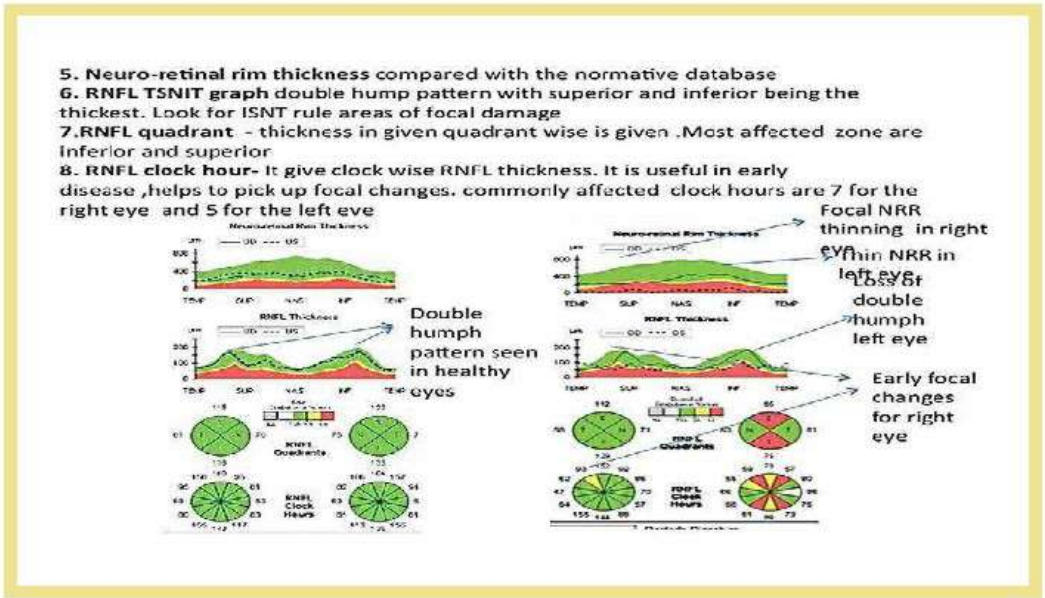

Figure 5: Steps of optical coherence tomography interpretation – Neuro-retinal rim thickness, retinal nerve fiber layer TSNIT graph, retinal nerve fiber layer quadrant and retinal nerve fiber layer clock hourwise thickness. (a) Neuro-retinal rim and retinal nerve fiber layer thickness in healthy eye,(b) Neuro-retinal rim and retinal nerve fiber layer thickness in glaucoma

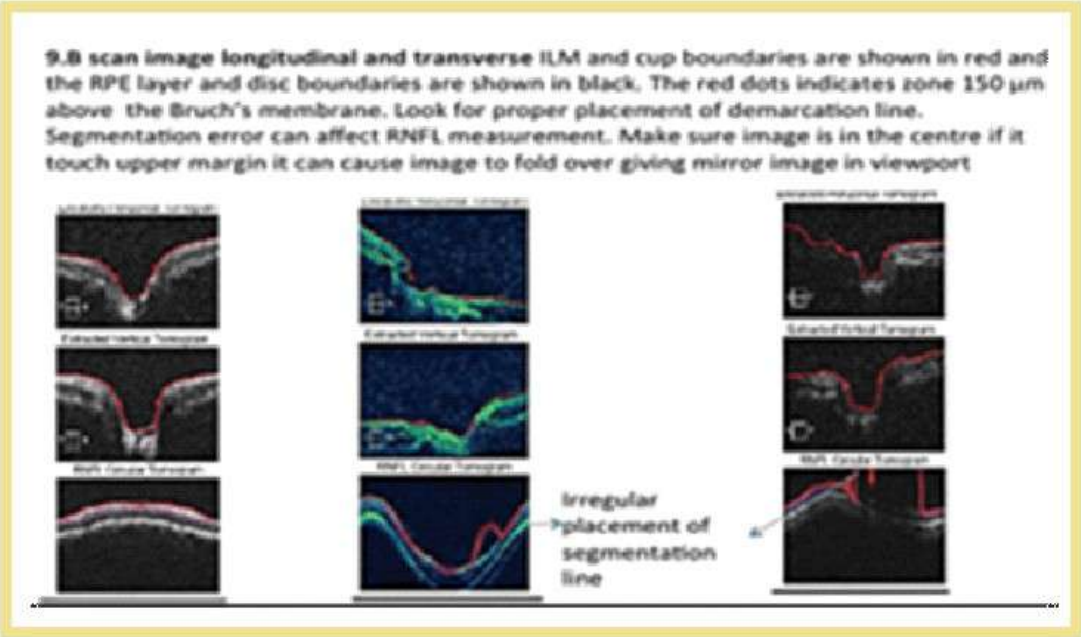

Figure 6: Steps of optical coherence tomography interpretation-B scan image longitudinal and transverse. (a) Properly placed segmentation line; (b and c) segmentation error

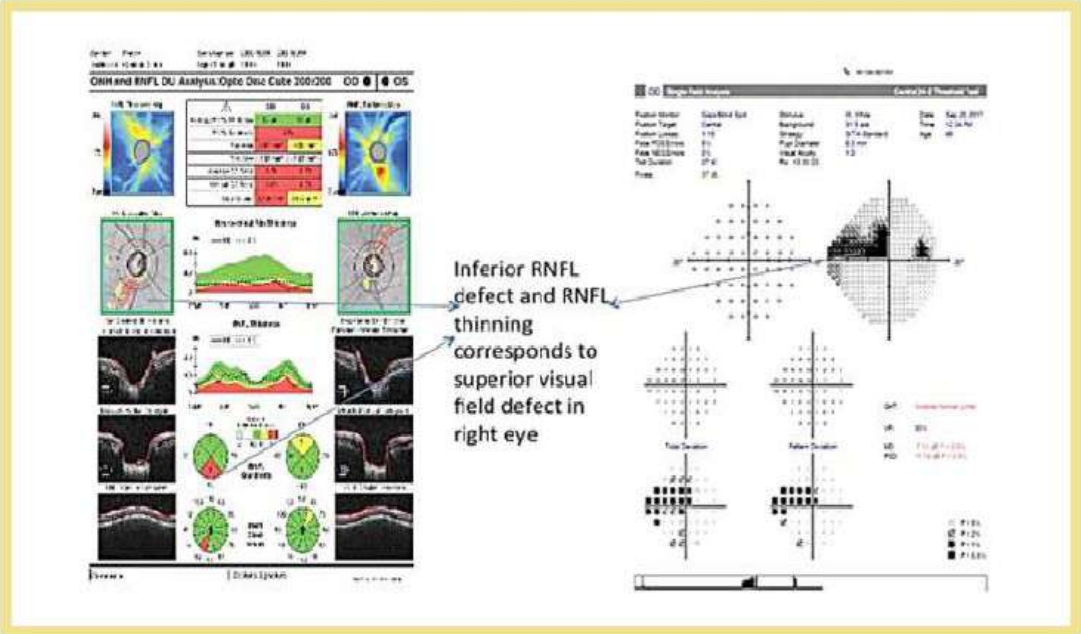

Figure 7: Structure and function correlation. (a) Optical coherence tomography optic disc and retinal nerve fiber layer shows inferior retinal nerve fiber layer defect and retinal nerve fiber layer thinning in right eye; (b) visual field test shows superior visual field defect in right eye

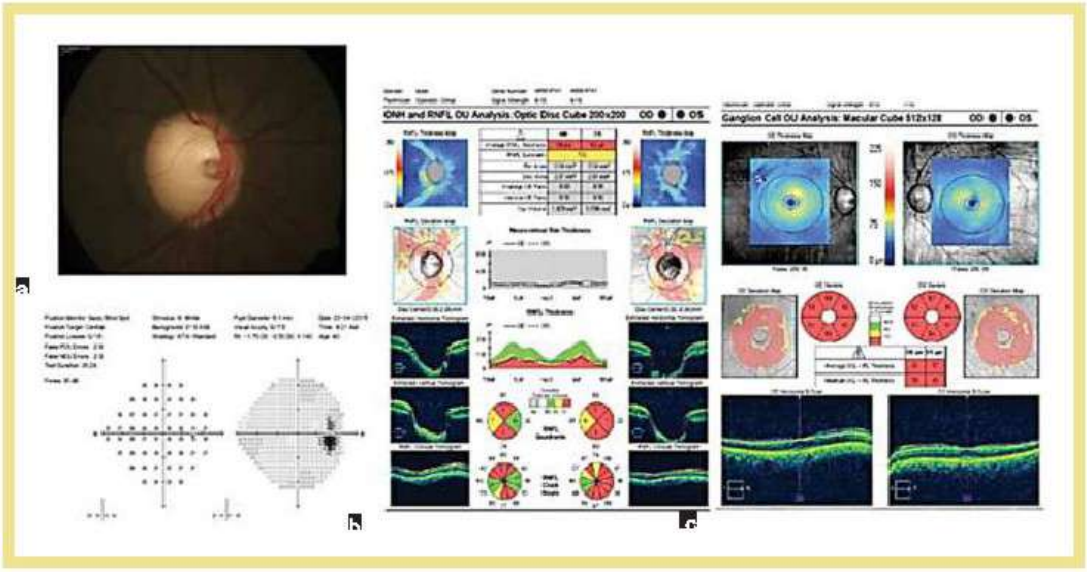

Figure 8: Pre-perimetric glaucoma. (a) Disc photograph of the right eye showing large disc with large cup with superior and inferior rim thinning. (b and c) optical coherence tomography retinal nerve fiber layer and macula confirms thinning. (d) Visual filed test noted to be within normal limit.

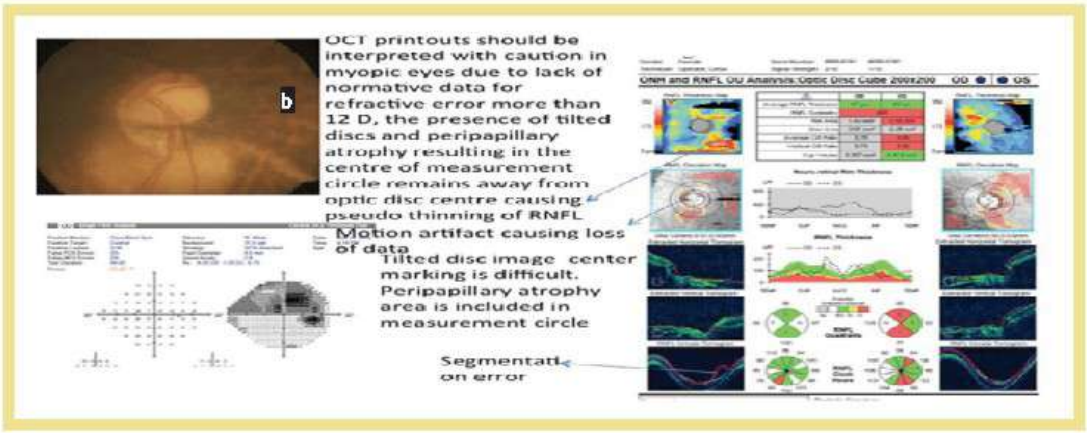

Figure 9: Optical coherence tomography in high myopia. (a) Disc photo showing tilted myopic disc with peri-papillary atrophy inferiorly with enlarged CD ratio with inferior rim thinning. (b) Optic nerve head and retinal nerve fiber layer optical coherence tomography of the same patient with poor quality scan. (c) Visual field test showing superior field defect

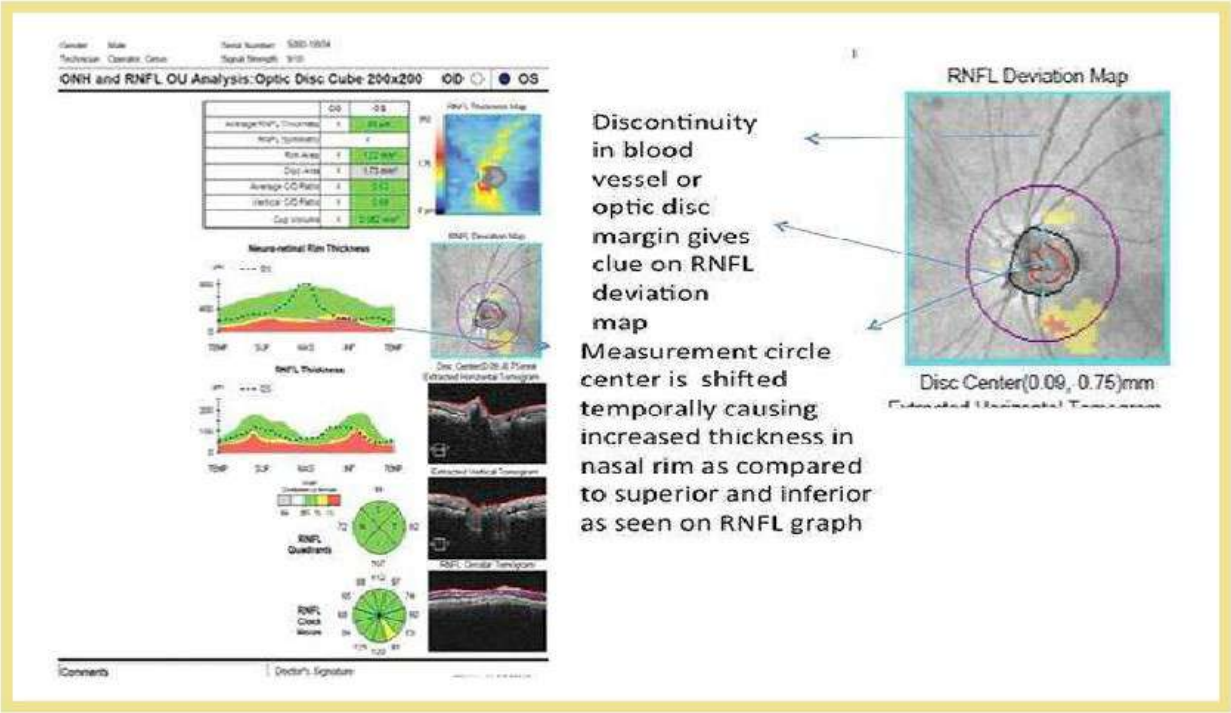

Figure 10: Motion artifact.

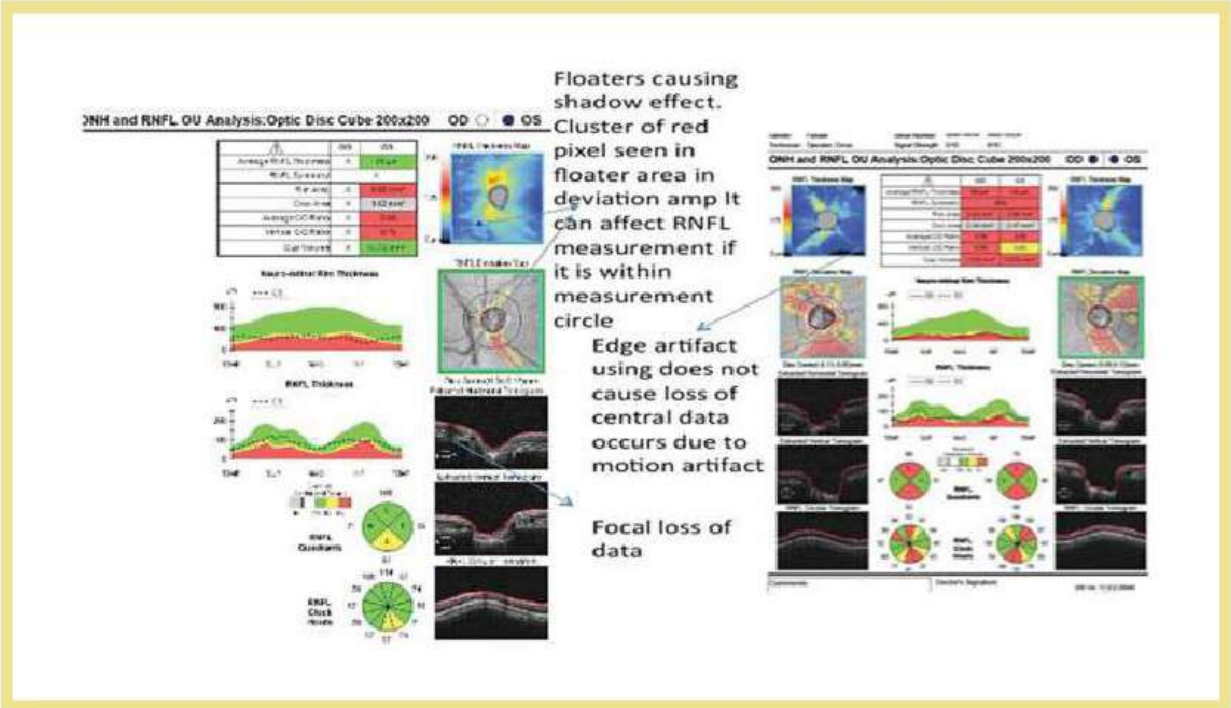

Figure 11: (a) Artifact due to floater. (b) Edge artifact.

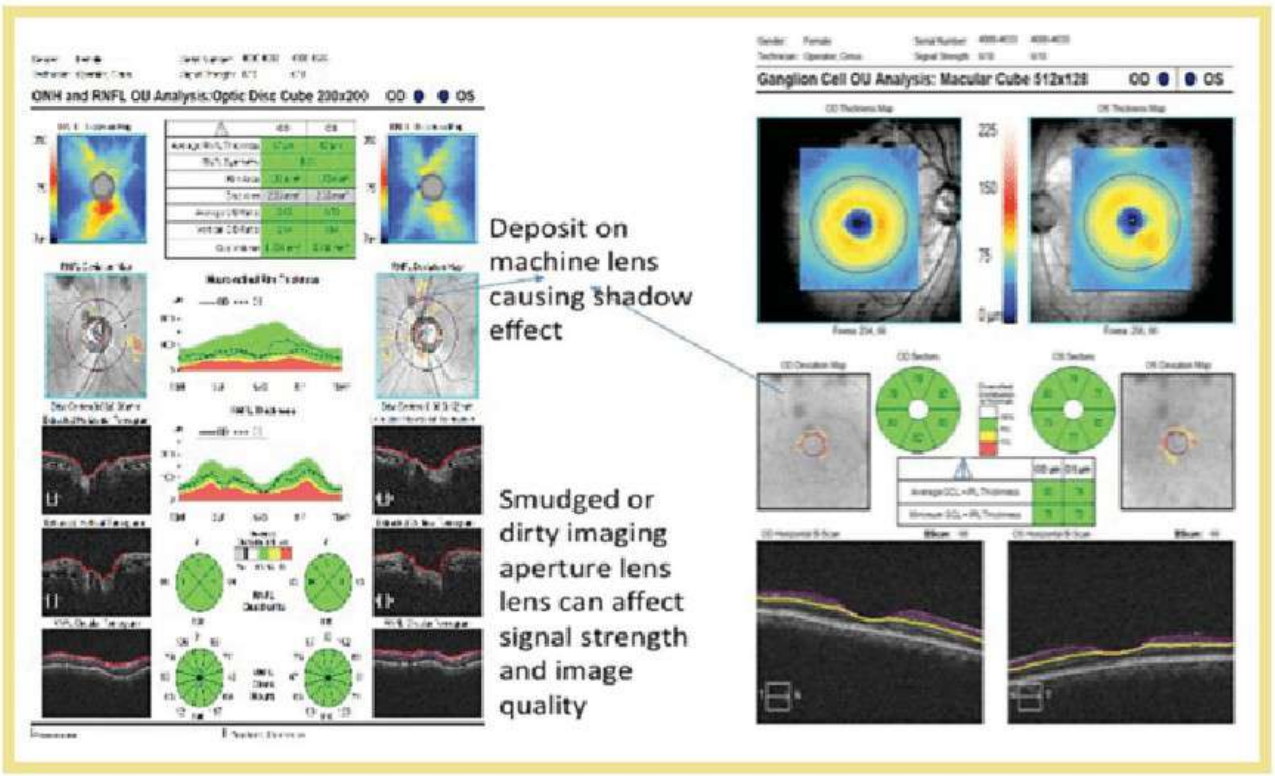

Figure 12: Machine related artifacts. A and B showing shadow effect due to deposit in machine lens.

Normative Database

The normative database is based on 284 healthy adults with an age range of 18–84 years, refractive error of − 12 to + 6D; ethnicity includes Caucasians 43%, Asians 24%, African America 18%, Hispanic 12%, mixed ethnicity 6%, and Indian 1%.[7-12] The average RNFL thickness in Indian eye reported in the literature is 104.8 ± 38.81 µm. Superior RNFL is 138.2 ± 21.74 µ, inferior RNFL 129.1 ± 25.6 µ, nasal 85.71 ± 21, and temporal 66.38 ± 17.37.[12] Caucasians had thinner mean RNFL values 98.1 ± 10.9 µm than Asians 105.8 ± 9.2 µm.

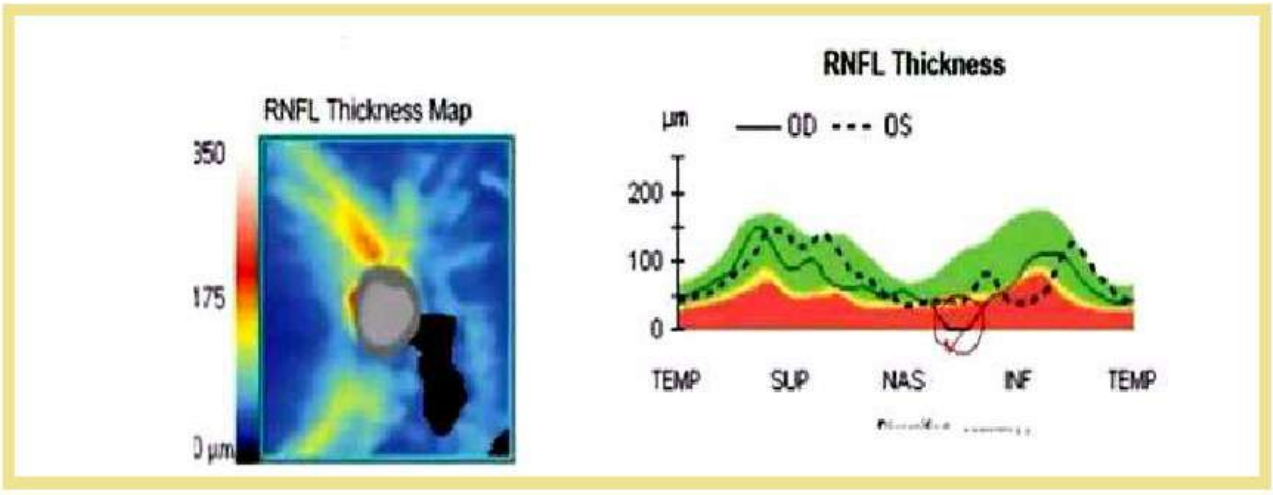

Figure 13. Floor effect: RNFL thickness never reduces to zero,Minimum-40-50 microns (glial tissue, vessels),No use in advanced glaucoma

Macula Optical Coherence Tomography

The macula has 50% of ganglion cells, which has been reported to help detect early glaucoma. It uses macular cube of 512 × 128 pixels with six linear scans in a spoke configuration. The inner boundary is formed by vitreous-retinal interface and outer boundary by retinal pigment epithelium. Yellow, green, and red represent thicker retina and blue represents thinner retina. It has thickness map, deviation map, sectoral value and average ganglion cell layer (GCL) + inner plexiform layer (IPL), and minimum GCL + IPL [Figures 14-16].

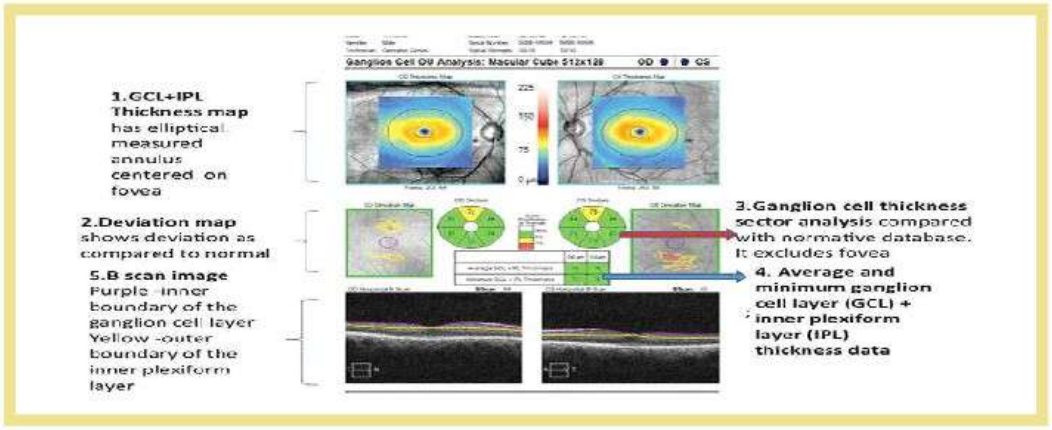

Figure14: Ganglion cell analysis report-based on macular cube analysis 512×128 or 200×200 scans

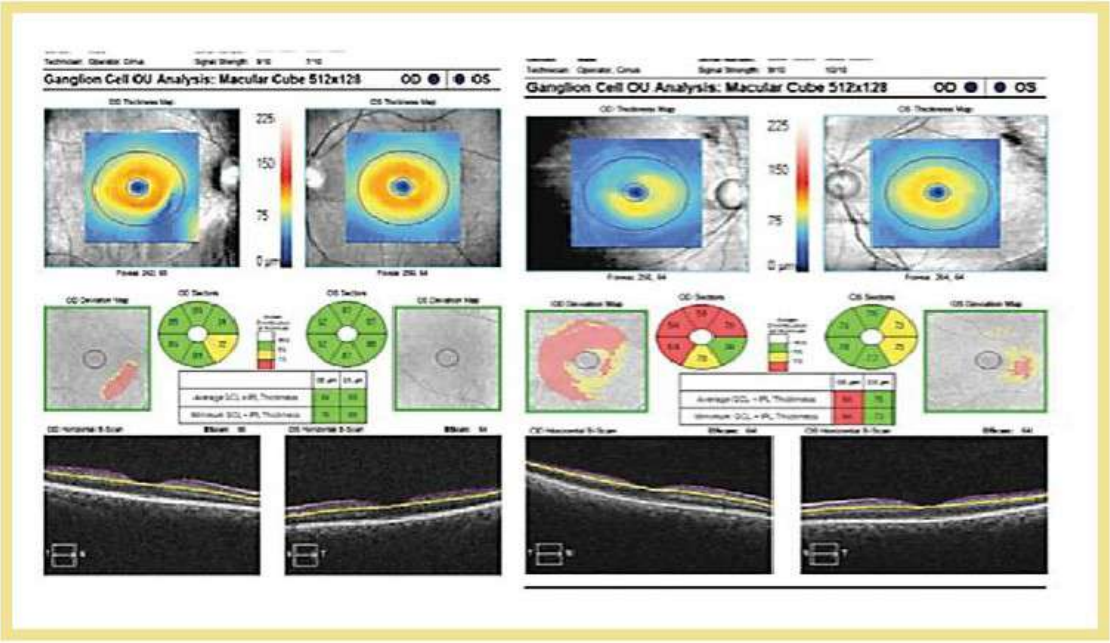

Figure 15: Ganglion cell analysis report. (a) Healthy and (b) glaucoma

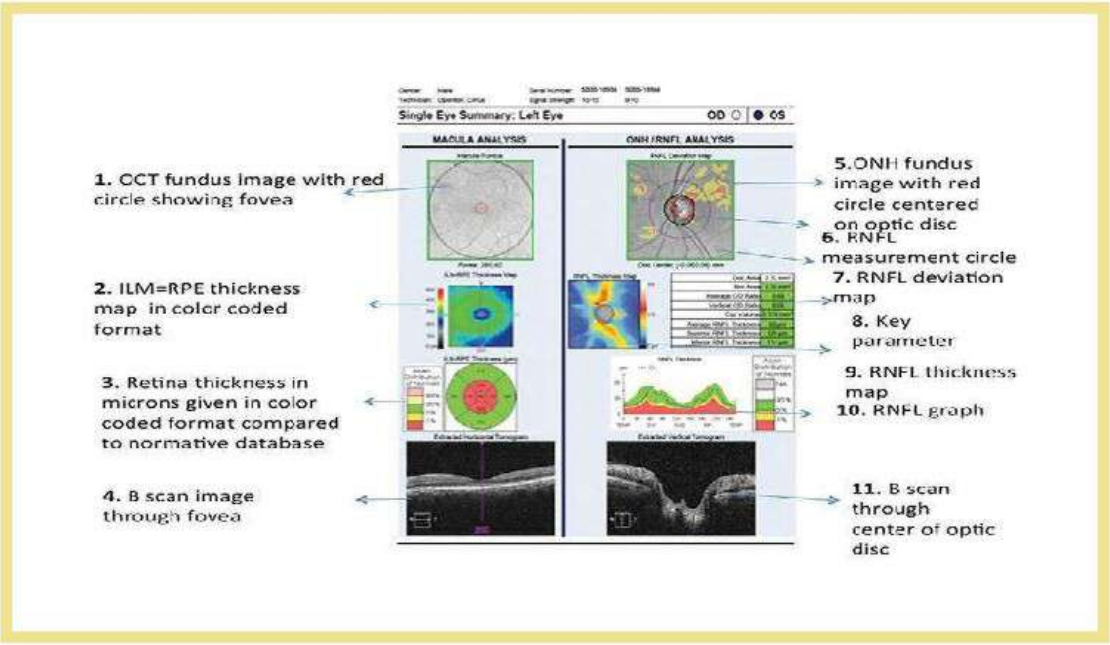

Figure16: Single eye summary report– Give over view of eye including macula , optic nerve head, and retinal nerve fiber layer analysis

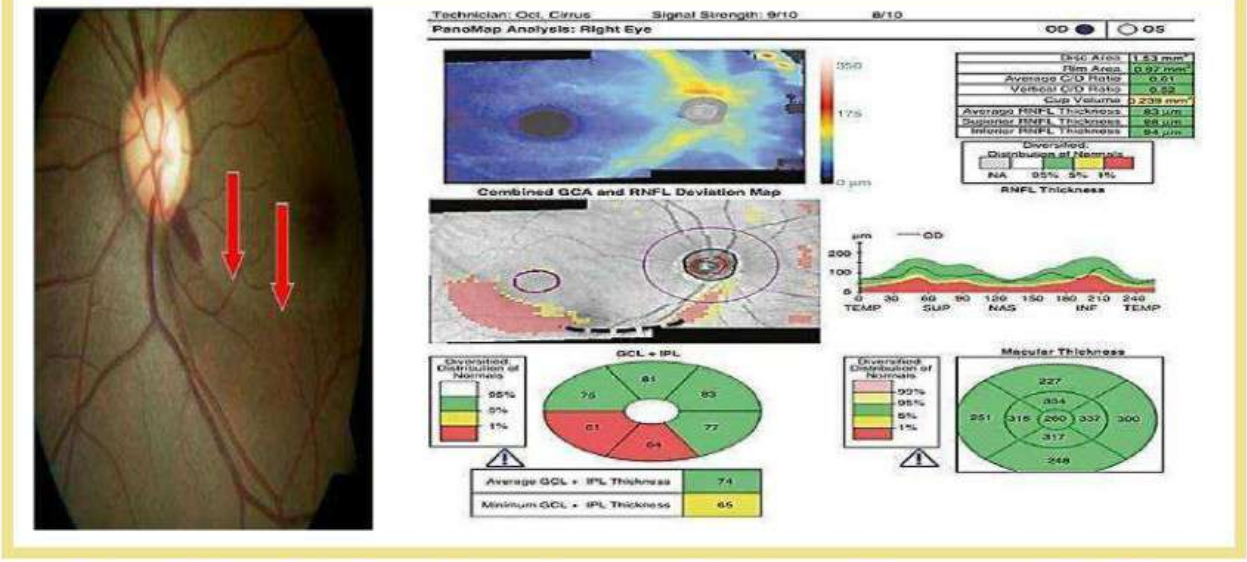

Figure17: (a) Fundus photo shows vertical cup diameter ratio 0.7, thinning of neuro-retinal rim with wedge-shaped inferior retinal nerve fiber layer defect and Visual field shows superior scotoma.(c)optical coherence tomography panomap analysis shows reduction in retinal nerve fiber layer thickness in the inferior-temporal quadrant with corresponding depression in the TSNIT graph and reduction of ganglion cell complex thickness.

Hood report : Thickness map of RNFL,GCL and RNFL and GCL+ probability maps with visual field test locations overlapped[18]

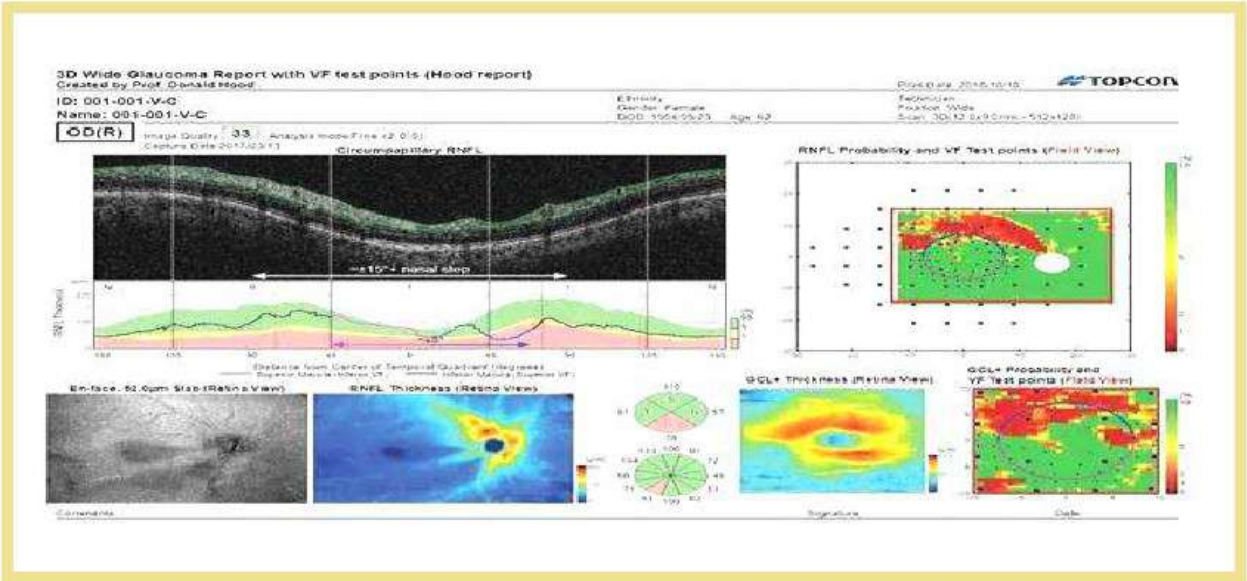

Figure 18: Hood report of RNFL and GCL + Probability maps with visual field test locations overlapped

Recent Advances

Lower vessel densities have been reported in glaucoma as compared to healthy adults and have shown good discriminatory abilities.[14,15] Enhanced depth imaging allows lamina cribrosa imaging which is a proposed site for retinal ganglion cell injury.[16] Lamina cribrosa deformation in response to IOP changes may play a role in patho-physiology of glaucoma and the OCT may help analyze this.[17]

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SURGICAL TECHNIQUE

Femtosecond Laser Assisted Cataract Surgery in Posterior Polar Cataract

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Posterior polar cataracts (PPC) are a challenge for every cataract surgeon due to their propensity for posterior capsule dehiscence and weakness. Although the initial reported incidence of posterior capsule rupture (PCR) was anywhere between 26 to 36 %,subsequent improvements in understanding of techniques and technology have led to a reduction in the rates of PCR to 6 to 7%.In order to protect the inherently weak posterior capsule, the surgical strategy for posterior polar emulsification involves the following principles:

a) avoid rapid build-up of hydraulic pressure within the capsular bag

b) creation of a mechanical cushion above the weak capsule, and

c) adherence to the principles of closed chamber technique

Cortical cleaving hydrodissection is usually avoided in these eyes as a rapidly passing fluid wave can lead to sudden build-up of hydraulic pressure within the capsular bag and lead to blowout of the posterior capsule. As a result, most surgeons prefer to perform conventional hydro-delineation or inside-out delineation to generate a plane of separation within the nucleus, as well as avoid buildup of pressure within the capsular bag. However, most of these techniques depend on injection of fluid within the nucleus to create a mechanical cushion.

As cataract surgery continues to evolve, the widespread adoption of femtosecond laser assisted cataract surgery (FLACS) promises to be yet another refinement in surgical performance and possibly even outcomes. Whereas, FLACS has been reported in normal as well as complicated cataract scenarios, including pediatric cataracts, subluxated and traumatic cataracts and phacomorphic glaucoma, its applicability to increase safety in posterior polar cataracts is one that has been widely accepted and applied by surgeons across the globe. We first published a technique of delineating the nucleus into multiple layers using femtosecond laser technology to enhance safety during posterior polar cataract surgery- Femtodelineation.

The femtosecond laser is equipped with different patterns for lens fragmentation. We find the cylindrical pattern of lens division particularly useful for posterior polar cataracts. The number, the diameter and the depth of each cylinder can be chosen by the surgeon guided by the live anterior segment optical coherence tomography (OCT) view (**Figure 1**).

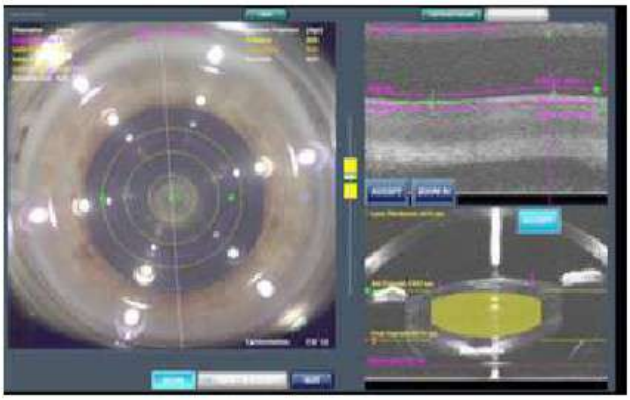

Figure 1. Femtosecond laser being programmed to create 3 cylinders within the lens with the offset from posterior capsule decided based on anterior segment optical coherence tomography (OCT).

Three cylinders are created within the nucleus. As the laser fires, it creates three distinct layers of demarcation within the nucleus, from the centre to periphery, shielded by a fourth peripheral epinuclear layer **(Figure 2)**.

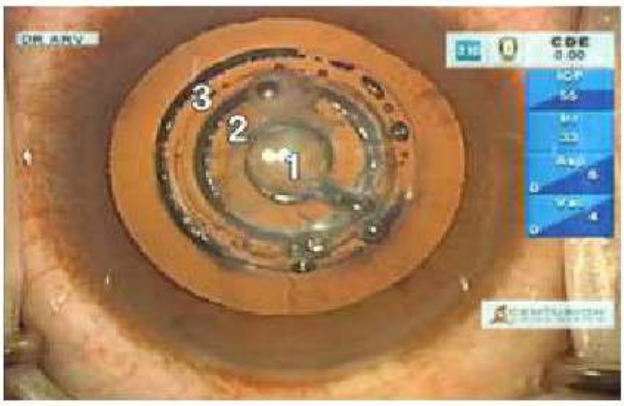

Figure 2. Four different zones of delineation within the lens, which provide enhanced cushioning to the posterior capsule.

The width of these cylinders can be modified manually, and an offset of at least 500 microns from the posterior capsule is preset based on the real-time OCT view. This helps in safeguarding the potentially fragile posterior capsule from injury.

Once the capsulorhexis flap is removed, we directly proceed to remove the nucleus without performing any hydroprocedure. Starting from the innermost layer, each of these sharply delineated layers is emulsified from inside out, within the cushion of the outer layer. This is done using a low aspiration flow rate (AFR) of about 14 to 16 cc/min, minimal ultrasound energy and a modest bottle height of about 60-70 cm. Since the walls of these layers have a very sharp and clear-cut demarcation, the entire delineated layer can be emulsified with minimal mechanical manipulation from the second instrument. This in turn reduces stress on the capsular bag-zonular complex. Also, there is a thick and uniform epinuclear cushion that remains even when the entire nucleus is removed (**Figure 3**).

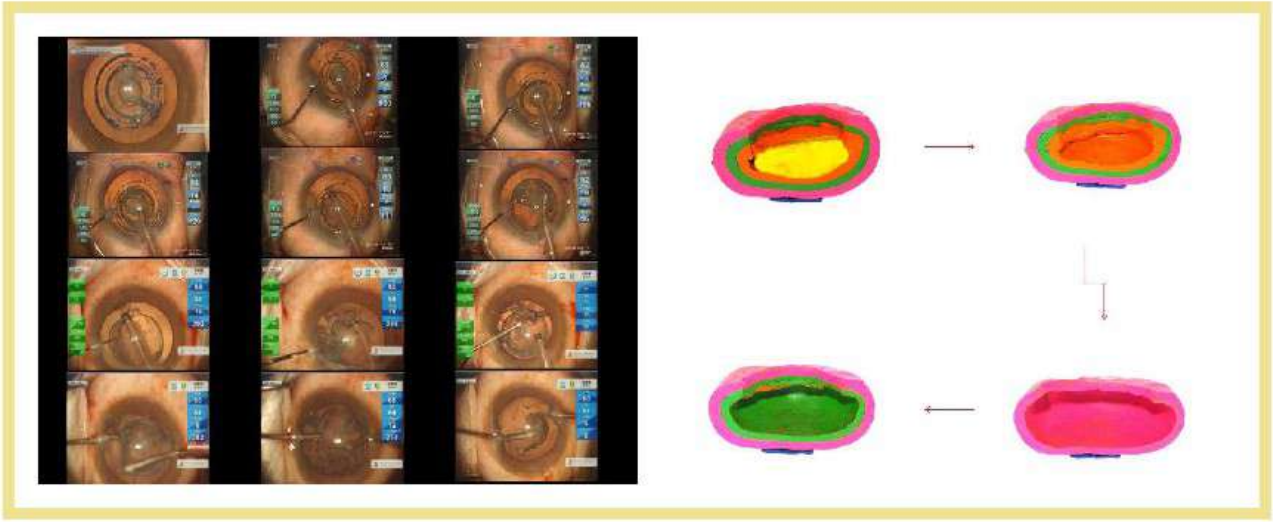

Figure 3. Sequential photographs in a clay model showing how easily each of the delineated layers is removed one by one, and finally a thick epinucleus left behind that can be easily removed with irrigation / aspiration.

At the very end, owing to the sharp vertical wall created by the laser circumferentially, removal of this epinuclear cushion becomes very easy. It is gently stripped off from the capsular bag fornices in the two quadrants (180 degrees) directly opposite the phaco tip using a low AFR of 14 to 16cc/min, a vacuum of around 200 mmHg with minimal ultrasound energy. At this stage, no attempt is made to completely aspirate it. Now, dispersive ophthalmic viscosurgical device is injected in the anterior chamber prior to removal of the phaco probe to prevent forward bulge of the capsular bag. The epinuclear cushion, which is already partially detached, is then detached in the sub-incisional quadrants using bimanual irrigation and aspiration (I/A). Once it is detached circumferentially, it is aspirated with bimanual I/A. At this point,

even if there is passage of fluid between the capsule and cortex, it will not lead to a dramatic buildup of hydraulic pressure.

In our clinical experience, we found a PCR rate of only 4.4% (only 2 out of 45 eyes) when performing femtodelineation for posterior polar cataracts. This rate is lower than our own reported rate with inside-out delineation technique.

The biggest advantage of performing femtodelineation is that it allows creation of multiple, precise, customized, layers within the lens, which provide cushioning to the weakest part of the posterior capsule, and thereby enhances safety in posterior polar cataract. Also, in the event of a pre-existing posterior capsule defect or a posterior capsule rupture, these multiple layers of cushioning will prevent enlargement of the defect and nucleus drop as well as vitreous prolapse. Further, the added advantage is the perfectly centered and customizable anterior capsulorhexis. This is very helpful in the event that there is inadequate posterior capsule support and an IOL needs to be placed in the ciliary sulcus.

To summarize, femtodelineation provides better safety during posterior polar cataract surgery by reducing rates of posterior capsule rupture.

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SURGICAL TECHNIQUE

Tunnel Construction in Manual Small Incision Cataract Surgery (MSICS) with Razor Blade Fragment

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Abstract

Manual small incision cataract surgery is preferred surgery for all types and situations of cataract. Each day evolution and modifications are seen in each step of clinical practice. Here we discuss tunnel construction by a razor blade fragment which plays an important role in MSICS and it has a domino effect on the quality of surgery, intra op complications and post-operative visual rehabilitation. This is one of the most cost effective as well as time efficient ways.

Keywords – razor blade fragment, scleral tunnel construction, SICS, MSICS.

Introduction

Manual Small incision cataract surgery (MSICS)is preferred by many surgeons as it can be done in all types of cataracts and pupils; and requires very little investment.

Tunnel construction plays an important role in MSICS and it has a domino effect on the quality of surgery, intra op complications and post-operative visual rehabilitation. The incision shape, size and length should be carefully planned taking into account the type of cataract, pre-existing refractive error, condition of the endothelium and age of patient. The sclerocorneal tunnel is desirable to be within the astigmatic funnel.

History and Other Techniques

Incision shapes have evolved over the years and commonly known types are namely chevron, frown, Blumenthal, straight and smile. The SIA depends upon the length, width, location, shape, depth and corneal entry. Kratz et al. mentioned self-sealing cataract incisions in 1980 and Girard in 1984.Kratz thought of scleral tunnel as an astigmatic neutral way of entering the anterior chamber. [1] In 1984, it was shown by Thrasher *et al.* that a 9.0-mm posterior incision induces less astigmatism than a 6.0-mm limbal incision. [1]In 1990,

Michael McFarland developed a suture less incision, and Pallin described a Chevron shaped incision. During the same period, Singer popularized the frown incision. [1]

Tunnel is created in three steps. The initial incision is placed, the sclera and cornea are dissected and then the entry into the anterior chamber is done by a different instrument or a single sharp instrument. Tunnel's valvular effect is established on apposition of two opposite channelled sclerocorneal surfaces, whereby, the upper surface inter-digitates in perfect unison with the lower surface.

The fact that scleral tunnels can be carried out in variety of ways is acknowledged. The procedure described in this article makes use of the cut-edge of a razor blade on blade breaker handle. Different instruments like crescent knife, keratome, micro-keratome, diamond knife, N.64 beaver blade or No. 11/15 blades can be used depending on the surgeon's comfort. [2][3] The author is acquainted with the use of razor blade for tunnelling as well as crescent knife and keratome. [2]However, the technique described here is a cost effective and faster as there is no constant change of instruments

Use of Razor Blade Fragment

It is a 23 x 43 mm rectangular stainless-steel blade. The blade is held on one corner edge by a Castroviejo blade braker and secured in place by rotating the lock. Then hold this apparatus in the dominant hand and clasp the free edge of blade between the thumb and finger. With this in position, twist the wrist 90^0 toward the body in a single swift movement thus breaking a fragment from the whole blade plate into an elongated quarter circle pattern. The straight edge of the blade is used for dissection and protrudes out of the edge of blade braker. The circular part is clasped by the beaks of the blade braker. From a single blade we a make upto four blade fragments (figure 1 A and B).

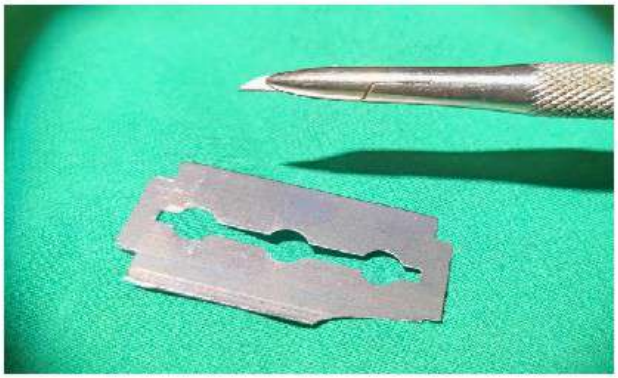

Figure 1 A Sharp edge fragment of razor blade to be used.

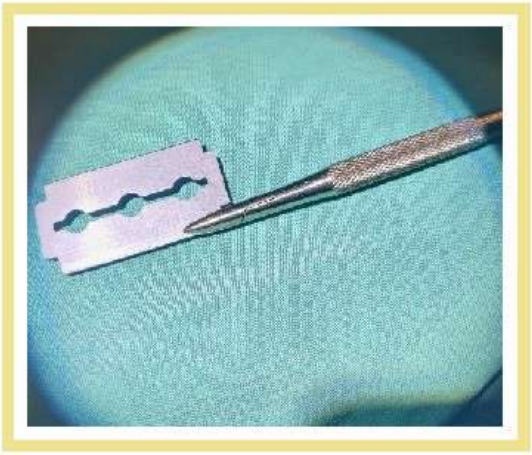

Figure 2 B Hold the edge of razor blade by a Castroviejo's blade braker

Sterilization of blade fragment

The blade is double packed in transparent packing and kept in ethylene oxide (ETO) for 12 hrs at 5 psi, on 55 ^{0}C. Once the packing is sterilized, it has a shelf life of 6 months.

Procedure of Tunnel Construction with Razor Blade

Stabilizing the globe while dissecting the tunnel is of vital importance. This can be achieved by holding the conjunctival edge with a toothed forceps. However, this can lead to button holing of conjunctiva. A better way to achieve this by making a partial scleral grove by blade and hold it gently. During this manoeuvre care should be taken to neither depress nor pull at the globe. This will maintain the globe within the correct view in the microscope and prevent tearing of the groove. *An initial vertical partial thickness scleral incision*s made on the proposed site of the tunnel. Linear scleral incision about 2 mm posterior to surgical limbus and about 5-7 mm long and 1/3 to ½ thickness of scleral thickness is made with a cut-edge of a razor blade. To judge the appropriate depth of tunnel the blade should 'just' visible from the upper scleral lip.

Lamellar scleral dissection with a half-thickness sclerocorneal tunnel incision, the direction of the blade fragment should always be parallel to the scleral plane. The cutting edge of the blade should lead the dissection on one side and it should be flipped to continue the same on the other side. While dissecting one should always follow the contour of the eyeball to judge the appropriated thickness of the upper sclera lip. The blade should just be visible on the scleral part. *Lamellar corneal dissection-* The plane of dissection should be continued from the scleral plane. While entering the cornea the orientation of blade should be changed along with corneal contour. To check the right plane of dissection one should press and check for the resistance and dimple formation but the lower half of corneal. In case of a premature entry the resistance is lost

and the tip is freely movable in AC If the scleral place is too deep it may lead to a premature entry, or if its superficial it can lead to deroofing of scleral/corneal part of the tunnel. *Side pockets* may be dissected on either side to accommodate the nucleus in cases with large and hard nucleus. To dissect the scleral pockets, side edge of the fragment is advanced laterally with a tilt along with the contour of corneal curvature. *Entry into the AC*-The heel of the blade fragment is raised until the blade becomes parallel to the iris plane, resulting in a dimple of corneal surface. The blade is then advanced anteriorly in the same plane until the AC is entered and internal wound is visualized as straight line. *Extension of inner lip*-The entry is made from one side with keeping the cutting edge of fragment to the side of motion. The advantage of using a blade fragment is for creating a entry in single sliding motion. During extension care should be taken to continue the same plane.

Advantages

Economic benefits, a box of razor blade contains up to 50 pieces and each piece makes four blade fragments. This is considerably cheaper than cost of crescent and keratome which is also used to create the tunnel. Faster, as less time in instrument changes and handling. Minimal instrumentation, same instrument is used for making holding groove, scleral incision, lamellar dissection and entry. Less instrumentation at the tunnel site maintaining integrity of the tunnel. Universally available in all parts of the country. Can be sourced at a moment's notice. Newer plane at same site in pre mature section in cases of a deep tunnel a new plane can be created on the upper lip as the blade is very thin and sharp and it is possible to make a new plane. Reproducible technique on a larger scale.

Limitations

Ill formed fragment, skill is needed for creation of correct size and shape of fragment. Unavailability of guard, there is no guard on the blade braker. Improper sized fragments may create hassles during tunnel creation. Learning curve is long, it requires training and practice to develop judgement on sharpness and shape of blade for use. Difficulty in superior section and deep sockets as there is no angle at the blade unlike in a keratome or crescent. Thereby making the approach difficult while tunnelling.

Conclusion

SICS is significantly faster, less expensive and less technology-dependent. MSICS may be the more appropriate surgical procedure for cataracts in our country due to its cost benefits, which make it an attractive option for high volume surgeries like community charity centres. MSICS with blade fragment fits the bill in such scenarios and can be adopted for large scale usage.

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SURGICAL TECHNIQUE

Sub-Retinal Fluid Drainage in Retinal Detachment (Buckling) Surgery

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Improved examination techniques, improved surgical equipment and improved scleral buckling materials have greatly improved the prognosis for patients with rhegmatogenous retinal detachment.[1] Yet many surgeons would probably agree that the success or failure of a detachment procedure is frequently determined by the success or failure of subretinal fluid (SRF) drainage and complication as a consequence of this step.

It is not necessary to get into an argument concerning the necessity for fluid drainage. In a certain percentage of retinal detachment procedures, drainage is not necessary but in many, it is a must (Table 1). The point to be made is that drainage is fraught with complications, should be avoided whenever possible, but should be done well whenever attempted. The decision regarding whether or not to drain is based on the possibility of fulfilling Gonin's postulates. In other words, if the retinal break can be made to lie against the choroid, and if it can be kept in that position for a few days, the fluid under the retina will disappear, and the retina will flatten. The one cardinal rule in drainage is to perform it carefully. Common complications of a poorly performed drainage are intraocular hemorrhage choroidal detachment, new retinal tears, retinal incarceration and loss of vitreous. It is thus easy to see why most retinal surgeons give attention to the finest details during this procedure.

Table 1: Indications of Subretinal Fluid Drainage

1. Long standing retinal detachment
2. Bullous retinal detachment
3. Inferior retinal detachment
4. Eyes with poor choroid circulation having retinal detachment
5. Retinal detachment with staphylomatous sclera
6. Retinal detachment with senile choroidopathy
7. Retinal detachment with increased IOP
8. Retinal detachment with high myopia
9. Retinal detachment with recent intraocular surgery
10. Retinal detachment with large tears

Selection of Drainage Site

It is imperative that significant fluid is present at the proposed drainage site. The surgeon must inspect the area for fluid immediately before proceeding, and scleral depression should be performed to ascertain the amount of fluid present.

While inspecting the eye for the best choice of drainage site, one has to consider many factors. The first of these is the facility of exposure; i.e., the area to be selected for drainage must be an area that can be handled easily with knife, sutures and cotton applicators without causing undue pressure rise within the eye. A second factor concerns the avoidance of vortex vein ampullae, large choroidal vessels and previously frozen areas during cryopexy. The presence of absence of large choroidal vessels can be made out by depressing the area being considered for drainage and looking for the presence of pink ribbon shaped vessels in the area. Incisions through the sclera are best made in radial meridian.

During drainage, the operator should monitor the fluid release from the external aspect, viewing the sclerotomy site at all times. When the globe softens, it is permissible to look into the eye to check the success of the drainage, but a temporary tie should be used to close the sclerotomy site. The decrease in intraocular pressure should be limited by maintaining some tension on the bridle sutures, and the total time taken for the procedure can be minimized by wearing the indirect ophthalmoscope throughout the procedure (though this can be cumbersome). Whether or not multiple drain sites are necessary depends entirely on the success of each preceding drainage.
Handling of the sclerotomy site after drainage is simple if it is done beneath the buckled area, since the buckle will tamponade the scleral wall. If the sclerotomy is not beneath the buckle, it must be closed with a 6-0 silk suture. The sclerotomy should be closed before injecting saline into the vitreous if the eye is too soft. It should always be closed if air is to be injected into the vitreous cavity.

Aside from care, looking into the eye prior to drainage, good exposure, and avoidance of vascular areas, there are some other thoughts that should be going through the surgeon's mind as he selects his area for drainage. These have to do with the relationship of this site to the retinal tears, it's relationship to the buckle, it's relationship to the macula in terms of configuration of the detachment and in terms of the configuration of the buckle and finally the consideration of postoperative positioning. If the drainage site need to be near a retinal break, it is wise to indent the sclera with a cotton applicator in the area where the retinal break has been marked on the sclera. This will tend to bring the retinal hole in contact with the choroid very quickly and allow only subretinal fluid to escape. Unfortunately, if the drainage site is close to the

hole, this indentation will also affect the flow of subretinal fluid through the drain site. It is, therefore better to place the sclerotomy at a comfortable distance from the retinal break unless one is tackling a large dialysis and the patient has a fluid vitreous. In this case, if substantial reduction of eye volume is required, one may occasionally deliberately make the drainage site near the area of the break.

As far as the relationship between the sclerotomy and the element used to buckle the side of the eye is concerned, there are three possible choices. The sclerotomy may be anterior to the buckle or band, underneath the buckle, or posterior to the buckle or band. Generally speaking, the sclerotomy posterior to the buckling element will give the most complete drainage, but this is also the riskiest site in case any complication occurs. Therefore, a site that will ultimately be closed by the exoplant is always preferred since it facilitates subsequent management if any drainage complication occurs. Therefore a site that will ultimately be closed by the expolant is always preferred since it facilitates subsequent management if any drainage complication occurs.

Superior temporal detachments may have serious complications as a consequence of subretinal fluid drainage. This is because any trickle of subretinal blood will tend to run down inferiorly toward the macula, and finally gravitates beneath the detached macula. For this reason, the detachment that involves the superior half of the fundus is preferentially drained on the nasal side if possible since it avoids the likelihood of subretinal blood trickling down behind the macula. Total detachments or inferior detachments are best drained through the temporal side because of availability of proper exposure.

The postoperative position of the patient should be considered when choosing a drainage site. There is one position which must be avoided at all costs i.e. the supine position with the patient's face directed toward the ceiling. Any subretinal blood will then trickle down to the macula and decrease the rate and extent of visual return. Except in rare circumstances, almost any other position is preferred.

Drainage Techniques

Drainage of subretinal fluid is performed in a standard fashion at the most optimal site available and this is determined by the configuration of the retinal detachment. The final selection of the location for SRF drainage is based on presence of sufficient subretinal fluid which allows safe drainage. This requires evaluation of:

i. The distribution of subretinal fluid when the eye is in a position at which drainage will be performed. This is important since SRF can shift in upto 25% of cases.
ii. The location of the retinal breaks.

iii. The location and configuration of the buckle.
iv. The vascularity of the choroid
v. Features like area of vitreoretinal and epiretinal membrane traction and the ease of exposure of the proposed drainage site.

The optimal locations for drainage are just above or below the lateral or medial rectus muscle, because major choroidal vessels are avoided and exposure of the sclera is ideal. Drainage is usually performed at or slightly anterior to the equator, and a site that will ultimately be closed by the exponent is always preferred. This facilitates subsequent management if drainage complications occur.

After proper exposure and careful localization of the drainage site, a 3 to 4 mm radial incision through the sclera is performed so that the centre of the sclerotomy will be at the appropriate location. All scleral fibers are divided until subtle prolapse of uveal tissue is observed. The choroid is then closely inspected for prominent choroidal vessels, using a loupe and/or the 20 diopter condensing lens and indirect ophthalmoscope. If visible vessels cannot be avoided during penetration, a second site nearby is selected, and another scleral incision is performed if the area of exposed choroid is free of prominent vessels, it is lightly treated by flat diathermy probe. This causes minimal retraction of the edges of the sclera to improve visualization, and it probably reduces the chances of hemorrhage. All significant traction upon the eye is then eliminated to reduce intraocular pressure (IOP) as much as possible. The subretinal space is then entered with a sharp tipped conical penetrating diathermy electrode. Modest pressure is used to insert the device perpendicular to the surface of the sclera until the subretinal space is entered. The event is usually heralded by a subtle "pop", which is usually perceived by touch or observation. Because of the tapered shape of the electrode, significant amount of SRF does not exit the eye until this device is very slowly withdrawn from the eye. The penetrating diathermy electrode is relatively blunt, compared to a new needle and penetration of a soft eye with congested choroid may be somewhat difficult. This is managed by modest elevation of IOP by traction upon the muscle fixation (bridle) sutures or by slight tightening of this encircling band.

An oblique or tangential path of penetration is recommended by some surgeons to avoid perforation of the retina, but this can result in a flap value of the choroid, which can limit or prevent the drainage.

If a proper drainage site has been selected, penetration of the retina with the tapered diathermy is exceptionally rare, because it is removed prior to the release of significant amounts of SRF. Lasers have also been employed to drain SRF, but the expense and time required to use them are not associated with a significant enough reduction of the rate of complications.

An alternate approach to the incisional sclerotomy technique is drainage of SRF by a 26 guage needle. In this technique, after proper localization and selection of the optimal drainage site (as mentioned in the above method), the subretinal space is directly entered through the sclera with a 26-guage needle placed on a 2CC glass syringe (with a plunger). As soon as the surgeon notices the fluid coming out through the hub of the needle into the syringe, the needle into the syringe, the needle should be immediately withdrawn from the eye and gentle pressure with the cotton applicators should be applied to facilitate the drainage. The assistant's role becomes very important at this stage. The appearance of pigment granules suspended in the subretinal fluid usually indicates that the last of the SRF is exiting the eye. This technique is preferred at our centre because of ease of the procedure, less time required, avoidance of large scleral incisions which need suturing and prevention of retinal incarceration. In the past many cases of SRF drainage, we have never encountered retinal incarceration with this technique.

As the globe softens during drainage, IOP is very slowly increased to encourage further drainage and to avoid complications associated with hypotony. If a large tear is present, the sclera or the buckle and sclera overlying the break are indented with a cotton-tipped applicator. This maintains intraocular pressure and inhibits passage of intravitreal fluid to the subretinal space. Pressure can also be increased by placing applicators on either side of the sclerotomy site and gently pushing both toward the centre of the eye. This maneuver also tends to keep the sclerotomy open. Relatively normal IOP can also be maintained by indenting the sclera at a location far away from the sclerotomy site with cotton applicators.

The drainage site is not touched as long as fluid flows through it. Sudden and significant increase in IOP are avoided to reduce chances of retinal incarceration, and any sudden cessation of drainage requires immediate stoppage and examination of the sclerotomy site. As mentioned above, the appearance of pigment granules in SRF usually indicates the completion of drainage. When drainage ceases, the sclerotomy site is closed by temporarily tying the sutures over an exoplant or by pulling together the ends of an encircling band. If the buckling material does not adequately close the sclerotomy, the scleral incision is closed with a suture prior to significant elevation of IOP with the buckle. These measures are however not necessary when employing our technique of SRF drainage.

The eye is quickly inspected following closure of the sclerotomy site and preliminary adjustment of the scleral buckle. The site of drainage is first evaluated for signs of subretinal bleeding, the amount of persistent SRF is then determined, and the need for further drainage is considered. Significant amounts of SRF can be allowed to persist if an optimal buckling effect has already been produced.

If drainage of additional subretinal fluid is required, the initial sclerotomy site must be closely evaluated by scleral depression, in the area of drainage. If the pigment epithelium is clearly not in contact with retina, the sclerotomy site can reopened by reducing intraocular pressure and or removing the portion of the exoplant that covers the scleral incision. Additional drainage usually occurs spontaneously, or it can be initiated by gently manipulating the edges of the sclerotomy with cotton applicators or a forceps. In some cases, particularly those with viscous SRF, the retina may flatten completely at the site of the sclerotomy, while large amounts of SRF persists elsewhere. In this situation, additional sclerotomies must be performed if additional drainage is required to produce an adequate buckling effect.

Common Complications and their management

This step of a scleral buckling procedure is usually considered the most hazardous. Although drainage complications are not statistically associated with subsequent anatomical failure, (Wilkinson CP et al Retina 4:1-4, 1984) they can require a modification of the surgical plan and can often compromise postoperative visual acuity.

The three classic problems associated with drainage of SRF are hemorrhage, retinal incarceration and iatrogenic retinal holes. The first of these is the most common, occurring in approximately 3 to 4% of cases. Most hemorrhage is confined to a small areas surrounding the sclerotomy site, and additional surgical maneuvers are not required for its management. If more significant hemorrhage occurs, then all traction upon the eye should be immediately released, and the retina should be inspected with the indirect ophthalmoscope. If active bleeding is observed, the pressure in the eye should be immediately raised with scleral depression over the bleeding site. The operation is completed with scleral bucking with or without a gas injection, and the patient is positioned so that the blood settles away from the macula. In this situation, it is preferable not to drain the subretinal fluid any further since its presence prevents clotting of the subretinal blood. This makes it possible to move it away from the macular area by positioning of the patient since blood, being heavier settles down in a gravity dependent manner.

Retinal incarceration is observed following drainage in 2 to 3% of cases. This usually occurs with incisional sclerotomy technique, soon after penetration of the choroid, and is associated with a sudden cessation of drainage. Increased IOP contributes to this problem. When retinal incarceration is suspected, all traction upon the eye is eliminated and the fundus is quickly examined with indirect ophthalmoscopy. Usually the incarceration is mild, with only a localized depression in the retina surrounded by radiating striae. In this situation the drainage site should immediately be closed with the exoplant or

with a suture, and then the retina should be more thoroughly evaluated with scleral depression.

If retinal incarceration is detected, a wider piece of silicone buckle is placed beneath the sutures to support both the break(s) and the incarceration site or the band is moved anteriorly or posteriorly to cover the defect. If an iatrogenic retinal break is discovered, it is treated with light cryotherapy, and supported on a modified scleral buckle. In the rare circumstances in which formed vitreous passes into the penetration site through a large nearby tear or through a new retinotomy, the gel should be amputated at the sclerotomy site, which should be supported on an encircling buckle.

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REVIEW ARTICLE

Phacoemulsification in the Presence of Anterior Capsular Tears

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Abstract

Anterior capsular tears can occur during cataract surgery and, if not appropriately managed, can lead to more severe complications, which can adversely affect postoperative outcomes. Specific surgical techniques can minimize the risk of the posterior extension of the capsular tear and increase the likelihood of good patient outcomes. This review discusses the risk factors and mechanism of anterior capsular tears during phacoemulsification and presents techniques to prevent and manage them.

Keywords: Phacoemulsification, anterior capsular tear, cataract surgery

Introduction

Anterior capsular tears can occur during cataract surgery, and the operating surgeon must be prepared to address this complication to ensure a good postoperative outcome. The incidence of anterior capsular tears has been reported to occur between 0.5 and 5% in cataract surgeries [1-3]. Failure to correctly manage an anterior capsular tear can lead to additional intraoperative complications, including posterior capsular rupture (PCR), vitreous loss, nucleus drop, suboptimal intraocular lens (IOL) placement, or postoperative decentration of IOL [4]. In a study by Carifi G et al., the conversion rate to a non-phaco technique was 24%, nucleus drop occurred in 5%, and over 11% of eyes underwent unplanned secondary surgical procedures [5].

Timing and Mechanism of Anterior Capsular Tears

1. Spontaneous

Pre-existing anterior capsular tear or spontaneous ruptures have been reported with hypermature or intumescent cataracts, anterior polar cataracts, anterior lenticonus, steroid-induced cataracts, or occult trauma.[6] Spontaneous rupture is rare otherwise, as the anterior capsule is thicker than the posterior capsule, more elastic and gradually expands with increasing nuclear sclerosis and cataract development.

2. During Capsulorhexis – *Primary Tear Radialized anterior capsulotomy*

This occurs as a result of high posterior or intralenticular pressure. Obvious causes for posterior pressure or vitreous up thrust include tight eyelid speculum, excessive use of the peribulbar anaesthetic solution for the block, pressure on the globe or a silicone oil-filled globe [7]. Capsulorrhexis tears are more common when the anterior capsule is highly convex, such as in patients with hypermetropia or in the presence of a shallow anterior chamber. Large capsulotomy can approach the more convex edge of the capsular bag, causing the capsulorhexis to radialize. Anterior extension of zonules or pseudoexfoliation material may alter the vector forces and cause radial extension. Intumescent white cataracts are notorious for causing radialization of the anterior capsulorhexis, resulting in the Argentinian flag sign (Figure 1) caused by the intralenticular pressure from the liquified cortex. This can lead to a rapid and uncontrollable posterior extension of the tear.

Figure 1. Intraoperative photograph showing anterior capsular split (Argentinian flag sign) in a hypermature cataract.

Manual vs Automated Capsulorhexis and Guided Devices

An ideal anterior capsulotomy is of adequate size, continuous, circular, well-centred, and overlaps the implanted IOL around its circumference by 1 to 1.5 mm. Manual continuous curvilinear capsulorhexis (CCC) in conventional phacoemulsification cataract surgery (CPCS) and femtosecond laser-assisted capsulotomy in femtosecond laser-assisted cataract surgery(FLACS) are the two most recommended techniques for achieving anterior capsulotomy for phacoemulsification. Although meta-analyses have shown that both techniques provide similar results for long-term cataract surgery outcomes, there is ample evidence to suggest that femtosecond laser-assisted capsulotomy is more accurate in size, shape, and centration, improving the accuracy of IOL positioning within the capsular bag compared to manual CCC [8-10]. Several alternative methods for anterior capsulotomy have also been developed, which aim to provide some of the advantages of laser capsulotomy at a lower cost. Among these, Zepto precision pulse capsulotomy (PPC) and selective laser capsulotomy (SLC) CAPSULaser selective capsulotomy laser (EXCEL-LENS, Inc.) have been investigated the most in the literature so far [11]. New data suggests that precision pulse capsulotomy may create a smoother capsulorhexis than both femtosecond laser-assisted cataract surgery and manual continuous curvilinear capsulorhexis [12-13]. These technological advances can help surgeons achieve an ideal and intact capsulorhexis in complex situations where rhexis runaway is anticipated.

During Phacoemulsification – Secondary Tear

The operating surgeon can inadvertently sculpt the capsulorhexis margin while creating the central trench during phacoemulsification. An anterior capsular tear can also occur during nuclear disassembly (cracking or chopping manoeuvres) if the chopper is positioned improperly above the capsule, especially when the rhexis is small or without clear visualization.

Prevention

Risk factors

Pseudoexfoliation (PXF) creates a higher risk for anterior tears and a more difficult capsulorhexis due to poor dilation, fragile capsules and deviation of vector forces [14].

Surgeons performing capsulorhexis in PXF may encounter a capsule splitting phenomenon in which the false anterior layers being fragile, tear abnormally compared to the underlying true anterior capsule. A small capsulorhexis may lead to an increased risk of anterior capsular tear and a higher incidence of postoperative capsular phimosis [15].

White Cataract

In an eye with an intumescent white cataract, high lenticular pressure can cause an Argentinian flag sign, where the initial capsular tear propagates rapidly to the periphery of the capsule [16-17]. Figueiredo et al. postulated that liquefied cortex exists both anterior and posterior to a large nucleus, preventing communication between the two compartments. Hence both these compartments must be decompressed by first creating a small puncture on the anterior capsule, aspirating the liquid cortex, followed by gentle retropulsion of the nucleus to decompress the posterior compartment [18].

Paediatric Cataract

The anterior lens capsule in children is strong and highly elastic and requires the application of more force to puncture before tearing begins. This coupled with the vitreous up thrust, makes manual CCC more challenging to perform and control in paediatric eyes [19-20].

Uveitic Cataract

In uveitic cataracts, separation of adherent synechiae off the anterior capsule and manipulation of the iris tissue to achieve pupillary dilation can sometimes disrupt the anterior capsule [21]. If unrecognized, this may cause a capsular tear that can extend progressively during the course of surgery.

It is essential to ensure good visibility by focusing the microscope on the anterior capsule during CCC and using trypan blue to stain the capsule whenever necessary. Adequate size rhexis of 5-5.5 mm is important. Good surgical practices include maintenance of the deep anterior chamber and judicious and minimalist lateral separation of nucleus fragments.

Management Options

Continuous curvilinear capsulorhexis is paramount for a safe phacoemulsification cataract surgery. The management options in compromised rhexis situations depend on the surgeon's experience, type of cataract, location and timing of the tear out, and the presence or absence of significant co-morbidities .When there is a lack of expertise, it is safer to hand it over to a more experienced surgeon. There are principally three choices; conversion to a non-phacoemulsification nuclear extraction like manual small incision cataract surgery or conventional extracapsular extraction by enlarging the incision, performing supracapsular phacoemulsification or continuing with endocapsular surgery. If the tear is large and extended to the periphery in a

patient with dense nuclear sclerosis and other comorbidities, it is advisable to convert to a non-phacoemulsification technique to prevent the risk of nucleus drop. The safest technique possible should be opted for with the available instruments and viscoelastics. The endocapsular and supracapsular phacoemulsification can be continued for patients with softer cataracts and no other comorbidities.

Enlarging the Capsulotomy

- If a tear is identified early in a case, the bottle height can be reduced, and infusion pressure should be lowered immediately to reduce stress on the capsular bag and prevent the posterior extension of the tear.
- When a capsulorhexis begins to extend peripherally, the surgeon should immediately attempt to flatten the anterior capsular plane and relieve the zonular stress with the most cohesive ophthalmic viscosurgical device (OVD) available. The OVD must be injected peripheral to the torn area.
- If radialization of the capsulorhexis occurs, it is often possible to retrieve the edge with a rescue manoeuvre described by Little et al. in which the capsulorhexis edge is tensioned by pulling it centrally back along the capsular plane in the direction from which it originated. [22]
- If the radialized tear is in a difficult position and hard to reach effectively with the standard Utrata forceps through the main incision, a micro scissor and forceps may be introduced through a side port incision to redirect and enlarge it by approaching at the optimal angle (Figure 2 a & b). Redundant capsular flaps can be trimmed to prevent entrapment in phaco or irrigation-aspiration (I/A tip).

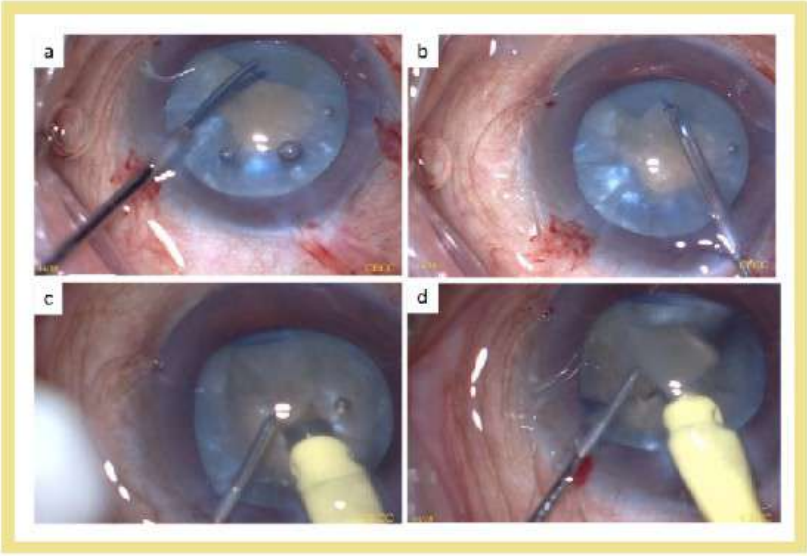

Figure 2. Intraoperative photograph showing a) the enlargement of capsulotomy using a microscissor and b) microforceps, c) Vertical chop following a high power sculpting, and d) anterior plane phacoemulsification of fragments.

- **Opposite Cut to Rhexis Margin** - An attempt may be made to complete the capsulotomy from the opposite direction, but once a tear is truly "lost," an additional opposite cut to the rhexis margin can be created in the anterior capsule, thereby redistributing stress on the capsular margin [23]. The relieving cut is also helpful when the surgeon decides to prolapse the nucleus to the supracapsular plane. Preventing a wrap-around tear is the surgeon's primary goal if a radialized tear is lost to recovery.

Hydrosteps

- In the presence of an anterior capsular tear, any attempt at hydrodissection stands a very high chance of extending the capsular tear around the equator.
- Hydrodissection and hydrodelineation should be performed with very small aliquots of balanced salt solution in a controlled fashion. The nucleus should rise up only a tiny bit before relieving the forces of the capsular block by gently tapping it back down again. Nuclear rotation must be avoided.

Maintain AC Depth and use of Dispersive OVD

To prevent the extension of a tear to the posterior capsule, surgeons should avoid sudden changes in pressure from above and below the anterior capsule. Liberal use of OVD is injected before phaco, or I/A probe is withdrawn to maintain the anterior chamber and prevent its sudden collapse. A copious amount of dispersive OVD is also injected to coat and protect the endothelium if the nuclear emulsification continues at an anterior plane.

Nucleus Management

The nucleus management can be done using a non-phacoemulsification technique by converting to a manual small incision cataract surgery or conventional extracapsular cataract surgery. If the surgeon is experienced, phacoemulsification can be continued.

Phacoemulsification

Gentle preliminary "High" power sculpting is useful to create the central trench without movement of the nucleus and sufficiently debulk the nucleus. During nuclear disassembly, techniques that require frequent rotation of the lens nucleus should be avoided to reduce stress on the anterior and posterior capsules and zonules, especially in the area of the tear. Vertical chopping with a chopper or Sinskey hook is preferred over horizontal chopping [24]. Minimal lateral separation and gentle rotation of fragments must be done using the Sinsky hook so that extra force is not transmitted to the area of torn rhexis.

Any centrifugal forces will result in propagation of the tear to the equator and beyond. Care must be taken to stay away from the rhexis tear. Anterior plane phacoemulsification of the nuclear fragments with low fluidics parameters can be preferred in this setting. (Figure 2 c & d)

Cortex Aspiration

During cortical removal, it is wise to remove the cortex in the area of the anterior capsular tear last. The cortex should be pulled gently and toward rather than away from the location of the tear to lessen the stress on the capsular bag and prevent a posterior wrap-around of the tear.

Recognition of Wrap-Around Posterior Capsular Tear, Vitreous Prolapse and Management

- In a study, the extension of the tear through the posterior capsule occurred in almost half the eyes with an anterior capsule tear, often requiring an anterior vitrectomy [1].
- Rohit Om Prakash et al. described the flap motility sign to differentiate a pre-equatorial from a wrap-around post-equatorial tear. Everted and fluttering flaps of the anterior capsular tears indicate pre-equatorial tear, while inverted and non-fluttering flaps indicate posterior capsule rupture following tear extension beyond the equator [25].
- A sudden unexplained deepening of the chamber with momentary pupillary dilatation or a sudden increase in the difficulty of manipulating the nuclear fragments indicate that the radial tear has progressed around the equator and that the posterior capsule's integrity has been compromised. More obvious signs include tipping, lateral, or downward movement of the nucleus. The wound must be enlarged, and the nucleus is manually delivered. Automated anterior vitrectomy should be followed by low flow cortex aspiration. As the wrap-around posterior capsular tear cannot be converted into a posterior curvilinear capsulorhexis, sulcus implantation is opted in these circumstances.

Intraocular Lens Management

Single piece endocapsular implantation of IOL

- Endocapsular IOL fixation is preferred more frequently in cases of secondary tear, and sulcus fixation is used more often in primary anterior capsule tear cases [4].
- Compared to a three-piece IOL, a one-piece IOL can be injected into the capsular bag in a more controlled manner, and it tends to unfold more slowly. The IOL is then rotated gently so that the haptics are oriented

perpendicular to the area of the anterior capsular tear. This minimizes stress on that area and avoids further propagation of the tear and also ensures better centration of the lens.

Three-Piece Hydrophobic IOL in Sulcus

- Even in the presence of an apparently intact posterior capsule, sulcus implantation may be opted as the tear can enlarge during IOL insertion into the capsular bag, with the risk for posterior dislocation [26]. Also, there is a risk for late postoperative decentration of the IOL as the remnants of the anterior and posterior capsules contract [27].
- If the integrity of the posterior capsule is in doubt, a three-piece IOL is chosen, and it should be placed in the sulcus with its haptics located away from the area of the anterior capsular tear. Optic capture can be performed with haptics perpendicular to the rhexis tear to prevent postoperative decentration **(Figure 3).**

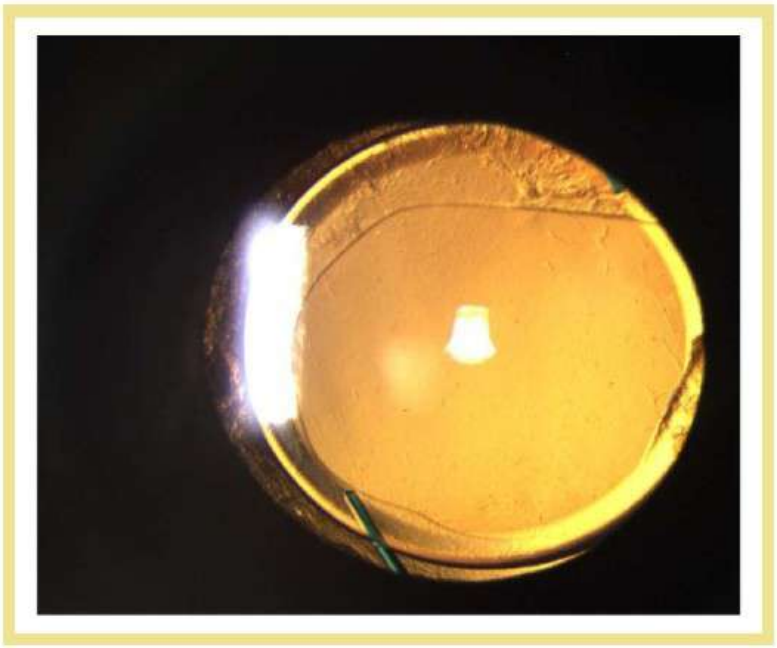

Figure 3. Postoperative slit lamp photograph of a three-piece IOL in the sulcus with optic capture and haptics oriented perpendicular to the rhexis tear.

OVD should be removed slowly to prevent fluctuations in AC fluidics which can cause an anterior capsular tear to extend posteriorly. Consideration may be given to suturing the main wound to allow the AC to remain formed postoperatively. A miotic agent injected intracamerally may help prevent a pupillary capture of the optic.

- A Toric or Multifocal intraocular lens can be implanted if the anterior capsular tear is small and pre-equatorial in location.

Conclusion

Anterior capsular tears can occur at any stage of cataract surgery like capsulorhexis, nuclear sculpting or chopping, and cortical removal. The proper management of these tears by lowering the infusion pressure, avoiding excessive rotation of the lens, and pulling the cortex toward the area of the tear, along with proper maintenance of intraocular milieu and nucleus management strategy,will prevent them from extending into the posterior capsule and compromising the structural integrity of the capsular bag. This is essential for proper intraocular lens placement and optimal visual outcomes for the patient.

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REVIEW ARTICLE

Retinal Vasculitis: An Update With Our Experience

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Introduction:

Retinal vasculitis is a sight-threatening inflammatory eye condition that involves the retinal vessels. It may occur as an isolated idiopathic condition, as a complication of infective or neoplastic disorders, or in association with systemic inflammatory disease.[1]

While rheumatologist classifies vasculitis on the basis of the size of the vessel affected, its location, and the associated histological changes, an ophthalmologist diagnoses retinal vasculitis on the basis of perivascular infiltrates, intraretinal hemorrhage, or a cotton wool spot indicative of local retinal ischemia.[2]Retinal Vasculitis is inflammation of the retinal vessels located in the inner plexiform layer of the retina.[Figure 1]

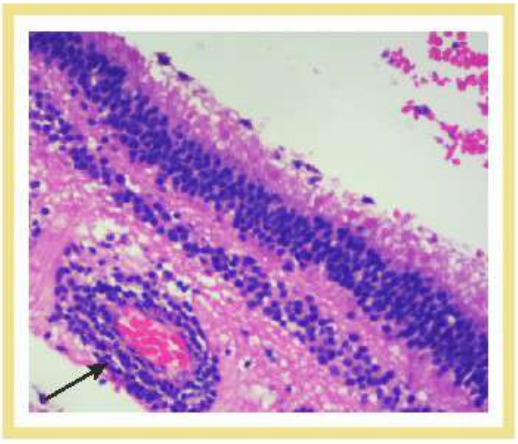


Figure 1:Histopathology slide showing inflammatory cells around blood vessel (arrow) suggestive of vasculitis.

Angiography is suggestive of retinal vasculitis when fluorescein stains the vessel wall or leaks beyond the vessel indicating increased vascular permeability. [1]

While the pathogenesis is often multifactorial, cell mediated immunity has a role as CD4+ T-cells are documented within and around the retinal layers. However, a biopsy is rarely done due to the potential of retinal damage. The associated features of vasculitis such as vascular occlusion can contribute to

increased vascular endothelial growth factor (VEGF) levels, further increasing the capillary permeability and increasing the severity.[3]

Diagnosis of the cause of these vasculitis entities still remains a challenge. Recently, with the advent of Optos imaging, wide field angiography , Swept source Optical coherence tomography and Optical coherence tomography angiography, newer insights of these pathologies have come into the armamentarium.

This necessitates us to update our knowledge on existing trends in understanding and management of these disease entities . This review focuses on some of the basic concepts relative to retinal vasculitis, relevant investigations and treatment options as described in recent literature and accepted in clinical practice.

Methods

A search of Medline database (2000-2020) was conducted. The following keywords were used: *retinal vasculitis, Eales disease, vitreous haemorrhage.* Additional sources included publications cited in other articles. Relevant articles were reviewed and included. One of the author's ophthalmic registry was used for photographs and images

Overview

Clinically occlusive retinal vasculitis affecting the retinal arterioles may cause cotton-wool spots representing micro-infarcts of the retina, seen commonly with Systemic Lupus Erythematosus, Polyarteritis Nodosa, Granulomatosis with Polyangiitis, Churg-Strauss syndrome, and cryoglobulinemia.[4]

Central and branch retinal artery occlusions are also reported. Inflammatory branch retinal vein occlusion is seen with Behçet's disease, tuberculosis and recently suspected to be associated with ongoing Covid-19 pandemic.[5]

Occlusive periphlebitis can cause retinal edema, intra-retinal hemorrhages, and hemorrhagic infarction of the retina. Late changes secondary to vascular occlusion and remodeling include telangiectasis, microaneurysms, and ischemia-induced neovascularization, with sequelae such as recurrent vitreous hemorrhage, traction retinal detachment, rubeosis iridis, and neovascular glaucoma .[4]

Intra-retinal infiltrates are noted with Behçet's disease, rickettsial infection, cat-scratch disease. Necrotising retinitis is seen in ocular toxoplasmosis, acute retinal necrosis, cytomegalovirus retinitis. Aneurysmal dilatations of the retinal and optic nerve head arterioles are associated with Idiopathic retinitis, vasculitis, Aneurysms and Neuro-retinitis and sarcoidosis. Frosted branch

angiitis in vasculitis in Viral retinitis, toxoplasma retino-choroiditis, SLE, Sympathetic ophthalmitis, infiltration with malignant cells (lymphoma or leukemia), SLE. Retinal ischemia that is noted as a consequence can be peripheral , or widespread involving the posterior pole contributing to poor visual outcome despite adequate therapy.[4]

Etiology of retinal vasculitis [3]

Retinal vasculitis can occur due to several causes. List of the causes are given according to the type of vessel involvement [refer table no.1] and classification of retinal vasculitis based on etiology [refer table no.2]

Table 1: Cause of retinal vasculitis according to the type of vessel involved and association with retinal ischaemia.

	Mainly involve arteries	Mainly involve veins	Associated retinal ischemia
Infectious disorders	Acute retinal necrosis Toxoplasmosis Cat scratch disease West Nile virus	Tuberculous hypersensitivity Syphilis CMV HIV Rift Valley fever virus	Tuberculous hypersensitivity West Nile virus
Non-infectious disorders	SLE Takayasu's disease IRVAN GPA Churg-Strauss syndrome Crohn's disease Polyarteritis nodosa Susac syndrome Dermatomyositis	Behçet's disease Sarcoidosis Multiple sclerosis Birdshot chorioretinopathy Acute Posterior Multifocal Placoid Pigment Epitheliopathy Pars planitis HLAB27 associated uveitis	Behçet's disease Sarcoidosis Multiple sclerosis SLE APHA Takayasu's disease IRVAN GPA Dermatomyositis Churg-Strauss syndrome Crohn's disease Polyarteritis nodosa Susac syndrome Idiopathic retinal vasculitis

Table 2: Classification of retinal vasculitis based on etiology

Primary	Uveitis etiology	Collagen vascular disease	Infections	Neurological disease
Eales	Sarcoidosis	SLE	Toxoplasmosis	Multiple sclerosis
IRVAN	Behcet's disease	Polyarteritis Nodosa	Tuberculosis	Susac syndrome
	CMV Retinitis	Behcet's disease	Syphilis	Central Nervous System lymphoma
	Intermediate Uveitis	ANCA associated vasculitis	CMV Retinitis	
	Acute Retinal necrosis	Immune complex mediated vasculitis	HIV	
			Rubella	
			HSV 1&2, VZV 1&2	
			Rickettsial	
			Epstein Barr	

Investigations

Fluorescein angiogram forms the basis of diagnosis and management of vasculitis. It is a more sensitive technique and will frequently show that the vasculitis is more extensive than the clinical examination .

Characteristic features seen with fluorescein angiography in active vasculitis include leakage of dye due to breakdown of the inner blood-retinal barrier, and staining of the blood vessel wall. Such leakage may be focal as seen in sarcoidosis or multiple sclerosis or more diffuse, as seen in Behçet's disease and Eales disease.It is very useful to delineate areas of capillary non-perfusion, and neovascularization secondary to retinal ischemia. It is also very valuable to diagnose the presence of inflammatory branch retinal vein occlusion.[4]

In our experience with retinal vasculitis, capillary non-perfusion was the most common fundus fluorescence angiography (FFA) finding seen in retinal vasculitis, found in 40% of the cases, followed by collateral vessels, seen in 19.5% of eyes with vasculitis.[7]

Widefield angiography helps in identification of location and extent of retinal vascular leakage. Indocyanine Green Angiography can provide additional information regarding choroidal ischemia and it can image hypo-fluorescent satellite lesions which are thought to be a non-infectious, inflammatory reaction.

Ultra-wide field retinal imaging or Optos can provide a wider view of approximately 200°, which covers 82% of the retina in a single image. [8]

Fundus Autofluoroscence (FAF) is valuable for the detection of subtle inflammatory change and monitor treatment response in uveitis patient with chorioretinal lesion.

The presence of complications associated with retinal vasculitis including cystoid macular edema (CME) and retinal neovascularization . CME can be better imaged with swept source Optical coherence tomography.[2]

Enhanced depth imaging OCT is suitable to visualize choroidal granulomas and to describe their characteristics.[9]

OCT angiogram is the newest modality offering non-invasive vascular imaging. Despite its limitation of not being able to show leaks or inability to image the periphery , it has emerged as a useful tool in delineating posterior capillary non perfusion areas and neovascularisation along with cystoid macular edema .[10] Figure-2 shows few images demonstrating its utility

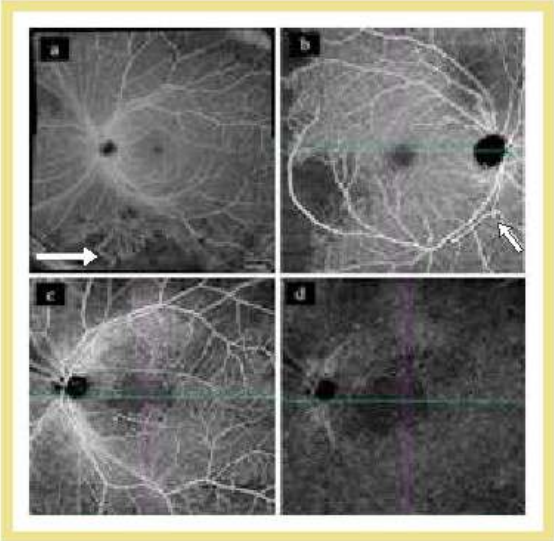


Figure 2 Use of Swept Source OCTA in various vasculitis conditions. a-Case of Eales disease with Neovascularisation (arrow) and capillary non-perfusion areas anterior to it b-Case of IRVAN with kinking of vessels (arrow) and capillary non perfusion inferno-temporal to fovea. c,d- Superficial and deep capillary plexus showing enlarged deficit in a patient of SLE.

Ultrasonography is required when vitreous inflammation or haemmorhage obscures view of the fundus. Findings associated with Toxoplasma retino-choroiditis are punctiform echoes within the vitreous as well as thickening of the posterior hyaloid membrane. A partial or total posterior vitreous detachment and focal retinochoroidal thickening are commonly observed as well.[11]

The search for a cause in patients with retinal vasculitis involves a multidisciplinary approach. The laboratory work-up of a patient must be tailored on the basis of patient's medical history, review of systems, and ocular examination. The absence of any diagnostic clues from history makes idiopathic retinal vasculitis most likely. Besides ophthalmic investigations ,complete blood count, erythrocyte sedimentation rate, C-reactive protein (CRP), High resolution computed tomography (HRCT) chest, Mantoux, QuantiFERON TB gold, Venereal disease research laboratory (VDRL)/Fluorescent treponemal antibody absorption test (FTA-ABS)/ Rapid Plasma Reagin (RPR) test, HIV serology etc must be performed but in a stepwise fashion. [4]

There may be a requirement for Aqueous tap or vitreous biopsy for PCR , or Serum ELISA in specific cases.

Treatment

The aim of the treatment should be to obtain a rapid resolution of inflammation, reduce the frequency of attacks. The choice of therapy is based on the severity of the disease.

In cases of associated anterior uveitis, topical prednisolone drops are preferred which can be gradually tapered in 6-8 weeks. Step-wise tapering prevents relapses and exacerbations. Topical nonsteroidal anti-inflammatory drugs such as indomethacin, diclofenac, and flurbiprophen could be added to topical corticosteroids to potentiate the corticosteroid activity. Topical mydriatic and cycloplegic is added twice or three times a day (tropicamide 1%, cyclopentolate 1%, and phenylephrine 2.5% and 10%) in order to relieve photophobia, pain, and discomfort and prevent synechiae formation.[12-13]

Posterior segment involvement often requires the use of immunosuppressive or immunomodulating agents.[14]·

Although corticosteroids (CS) are still used for the treatment of acute inflammatory episodes. When a rapid response is desired, the most commonly used treatment protocol consists of 1 g/day intravenous (IV) pulse methylprednisolone for 3 consecuitive days, followed by high-dose oral prednisone (1 mg/kg/day) which is tapered gradually and reduced to the maintenance dose (≤7.5 mg) after active inflammation has been suppressed. [15]

Starting with a high oral dose (1-1.5 mg/kg) is another option. Immunosuppressive agent(s) should be started simultaneously and used in conjunction with corticosteroids until they take effect. Periocular or intravitreal CS can be used as an adjunctive therapy in cases where systemic CS cannot be used or an adequate response is not achieved and in refractory Cystoid macular edema. [16]

A bio-degradable intraocular implant containing dexamethasone given through the pars plana with a 22-gauge applicator has been approved for the treatment of posterior uveitis as it was effective in improving vision and macular edema in the majority of the patients with non-infectious causes of uveitis. [17]

A sustained-release fluocinolone acetonide implant has been approved in the United states for chronic non-infectious posterior uveitis since 2005. These patients eventually require cataract surgery and one-third require filtering surgery to control glaucoma refractory to topical medications. [18]

Systemic immunosuppressive agents

These agents are indicated in corticosteroid dependent or intolerant cases as a steroid sparing purpose. Commonly used antimetabolite agents for long-term control of systemic and ocular inflammation include methotrexate, mycophenolate mofetil, or azathioprine. Among T cell inhibitors, cyclosporine and tacrolimus, have shown efficacy in decreasing inflammation in uveitis. [19] Subconjunctival/intra-vitreal sirolimus has demonstrated dose dependent benefit. [20]

Biologic agents

Patients who are refractory to steroid-sparing agents often benefit from biologics. Biologic agents studied for the treatment of non infectious uveitis are Tumor Necrosis Factor (TNF)-α inhibitors, including infliximab, adalimumab, etanercept, golimumab and Certolizumab. Among TNF-α inhibitors, Adalimumab, is the only systemic non-corticosteroid agent which has been approved by the US-Food and Drug Administration (FDA) for the treatment of non-infectious uveitis. Jaffe et al. reported the result of a multinational phase 3 trial (VISUAL I) involving patients with active non-infectious intermediate uveitis, posterior uveitis, or pan-uveitis resistant to topical and systemic steroids. [21]

In this trial, adalimumab was found to be associated with a lower risk of uveitic flare or visual impairment demonstrated a steroid sparing effect. Nguyen et al. published another multi-center, double-masked, randomized, placebo-controlled phase 3 trial (VISUAL II) with adalimumab. [22] and showed that it significantly lowered the risk of uveitic flare or loss of visual acuity on corticosteroid withdrawal with inactive uveitis patients. Adalimumab also showed the efficacy for pediatric patients with anterior uveitis. [23]

Leyre et al. showed the effectiveness of adalimumab in refractory sarcoid uveitisin 17 patients.[24]

Due to limited long term safety and efficacy data, biologic therapy is reserved as secondary or tertiary treatment of uveitis associated with sarcoidosis.

Surgery

Surgical interventions may be needed in cases resistant to medical treatment to remove media opacity such as vitreous opacity and cataract. With the use of micro-incision vitrectomy surgery (MIVS) using smaller gauge instruments and wide-viewing system , there are improved outcomes of vitreo-retinal surgery which may be required in cases of retinal detachment which can occur as a sequelae of retinal necrosis. Individual types of retinal vasculitis with etiology , clinical features, ancillary and laboratory investigations with management outline , are described below

Eales Disease

The most common primary cause of Retinal Vasculitis is Eales disease. Periphlebitis is more common .The clinical spectrum is divided into various stages of inflammation, ischemia, neovascularisation and fibrovascular proliferation.[25]·The strong expression of VEGF in eyes with Eales' disease leads to neovascularization and recurrent hemorrhages.[26] Ultra-wide field images (Optos) and wide-field FA detected significantly more number of active vasculitis cases in Eales disease. [27] The etiology of Eales disease is very poorly understood with proposed immunogenic mechanisms. The most favoured etiology is hypersensitivity to tuberculoprotein. Possible mechanisms resulting in venous occlusion secondary to tuberculous inflammation or by a hypersensitivity reaction to M. tuberculosis antigen. [3] Nested polymerase chain reaction for the detection of M. tuberculosis genome in vitreous and epiretinal membrane of patients with Eales disease denotes its possible association of the Eales disease and tuberculosis.[28,] Gupta, P. et al observed that patients of Eales disease had one or more tests positive for Tuberculosis suggesting a pivotal role of TB as a primary etiology for this disease.[29]

Immunohistochemical studies of epiretinal membrane in Eales disease have demonstrated neovascularisation with lymphocytic infiltration with predominance of T-cells indicating role of cell mediated immunity triggered by sequestered M. tuberculosis antigen.[30] Higher frequency of human leukocyte antigen HLA-B5(51), CW1,DR1,DR4,DR52 were found indicating the role of such HLAs in immuno-pathogenesis of Eales disease.[31]

Management of Eales disease depends upon the stage of the disease. Oral or periocular steroids are indicated in active inflammatory perivasculitis stage. Patients using oral steroid during acute stage disease had significantly better visual outcome at the final visit as compared to patients who did not receive oral steroids and laser therapy had significantly better visual outcome at the

final visit as compared to those who did not undergo laser treatment[32]. Use of Intra-vitreal AntiVEGF agents and laser photocoagulation is indicated in cases of neovascularisation. Vitreo-retinal surgery in Eales is indicated in case of non resolving vitreous haemorrhage (of more than 3 months) and in tractional or combined rhegmatogenous and tractional retinal detachments. Clinical features and imaging of cases managed by us have been described in [Figure 3 and 4]

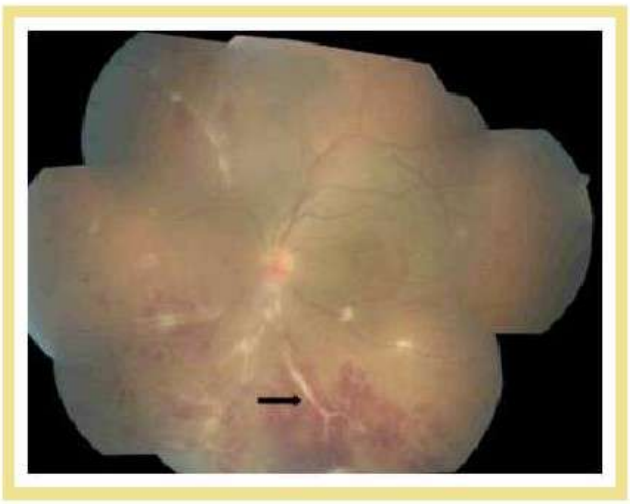


Figure 3 : Fundus photo of left eye with vasculitis (arrow) suggestive of periphlebitis. Patient was positive for Mantoux and quantiFERON TB gold. Diagnosis of Eales disease was made.

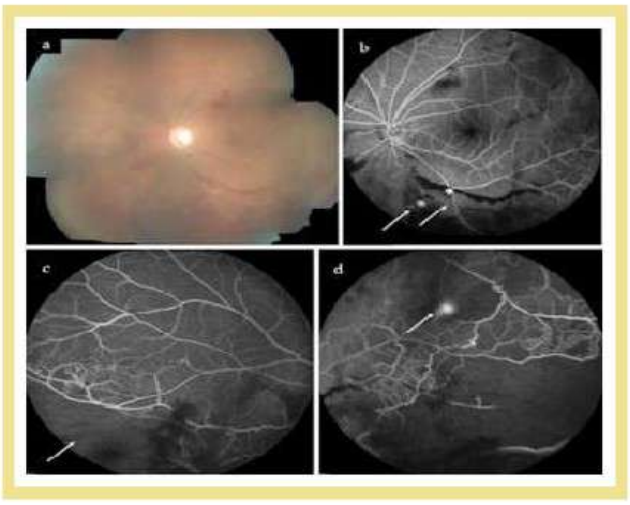


Figure 4 a- Fundus photo montage of a 31 years old male patient diagnosed to have Eales disease, 4b- Fluorescein angiogram of same patient showing leakage suggestive of NVE (arrow), 4c- Areas of peripheral capillary non perfusion(CNP) (arrow), 4d- Areas of peripheral capillary non perfusion areas, veno-venous communication, Neovascularisation elsewhere (arrow).

This patient was treated with fluorescein angiography guided laser to CNP areas under the cover of steroids The other eye of this patient had vitreous hemorrhage and was managed with Vitrectomy and endolaser.

Retinal Vasculitis and Behcet's Disease

It is a chronic multisystem disorder characterized by oral and genital ulcers, ocular inflammatory involvement, skin lesions, and vascular involvement. International classification of Behcet's disease is the most sensitive criteria for diagnosis/ classification [Table 3]

Table 3 : [33] International Study Group criteria for the diagnosis of Behçet's disease [9].

Recurrent oral ulceration	Minor/major aphthous or herpetiform ulcer observed by the physician or patient which recurred at least three times in one 12-month period
Plus two of the following:	
Recurrent genital ulceration	Aphthous ulcer or scarring observed by the physician or patient
Eye lesions	Anterior/posterior uveitis, cells in the vitreous on slit-lamp examination or retinal vasculitis observed by an ophthalmologist
Cutaneous lesions	Erythema nodosum observed by physician or patient, pseudofolliculitis or papulopustular lesions, or acneiform nodules observed by physician in post adolescent patients not receiving corticosteroids
Positive pathergy test	Interpreted by the physician at 24–48hr

Although its etiopathogenesis is not known, adaptive and innate immune systems, genetic predisposition, and environmental factors have all been implicated. It is more frequent and more severe in males in third and fourth decade of life. The most well-known genetic link is its association with HLA-B51.[34,35]Other implicated pathogenesis of the disease include abnormal cellular responses, T-cell-mediated immune responses, abnormal response to bacterial antigens, increased Th1 cytokine production, disorders of the complement system, up regulation of endothelial cell surface molecules, hemodynamics, and coagulation factor abnormalities [36].

Ocular involvement ranges from anterior, intermediate, posterior and panuveitis to obliterative and necrotising vasculitis, affecting both arteries and veins. The ocular involvement is the initial manifestation in 20% of the cases or may develop 2 to 3 years after the beginning of the extraocular signs. Anterior uveitis is always non granulomatous, sometimes associated with mobile

hypopyon. Posterior uveitis includes the presence of hyalitis, retinal vasculitis, (mainly venous,) central or branch retinal vein occlusion, attenuated or ghost vessels, neovascularisation at disc or elsewhere, macular edema, and/or foci of necrotizing retinitis. Tractional retinal detachment, retinal atrophy, papilloedema, and optic atrophy are late sequelae of the disease.[37]

The retina is the predominant affected site in posterior Behcet's, uveitis [figure5a]. Fluorescein Angiogram(FA) demonstrates fluorescein staining on the vessel wall and/or fluorescein leakage from the vessel even before clinical appearance. FA may reveal macular hyper-fluorescence, vascular leakage, capillary non-perfusion, occlusion of the retinal vessels, collateral formation, and neovascularization. "Fern-like fluorescein leakage" due to inflammation at retinal capillaries is demonstrated on the mid-phase FA which is a characteristic fluorescein angiographic appearance of Behçet's disease.[figure 5b].

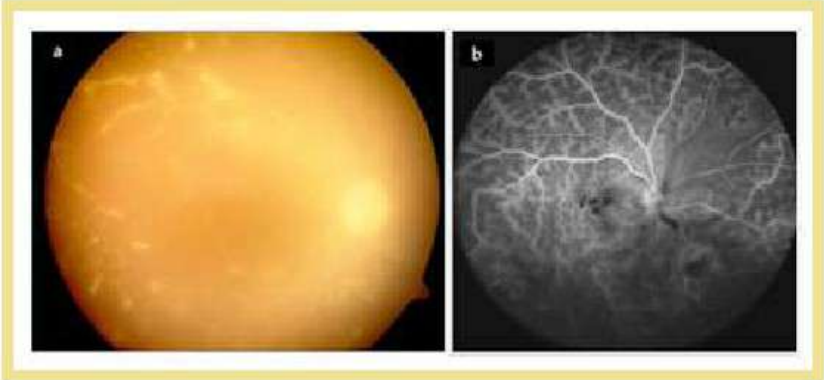


Figure 5a-Fundus photo of right eye showing periphlebitis causing occlusive vasculitis with optic atrophy, one of the manifestation of Behçet's disease. 5b-Fluoroscein Angiogram showing "Fern like appearance" in mid phase due to inflammation of retinal capillaries pathognomic of Behcet's disease.

ICG Angiography and FFA would be useful for examining choroidal involvement. Ultra wide field imaging has demonstrated that peripheral retinal vasculitis could be detected in 85% of eyes that did not have ophthalmoscopic evidence [38].

OCT provides both high-resolution cross-sectional imaging of the retina and quantitative measurement of the retinal thickness. For the detection and follow-up of macular edema in Behcet disease, OCT and FFA are necessary [39].

Combination therapy is required in most of the patients. Early and aggressive treatment should be administered whenever following features are present: male sex, young age, characteristic geographical origin, complete Behcet disease, posterior segment, bilateral and central nervous system involvement.

Experts recommend the use of anti-TNF agents like Infliximab(IFX) and adalimumab (ADA) as first-line treatment for Behcet uveitis. Single-dose IFX infusion was faster acting than IV or intra-vitreal CS in the suppression of acute episodes. IFX and ADA were compared, and it was found that both agents suppress uveitis, IFX has a fast-acting and potent anti-inflammatory effect equivalent to that of IV pulse methylprednisolone but should be combined with an antimetabolite due to its high immunogenicity whereas ADA is more effective at inducing sustained remission and is safer and more appropriate as monotherapy. IFX is administered IV in hospital conditions, while ADA is administered subcutaneously.[41]

In cases where an adequate response is not achieved even with biologic agents, the current biologic agent should be increased in dose and/or frequency or treatment should be switched to an alternative biologic. Promising drugs include Tocilizumab, an anti-interleukin-6 (IL-6) receptor antibody, golimumab, (anti-TNF-α) agent, IL-1 inhibitors anakinra and canakinumab. [42-44]

Retinal Vasculitis in Sarcoidosis

Ocular involvement has been observed in 25–60% of patients with systemic sarcoidosis. Retinal vasculitis in the form of multifocal periphlebitis has been reported in 37% of patients.[45] Retinal periphlebitis is a common ocular manifestation and was considered by the first International Workshop on Ocular Sarcoidosis as one of seven clinical signs.[46] Although ocular sarcoidosis is typically associated with non-obstructive vasculitis, ischemic retinal vasculitis has rarely been reported. Typical features include segmental cuffing or extensive sheathing and perivenous exudates, known as "candle wax drippings" associated with vasculitis [Figure 6a].

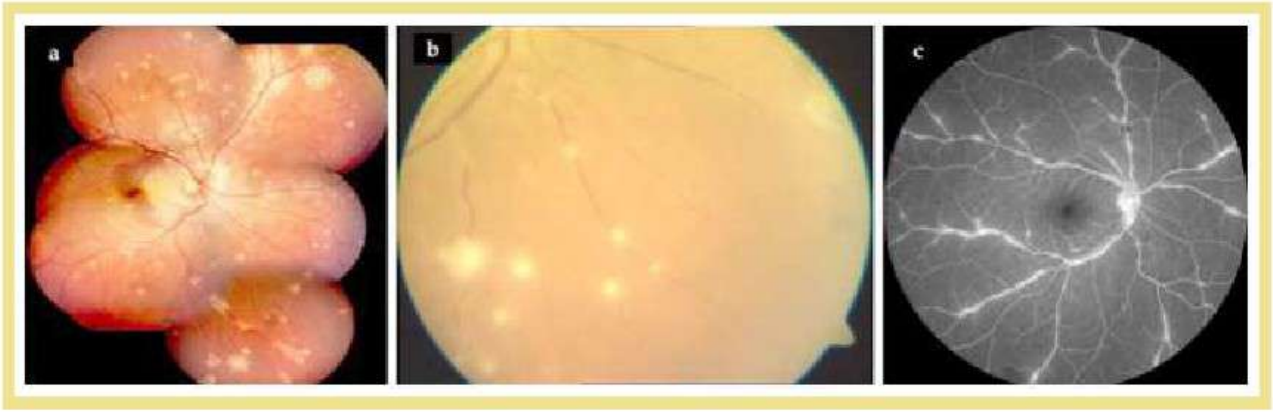


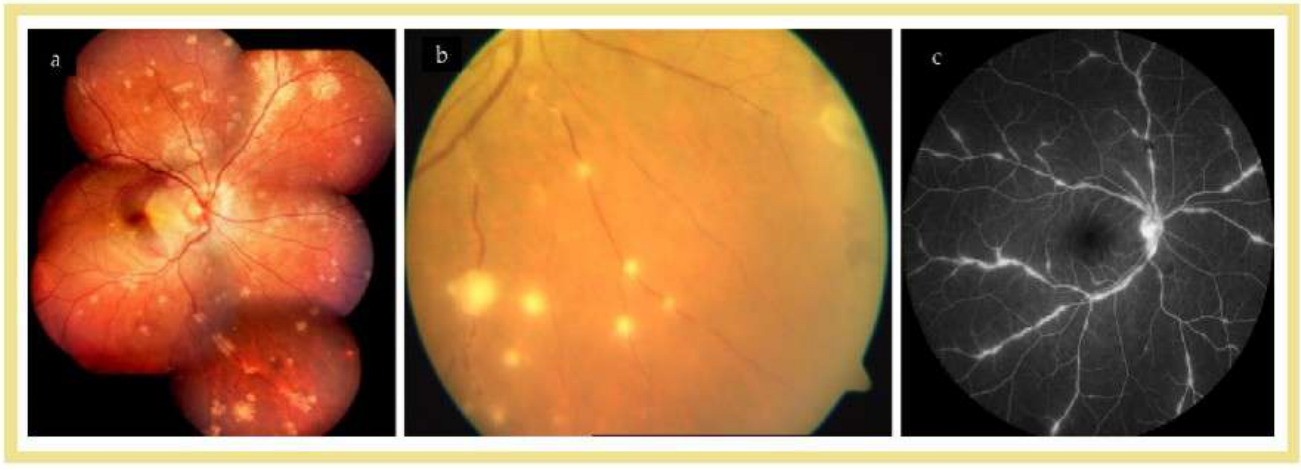
Figure 6: 6a-Fundus photo montage demonstrating retinal periphlebitis as a 'candle wax dripping sign' pathognomic of sarcoidosis vasculitis.

Diagnostic criteria for sarcoidosis associated uveitis have been proposed at first International workshop on ocular sarcoidosis (IWOS) in 2009.[46] [Table 4]

Table 4: Diagnostic criteria and terminologies for ocular sarcoidosis

Definite ocular sarcoidosis	Biopsy-proven with a compatible uveitis
Presumed ocular sarcoidosis	Biopsy not performed; bilateral hilar adenopathy (BHL) with compatible uveitis
Probable ocular sarcoidosis	Biopsy not performed and no BHL detected; presence of 3 suggestive ocular signs and 2 positive above laboratory tests
Possible ocular sarcoidosis	Biopsy negative; presence of 4 suggestive ocular signs and 2 positive above laboratory tests

Fundus photography for the uveitis patient with vitreous opacities may be helpful for the documentation and future comparisons. The area of apparent fluorescein leakage due to retinal vasculitis can be more diagnostic with wide field angiogram [Figure 6 b, c].[47]



Figures 6, b, and c: Widefield angiogram of the same patient demonstrating active vasculitis.

While serum ACE levels remain the most widely performed diagnostic test, elevated level of soluble interleukin 2 receptor (sIL2R) suggests sarcoidosis with uveitis more convincingly than ACE,·[48]. Significant lymphopenia ($<1.0\times10^9$/L) is an independent predictor of sarcoidosis.[49]

Visual prognosis and disease course can be assessed and monitored by quantifying retinal capillary changes in Optical Coherence Tomography Angiography[50].

Evaluation of inflammatory cells in anterior chamber of sarcoid uveitis by optical coherence tomography showed a predominantly mononuclear pattern. [51]

In limited cases, vitreous biopsy can be done for the diagnosis. Masaru et al. showed that high-mobility group box-1 (HMGB1), which is secreted by activated leukocytes and acts as a primary inflammatory cytokine was detected in the vitreous of 23 of 24 patients (95.8%) with ocular sarcoidosis and the level of HMGB was high as 52.5 ng /ml compared to proliferative diabetic retinopathy patients (85.2%, 9.84 ng/ml) and epiretinal membrane patients (66.7%, 6.99ng/ml). It is associated with vitreous levels of Th1 and regulatory T-related cytokines [52]

These investigations suggest that cytopathological examination of vitreous samples may contribute to the diagnosis of intraocular sarcoidosis. Goto H. et al suggested Propionibacterium acne as a possible pathogen of granuloma in ocular sarcoidosis patients by showing this organism in a removed epiretinal membrane.[53]

Siasos et al. reported that ocular involvement in sarcoidosis patients was associated with impaired endothelial function and revealed a possible clinical importance of the use of endothelial function tests [54].

Ocular inflammation (chorioretinal lesions) can be a marker of microcirculatory damage in sarcoidosis patients and choroidal involvement is associated with an increased risk of cardiac disease. The first-line treatment for ocular sarcoidosis is corticosteroids.

Vasculitis In Cytomegalovirus (CMV) Retinitis

CMV retinitis can present in various patterns: Fulminant: There are dense, white, well-demarcated areas of retinal necrosis with retinal haemorrhages, often described as a “pizza pie” appearance (figure 7a).

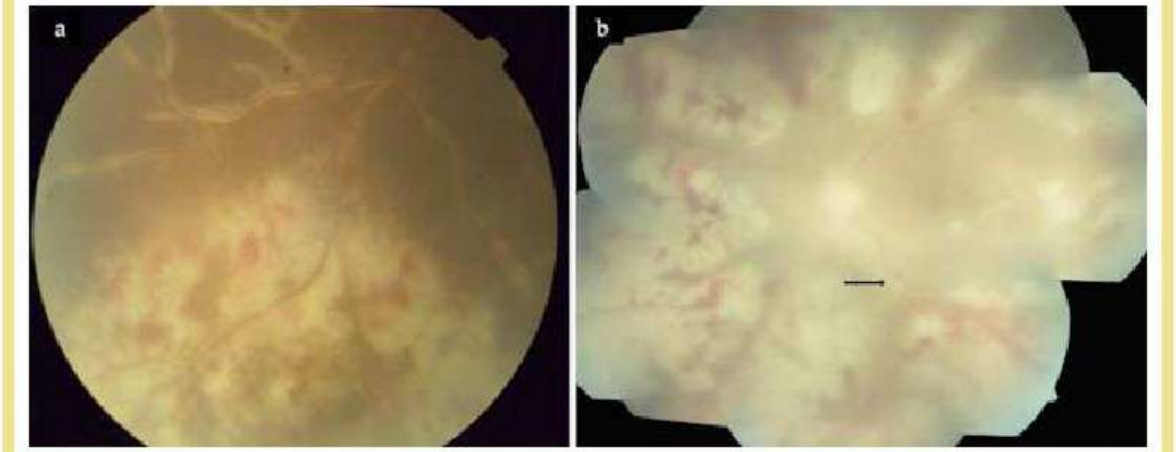


Figure 7; 7a-. Fundus photomontage demonstrating retinal necrosis with haemorrhages giving the appearance of 'pizza-pie' pathognomic of CMV Retinitis.7b-. Fundus photo montage of left eye demonstrating disc pallor with areas of peripheral necrotic retina with haemorrhages and vasculitis (arrow) in a case of acute retinal necrosis.

It tends to progress along the vessels in a 'bushfire-like' pattern.

Indolent: Mild, granular opacification of the retina with very few retinal haemorrhages, which starts in the retinal periphery and progresses slowly.

Frosted branch angiitis: The least common form, in which perivascular exudation is the most obvious[55-56]

Significant visual impairment is caused by retinal necrosis involving the macula or optic nerve. Peripheral disease can lead to retinal detachment.

In cases where the diagnosis is unclear, CMV polymerase chain reaction (PCR) may be performed on aqueous and vitreous samples. Cytomegalovirus (CMV) retinitis activity is accurately reflected by the presence and level of CMV DNA in aqueous humor and vitreous.[57]

Fundus photography may have utility for screening and monitoring CMV retinitis. [58-61]

Kyrieleis plaques (segmental retinal periarteritis) may be seen. [62]

Neovascularization is rare, but choroidal neovascularization and optic disc neovascularization have been reported.

Diagnosis is mainly clinical. Areas of active retinitis exhibit hypo-auto-fluorescence (especially the advances border suggestive of reactivation) sometimes with stippled hyper-Autofluoroscence, and areas of atrophy from healed retinitis exhibit hypo-Autofluoroscence[63]

Current drugs for the treatment of CMV infections are ganciclovir, valganciclovir, cidofovir and foscarnet and is summarised in table 5.

Table 5 [64]

Summary of the Existing Drugs for the Treatment of CMV Retinitis

Existing Antiviral Agents	Mechanism of Action	Mode of Delivery	Side Effects
Ganciclovir	Inhibits viral DNA synthesis	Intravenous formulation-Cytovene IV® (Roche) Oral formulation-Cytovene® (Roche) Intraocular implant-Vitrasert® (Bausch & Lomb)	Hematological abnormalities Poor oral bioavailability (5%) leading to insufficient viral suppression Infection in the contra-lateral eye and risk of retinal detachment
Valganciclovir	Inhibits viral DNA synthesis	Oral formulation-Valcyte® (Roche)	Myelo-suppression
Foscarnet	Blocks the cleavage of pyrophosphate from the terminal nucleoside triphosphate	Intravenous formulation-Foscavir® AstraZeneca LP)	Renal impairment
Cidofovir	Inhibits viral DNA synthesis	Intravenous formulation-Vistide® (Gilead Sciences)	Severe renal toxicity
Fomivirsen	Inhibits translation of early CMV proteins	Intravitreal injection-Virtanen® (Isis Pharmaceuticals)	Adverse ocular side effects such as uveitis

Retinal Vasculitis in Acute Retinal Necrosis (ARN)

The Executive Committee of the American Uveitis Society [65] defined ARN on the basis of the following clinical characteristics:

(1) 1 or more foci of retinal necrosis with discrete borders located in the peripheral retina,

(2) Rapid progression in the absence of antiviral therapy,

(3) Circumferential spread,

(4) Evidence of occlusive vasculopathy with arterial involvement,

(5) A prominent inflammatory reaction in the vitreous and anterior chamber.

Herpetic causes (mostly Varicella zoster virus (VZV) followed by herpes simplex virus (HSV) types 1 and 2) have been established by polymerase chain reaction (PCR)-based techniques, serum or intraocular fluid antibody testing, viral culture, retinal biopsy, and immunocytochemistry.[66]

Figure 6b.shows the clinical features of ARN .

Retinal detachment is the most common cause of decreased vision. Other causes include chronic vitritis, epiretinal membrane, macular ischemia, macular edema, and optic neuropathy.[67] Bilateral ARN occurs in up to 70% of untreated patients. Contra-lateral involvement usually occurs within a few months but may occur years later but has reduced with the advent of newer antivirals.[68]

Diagnosis is mostly clinical.

Histopathology, immuno-histochemistry and molecular biology of an enucleated globe of ARN, 6 years after the onset showed persistence of chronic inflammatory cells with herpes virus inclusion body. Semi nested polymerase chain reaction showed varicella zoster virus. [69]

Vasculitis in Toxoplasma Retinochoroiditis

Toxoplasma retinochoroiditis (TRC) is likely the most common cause of infectious retinochoroiditis worldwide. Patients may become infected in uteri or via ingestion of the parasite.

There may be sudden onset of floaters, vision loss, hazy vision, pain, and/or photophobia. Small, active peripheral lesions, may be asymptomatic. Retino-choroiditis appears as a yellow-white lesion with indistinct margins combined with an overlying focal vitreous infiltrate (described as "a head light in the fog") adjacent to an old chorioretinal scar.[figure 8a].

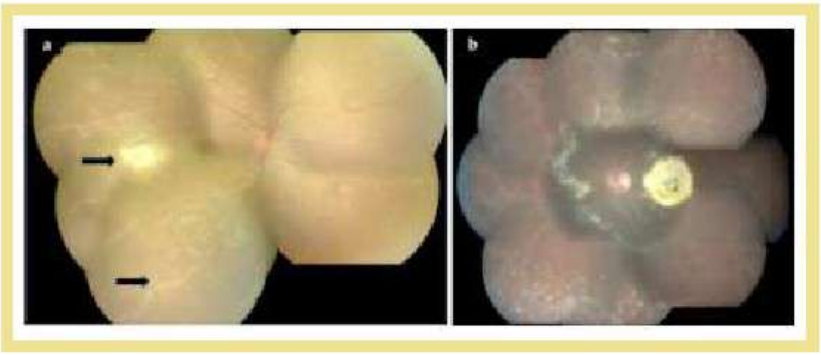


Figure 8: 8a-Fundus photo demonstrating solitary retinochoroiditis(arrow) patch temporal to fovea with kyrielesis arteritis(arrow). 8b-Fundus photo montage left eye indicative vasculitis associated with IRVAN. Localized perivascular plaques called Kyrieleis plaques are very common

Immunocompromised patients can have severe clinical picture and resemble acute retinal necrosis. Atypical cases may require either a vitreous or aqueous sample for anti-toxoplasma immunoglobulin G or A (IgG or IgA) antibodies. The classic triple-drug therapy of pyrimethamine, sulfadiazine, and corticosteroid is an effective choice.

Intra-vitreal injection of clindamycin with dexamethasone, or combination of azithromycin with pyrimethamine have been shown to be effective against ocular toxoplasmosis. The use of the trimethoprim-sulfamethoxazole combination is now preferred due to better patient compliance, faster resolution and is well tolerated for long-term prophylaxis. [71] Intra-vitreal injection of clindamycin (1mg/0.1ml) and dexamethasone (400ug/0.1ml) is an effective regimen of treatment for active toxoplasmic retinochoroiditis. [72]

Retinal Vasculitis in Ocular Syphilis

Syphilis, 'the great imitator' has extensive range of clinical manifestations Chorioretinitis with vitritis is the most common finding in syphilitic posterior uveitis. These lesions are believed to be the result of active inflammation of the choriocapillaris – pigment epithelial retinal photoreceptor complex.[73] Acute syphilitic posterior placoid chorioretinitis (ASPPC) is characterised by the presence of one or more yellowish, placoid, outer retinal lesions, typically at or near the macula, with a faded center and stipulation of the adjacent retinal pigment epithelium. [74] Ischemic retinal vasculitis can be a presentation. Non treponemal tests such as Rapid plasma Reagin tests and Venereal disease research laboratory test and treponemal tests such as fluorescent treponemal antibody absorption test can help in confirmation. Retinal lesions tend to heal with minimal disruption of the retinal pigment epithelium.

Retinal Vasculitis in SLE

Systemic lupus erythematosus(SLE) is a multisystem autoimmune disease of undefined etiology and with remarkably heterogeneous clinical features.

Ophthalmological examination and fundus fluorescein angiography (FFA) reveal sheathed or tortuous retinal vessels, papilledema, hemorrhagic or cotton- wool spots, multiple large vessel branch retinal artery occlusions and areas of capillary leakage and multiple capillary non perfusion areas.

The pathogenesis of SLE retinopathy, that has been ascribed to vasculitis include local micro-infarction and micro- embolism. About 23% of SLE patients have been associated retinal vasculitis. [75]

Irvan Syndrome

Idiopathic retinal vasculitis, aneurysms, and neuro-retinitis (IRVAN) syndrome is a rare clinical entity of unknown etiology.[76]

Arterial involvement is a common finding in cases of IRVAN [Figure 7b], which is associated with multiple aneurysmal dilatations of the retinal and optic nerve-head arterioles measuring 75–300μm in diameter, and are present at or near major branching sites on retinal arterioles.[77]

Samuel et al[76] classified the clinical features of IRVAN into major and minor criteria, and diagnosis is based on a constellation of these clinical features. The three major criteria are retinal vasculitis, aneurysmal dilations at arterial bifurcations, and neuro-retinitis, while minor criteria are peripheral capillary non-perfusion, retinal neovascularization, and macular exudation.

Post Pyrexia Retinal Vasculitis

Post pyrexial retinitis is a infectious or parainfectious uveitic entity caused by several viral or bacterial infections such as typhoid, dengue, west Nile, Chikungunya , Rickettsial and Zika virus infection. It may be direct invasion or immune mediated attributed to post infection immunologic effects or molecular mimicry leading to autoimmunity [78]

In 2015, case reports and small series added to the differential diagnosis for retinal vasculitis which was reported to occur subsequent to vaccination for influenza and in a patient who had both malaria and Dengue Fever [79]

COVID-19 infects the host using the angiotensin-converting enzyme 2 receptor, which is expressed in several organs including retinal endothelial cells[80]. Viral RNA of COVID-19 has been detected in the retina of affected patients suggesting that COVID-19 may cause retinal vasculitis and ischemia[81]

Takayasu's Arteritis

Takayasu's arteritis is a rare, systemic, large-vessel vasculitis. Impairment of vision is associated with involvement of the carotid and vertebral arteries and their branches that leads to decreased perfusion of the eye. Funduscopic evaluation revealed impaired circulation in retinal arteries and veins, and fundus fluorescein angiography showed segmented retinal blood flow (also known as "box-carring ")[82]

Susac Syndrome

Susac syndrome is characterized by the triad of encephalopathy, sensorineural hearing loss, and branch retinal artery occlusion. The etiology is unclear but it is believed to be an autoimmune-mediated endotheliopathy that affects the microvasculature of brain, retina and inner ear. [83]

Drug Induced Vasculitis

Rifabutin dose more than 300 mg/day causes anterior uveitis with or without hypopyon, intermediate uveitis, retinal vasculitis, or panuveitis [84]

Immune checkpoint inhibitors (ICPI) such as nivolumab (produces VKH like syndrome)and Pembrolizumab (placoid lesions) have been reported to develop retinal vasculitis as a part of uveitis spectrum.

Intravitreal injection of vancomycin as is done in some centers after cataract surgery has been rarely associated with haemmorhagic outer retinal vasculitis. [85].

Genetic Causes

Very rare genetic causes of retinal vascular inflammatory disease are now being recognized and may help better elucidate the pathogenesis of certain types of retinal vasculitis. ADNIV (autosomal dominant neovascular inflammatory vitreo-retinopathy), a condition that results in anterior chamber and vitreous inflammation, as well as retinal and iris neovascularization, is caused by mutations in Calpain 5 [86]

A novel Behçet's-like auto-inflammatory disease was recently reported due to mutations in TNFAIP 3 (tumor necrosis factor alpha-induced protein 3) leading to A20 haplo insufficiency and increased expression of NF-κB–mediated inflammatory cytokines [87]

In summary, the etiological diagnosis of retinal vasculitis may be challenging and tailored systemic investigations depending on suggestive clinical picture may be necessary. It is important to perform a thorough examination of the other eye and also document the findings with newer imaging modalities.

In few cases, the diagnosis of vasculitis may uncover an unknown underlying systemic pathology. Early, effective and prompt management may be vision saving and contribute to minimizing comorbidities.

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CLINICAL STUDY

Membranectomy with Secondary Intraocular lens Implantation with Posterior Optic Capture in Paediatric Aphakia

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Abstract

Background: Visual axis opacification (VAO), also referred to posterior capsule opacification, is a major concern in paediatric cataract surgery, especially in infants, which would cause postoperative vision loss. Rapid opacification, thickening and fibrosis of the posterior capsule and soemmering's ring formation are often seen in young children after cataract surgery. Thus, management techniques of posterior capsule and decision of intraocular lens implantation in pediatric cataract are different from that in adult cataract surgery. The purpose of this article is to investigate a surgical technique of membranectomy with secondary intraocular lens (IOL) optic capture in paediatric aphakia cases to reduce the incidence of visual axis opacification and late decentration of intraocular lenses.

Methods

In a prospective evaluation of eyes in which primary lens aspiration was performed for pediatric cataract at an earlier date, central membranectomy was done using vitrectomy head andsecondary IOL optic capture was performed posteriorly with haptics in sulcus as a surgical technique for preventing further visual axis opacification and decenteration of IOL. Perioperative complications and incidence of secondary cataract are presented in this manuscript.

Results

In 10 consecutive eyes operated in children with mean age of 62.6 months (range 42-102 months), there was no opacification of the visual axis or IOL decentration at a mean follow-up of12.4 months (Range 8- 18 months).

Conclusion

Membranectomy with optic capture holds promise as a technique for preventing secondary membrane formation and better IOL stabilization in pediatric eyes with aphakia and secondary membrane formation. This maneuver can be used to create an opening of desired size in a controlled manner with the help of an automated vitrector and ensures centration of the posterior chamber intraocular lens.

Financial Disclosure: No author has a financial or proprietary interest in any material or method mentioned.

Introduction

The use of intraocular lenses (IOLs) to correct pediatric aphakia has become increasingly common in recent years and is now regarded as a well-accepted approach for children beyond infancy.[1]Visual axis opacification (VAO), also known as posterior capsule opacification (PCO) or after-cataract, can occur despite a primary posterior capsulectomy and anterior vitrectomy. In infants, this complication is much more common when an IOL is implanted than in cases of primary aphakia.[1,2]According to the literature, the mean VAO rate with primary posterior capsulotomy, vitrectomy, and hydrophobic acrylic IOL implantation is 44.0%, ranging from 8.1% in all children younger than 2 years at surgery to 80.0% when children younger than 5 months were included.[3]

The difficulties with inflammation occurring after pediatric IOL implantation surgery, as well as its association with secondary membrane formation, iridolenticular adhesions, and IOL decentration, are well known to pediatric IOL surgeons despite all preventive measures. Gimbel and DeBroff[7] reported that placing the optic behind the primary posterior continuous capsulorhexis (PCCC) and fixating the haptics in the bag (optic capture through posterior capsulorhexis) keeps the optical axis clear after cataract surgery in pediatric eyes. They believed this technique may allow surgeons to avoid planned anterior vitrectomy in children and minimize the risk of posterior capsule opacification (PCO). Based on this assumption, surgeons all over the world are going on for primary PCCC with optic capture.

This paper describes the surgical technique of membranectomy with secondary IOL implantation and posterior optic capture in the treatment of paediatric aphakia.

Patients and Methods

Ten eyes of ten children, ranging in age from 3 years to 10 years, who originally had aphakia after cataract extraction with visual axis opacification were operated on with the intent to clear the visual axis and to implant an intraocular lens (IOL) for visual rehabilitation. Secondary IOL implantation was performed because of contact lens intolerance or poor compliance to glasses and visual axis opacification. All patients had congenital or infantile cataracts removed within the first 12 months of life. Each patient had undergone a primary anterior and posterior capsulotomy along with an anterior-vitrectomy. Patients were chosen for in the sulcus secondary implantation because the posterior and anterior capsules were fused.

Surgical Technique

Under general anaesthesia, two stab incisions were made with a microvitreoretinal blade. Using a viscoelastic cannula, the posterior synechiae attaching the iris to the capsular remnants were broken. A dense, white ring made of the fused edges of the anterior and posterior capsule visible in the center of the pupillary space was dissected. The sommering ring was aspirated from within the doughnut-shaped capsular bag remnant using the 23G vitrector in the irrigation-aspiration & cut mode or a bimanual irrigation/ aspiration handpiece. Care was taken to remove the cortical material as completely as possible. A viscoelastic substance was injected to form the sulcus. The anterior capsular edge was identified for 360°. A vitrector was used to cut the membrane to create a central opening of nearly 4.5 – 5.0 mm and a limited anterior vitrectomy was done.

A limbal corneal tunnel of 3.2 mm was made. A multi-piece foldable hydrophobic acrylic IOL was implanted in the sulcus. The inferior edge of the optic was slipped under the PCCC border with a Sinskey hook. The same maneuver was performed with the superior edge. Thus, the IOL optic was positioned behind the PCC while the haptics remained in the sulcus. If the round opening was stretched into an elliptical one, it indicated complete capture. The viscoelastic was then removed with a bimanual irrigation and aspiration probe. The wound was closed by 10-0 monofilament nylon suture.

Results

Secondary IOL implantation was performed at a mean age of 62.6 months (Range 42-102 months). In all patients, the original cataract surgery was performed within the first 12 months of life (mean, 4.35 months; range,2- 7 months). Each patient achieved a postoperative visual acuity equal to or better than the best-corrected visual acuity obtained before aphakic contact lens trial (range 20/30-20/80) as shown in table 1. All the IOLs were successfully implanted into the ciliary sulcus of the patients with posterior optic capture. No eye had uveitis or glaucoma. The visual axis was free of opacity and the IOL was well centered. No secondary procedures were required. The mean follow-up was 12.4 months (Range 8- 18 months).

Table 1: Table showing demographic data and follow up results

Case (Eye)	Gender	Age of 1st Surgery (months)	Age of IOL implantation (months)	Post op BCVA (6 weeks)	Follow up (months)
1	M	5	48	20/30	16
2	F	5.5	56	20/40	14
3	M	2	42	20/30	18
4	M	4	84	20/40	10
5	M	7	102	20/80	14
6	F	3.5	60	20/30	8
7	M	3.5	42	20/60	12
8	F	4.5	66	20/40	10
9	M	6	80	20/30	10
10	M	2.5	46	20/30	12

Discussion

Surgery for cataracts in infancy usually includes an anterior capsulorhexis, lens aspiration, a primary posterior capsulorhexis, and an anterior vitrectomy.[4] Over the time, remaining equatorial lens epithelial cells often produce new cortical fibers. This process can produce a ring of cortex trapped between the lens equator and the fused anterior and posterior capsulotomy edges. This scarred fibrous fusion may make the anterior and posterior capsule remnants inseparable and the in-the-bag option may not be feasible. At the present time, secondarily implanted lenses in children are relegated almost exclusively to the ciliary sulcus in cases where in the bag option is contraindicated because of the risk of IOL dislocation.[5,6]

In the past, optical rehabilitation of pediatric aphakia was limited to the use of contact lenses or aphakic spectacles. However, in recent years the use of primary IOLs in the optical management of pediatric cataracts has become

quite widespread, at least in children<2 years.[8-12] Nevertheless, because of inadequate axial length and capsular bag dimensions, considerable technical challenges remain in primary IOL placement,[13,14]and their use has not become widespread in very young children and infants, thus resulting in a large population of potential candidates for secondary IOL implantation. The difficulties with inflammation occurring after pediatric IOL implantation cataract surgery, as well as its association with secondary membrane formation, iridolenticular adhesions, and IOL decentration, are well known to pediatric IOL surgeons. Several techniques to prevent after-cataract in children have been described. Optic capture in the posterior capsulorhexis was invented to decrease after cataract formation[15,16]; however, in the follow-up it was found that the anterior vitreous face became semi opaque in many eyes[17]and opacification of the anterior IOL surface occurred.[18]Another important aspect is that greater synechia formation and deposits on the IOL were found, indicating an increased uveal inflammatory response.[28]The same study showed that optic capture resulted in better IOL centration, which is important in cases with decentered or incomplete anterior capsulorhexis typically found in eyes after trauma.

The reported incidence of primary capsulotomy closure is greater than 60% when the capsulotomy is not combined with anterior vitrectomy.[21,22] The visual axis may be obstructed by inflammatory membranes, thickening and opacification of the hyaloid face, and lens epithelial cell (LEC) proliferation.[21,22] Thus, many surgeons routinely perform anterior vitrectomy with primarycapsulotomy.[20,23] The concept of optic capture was used to develop the technique of posterior capsulorhexis with optic capture, first described in 1994 by Gimbel and DeBroff[7] to maintain a clear visual axis in pediatric IOL surgery. Additionally, IOL optic capture through a capsulorhexis opening or a capsular membrane opening have been used to achieve stable and long-term IOL fixation in challenging or complicated situations.

We believe that, as in young children, the process of equatorial epithelial cell proliferation produces a dense ring of cortex between lens equator and fused anterior and posterior capsulotomy edges; a potential space for in-the-bag placement of an IOL may be maintained. In eyes undergoing cataract surgery later in life, perhaps less dense Sommering ring leads to fusion of anterior and posterior capsular leaflets. This scarred fibrous fusion may make the anterior and posterior capsule remnants inseparable and the in-the-bag option may not be feasible.

We described a surgical technique of membranectomy with secondary optic capture in that group of patients in which it is difficult to open the capsular bag. A previous study showed that optic capture results in better centration but predisposes the eye to an increased uveal inflammatoryresponse.[24]In the present study, none of the eyes showed any significant postoperative inflammation.

Awad et al[25] in 1998 reported a series of 57 patients undergoing secondary IOL implantation with 1-piecerigid posterior chamber polymethylmethacrylate lenses implanted in the ciliary sulcus with good visual results. Epley et al[26] reported 28 eyes with either posterior chamber sutured lenses or anterior-chamber lenses. They reported significantly fewer complications with the posterior-chamber lenses. In 1999, Wilson et al[27]reported good results using in-the-bag placement of secondary IOLs. Although this method appears advantageous to sulcus placement when carried out successfully, in-the-bag placement can be technically difficult and not practical in many cases because of tight fusion or partial absence of the anterior and posterior capsules.

Although it is hoped that IOL implantation, both primary and secondary, will ultimately improve visual outcomes in our cataract patients, we must first demonstrate the safety and feasibility of implantation. This series has shown the relative safety of sulcus secondary placement of a multi-piece IOL with membranectomy with optic capture in pediatric patients with adequate capsular support. This procedure shows promising result with minimal complication and none of the patient showing any PCO or decentration till the last follow up.

The benefit of this technique is the creation of a tight barrier between the anterior and posterior segments. This prevents the reformation of secondary PCO. Another added benefit is that by capturing the IOL through the membranous opening will result in better long term stability in position of the IOL.

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CLINICAL STUDY

Retro Pupillary Iris Claw Lens Implantation in Aphakia: Is it a Viable Alternative?

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Abstract

Aim: To evaluate the Indications, surgical outcomes and complications of implantation of Retro pupillary Iris Claw intra ocular lens (RPIC –IOL).

Settings and Design: Prospective, Interventional, Hospital Based study.

Material and method: This study included 42 patients with various indications for RPIC-IOL implantation at a tertiary care centre from September 2020 to August 2021. Informed consent was obtained and the study followed the guidelines of the Declaration of Helsinki. A comprehensive preoperative assessment was done .All the patients underwent RPIC-IOL fixation by a single experienced surgeon using the same standard technique. The patients were examined on postoperative day 1st, 7th day, at one month and three months.

Statistical Analysis: P-value <0.05 was taken as significant. Data entry was done in Microsoft excel. Medcalc 12.2.1.0 version software was used for all statistical purpose.

Results: The most common indication was posterior capsular tear in 23 patients (54.76%). When BCVA was compared pre- and post-operatively at 3 months, *P* value was found to be <0.001, and a significant increase in BCVA was noted in 39 patients between pre- and post-operative values. The mean postoperative IOP at 3 months was 14.21+2.14 mmHg with a range of 10–20 mmHg. Most common postoperative complication was distorted pupil (35.71%).

Conclusion: RPIC-IOL implantation is a viable option for management of aphakia in complicated cases. The technique is safe with short learning curve and good functional outcomes.

Keywords: Retro pupillary, Iris claw lens, Aphakia.

Introduction

Aphakia has posed as a challenge to ophthalmic surgeons since ages. It occurs as a complication following cataract surgery, trauma, the dislocation of a natural lens or an artificial intraocular lens (IOL) into the vitreous chamber. [1,2] It significantly reduces patient's quality of life (PQLI) as it causes hyperopia and anisometropia. But since complications are inevitable hence every cataract surgeon experiences the need of a surgical option to correct aphakia in the absence of capsular and zonular support. With the ongoing development and innovations various types of IOL have gained popularity for the restoration of vision in such cases. Angle supported or iris fixated anterior chamber intraocular lens (ACIOL), a scleral fixated IOL (SFIOL), a fibrin glue assisted suture less posterior chamber intraocular lens (PCIOL) - Glued IOL or an retro pupillary iris claw lens (RPIC-IOL) are amongst them.[3, 4,5]

However, all these options do have their pros and cons. ACIOL is associated with complications like corneal decompensation, cystoid macular edema (CME), secondary glaucoma, uveitis and retinal detachment (RD), hence its use in complicated situations is not recommended. [3,4] SFIOLs, are technically demanding, time consuming with a high incidence of complications such as suture erosions, knot exposure leading to endophthalmitis, lens tilting, decentration, choroidal haemorrhage, RD and CME. [6] Glued IOLs are associated with haptic related complications such as IOL decentration, haptic extrusion and subconjunctival haptic in the long term. [7]Technique of posterior fixation of iris-claw lenses was proposed by Amar et al [8] and later modified by Mohr et al.[9]RPIC has the advantage of retropupillary posterior chamber location and a shorter learning curve.[9,10,11]Although its implantation is technically easy, disadvantages of this method include the size of the incision, which when sutured usually generates astigmatism, and the relatively high cost of the IOL. Since RPIC IOL implantation is gaining popularity in these years we aimed to study the indication postoperative outcomes and complications of it at our tertiary care centre.

Patients and Methods

It was a prospective interventional study, which included 42 patients implanted with RPIC-IOL lens at a tertiary care centre from September 2020 to August 2021. Informed consent was obtained and the study followed the guidelines of the Declaration of Helsinki. Aphakic patients secondary to previous cataract surgery, intraoperative complications leading to inadequate capsular support, zonular dialysis ≥180°, subluxated lens {Figure 1} and IOL, and spontaneously absorbed lens in cases of hyper mature senile cataract were included in the study. Patients with pre-existing corneal pathologies, that intervened with the visual outcome, patients with inadequate support for the iris-claw lens, glaucoma, and posterior segment pathologies were excluded.

Five patients were aphakic at the time of the surgery and 23 patients underwent primary iris-claw lens implantation secondary to intraoperative complications and rest 14 were planned for iris claw lens keeping in mind suspicion of inability to place PCIOL as per the status of natural lens. A comprehensive preoperative assessment was done by history taking and ocular examination including best-corrected visual acuity (BCVA) using Snellen's chart. Slit-lamp examination for assessment of anterior segment, shape of pupil and status of the crystalline lens was done. A thorough fundus examination was done with direct and indirect ophthalmoscope and intraocular pressure measurement with Goldman Applanation Tonometry (GAT). Ultrasound biometry in aphakic mode with IOL power calculation using SRK-II formula with an A-constant of 116.8 for retro pupillary fixation was used uniformly in all the cases of secondary implantation.[12] Primary implantation of RPIC-IOL was under corrected by +2 D in surgeries with intraoperative complications based on the surgeon's discretion. All the patients underwent RPIC-IOL fixation by a single experienced surgeon using the same standard technique. Peripheral Iridectomy (PI) was done in all cases. The patients were examined on postoperative day 1st, 7th day, at one month and three months.

Statistical Analysis

P-value <0.05 was taken as significant. Data entry was done in Microsoft excel. Medcalc 12.2.1.0 version software was used for all statistical purpose.

Results

Forty-two patients with RPIC-IOL were enrolled in our study. Majority of patients were from rural background i.e. 66.66%. The age group of the patients ranged between 27 years and 70 years with majority being above 60 years (26){Graph1}. The most common indication was posterior capsular tear in 23 patients (54.76%). Five patients presented to our OPD with Aphakia secondary to cataract surgery due to posterior capsular tear or subluxated PCIOL, were planned cases for RPIC. Other indications have been tabulated in Table 1.

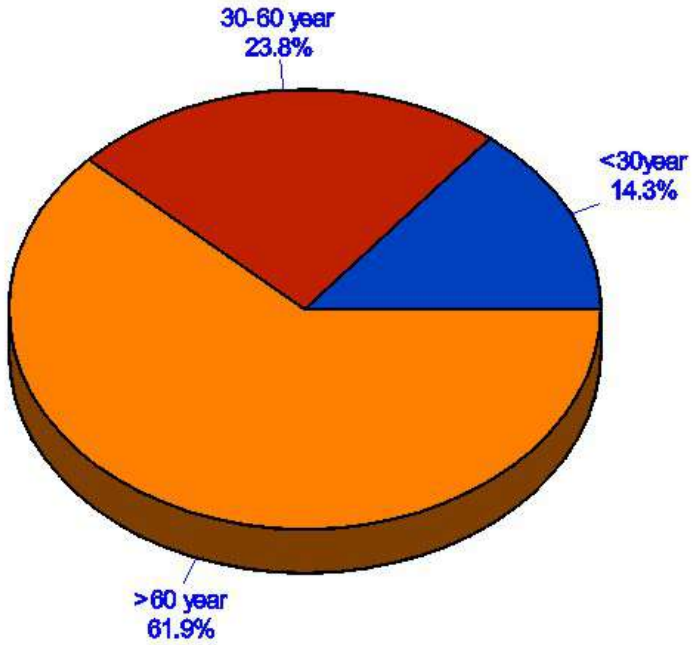
Table 1

Indication of surgery

S.N.	Cause	No. of case	Percentage
1.	Intra operative PC insufficiency or rent	23	54.76%
2.	Subluxation of lens	14	33.33%
	i.Trauma	05	35.71%
	ii.congenital disease	01	7.14%
	iii.Cataract (NS IV,NS V ,HMSC,PXF)	08	57.14%
3.	Aphakia secondary to ECCE Subluxation of IOL post cataract surgery	03 02	7.14% 4.76%

The preoperative BCVA ranged from hand movements + to 6/12. BCVA levels measured preoperatively and postoperatively during 1 and 3 months of follow-up are elucidated in graph 3. When BCVA was compared pre- and post-operatively at 3 months, *P* value was found to be <0.001, and a significant increase in BCVA was noted in 39 patients between pre- and post-operative values while three patients had the BCVA less than 6/60 owing to complications discussed later.

The preoperative IOP ranged between 10 and 30 mmHg with a mean of 16.90+3.52 mmHg. The mean postoperative IOP at 1 week was 17.26+4.49 mmHg and ranged between 8 and 30 mmHg. The mean postoperative IOP at 3 months was 14.21+2.14 mmHg with a range of 10–20 mmHg. Early postoperative rise in IOP was noted in six patients due to retention of viscoelastic, which was controlled by antiglaucoma medications (AGM). One patient had hypotony, which was managed conservatively. (Graph 3)

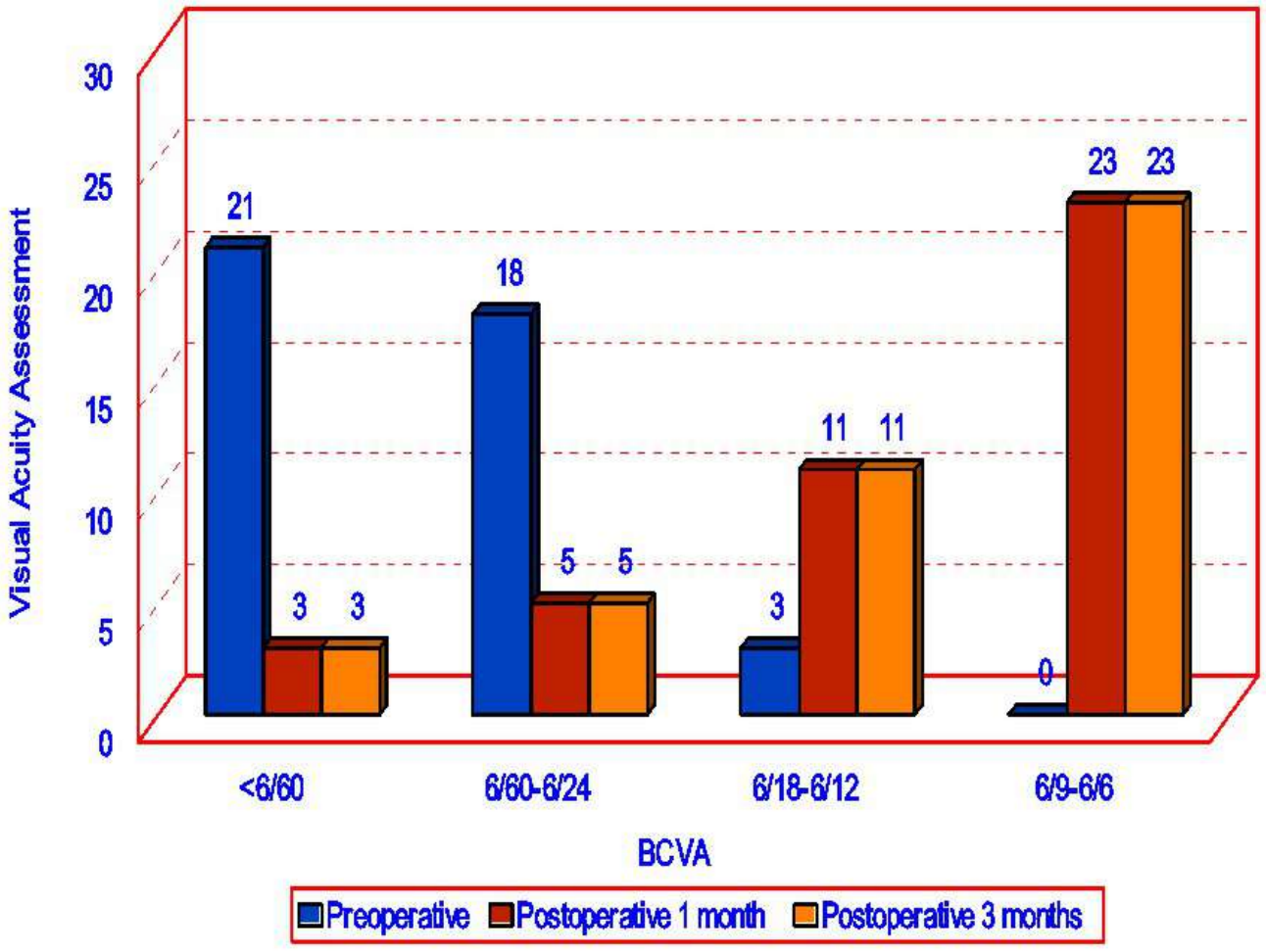


Graph 1 Age distribution of the study population

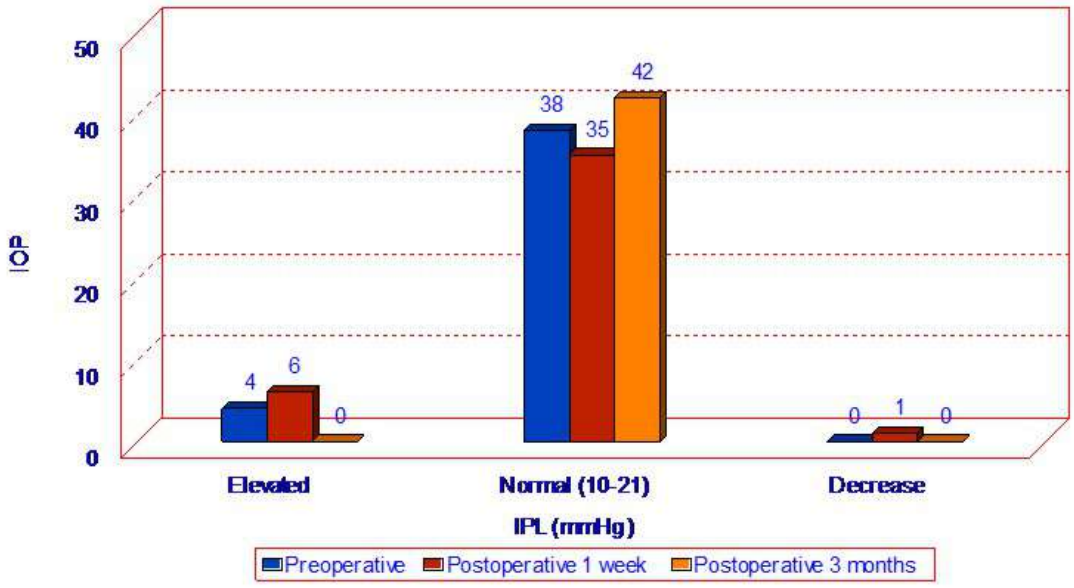
Most common postoperative complication was distorted pupil (35.71%). Two patients developed pseudophakic bullous keratopathy(PBK) due to excessive manipulation during surgery. One patient had cystoid macular oedema (CME) and two of our patient required re-enclavation of RPIC -IOL.(Table 2)

Table 2- Complications

S.No.	Complication	No of patient	Percentage
1	Pseudophakic bullous keratopathy	2	4.76%
2	Raised IOP	6	14.28%
3	Hyphaema	3	7.14%
4	AC reaction	10	23.80%
5	Distorted Pupil	15	35.71%
6	Subluxation of iris claw	2	4.76%
7	Vitreous wick syndrome	1	2.38%
8	Macular edema	1	2.38%



Graph 2- Visual Acuity Assessment



Graph 3: IOP Assessment

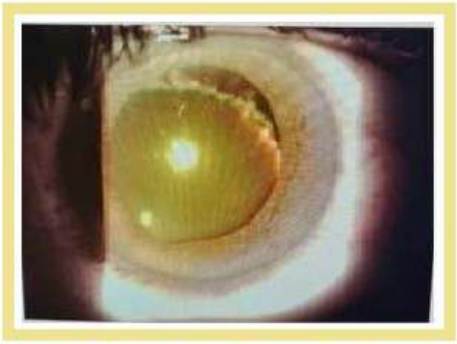


Figure 1 Showing Lens Subluxation

Figure 2 Showing Ovalization of pupil

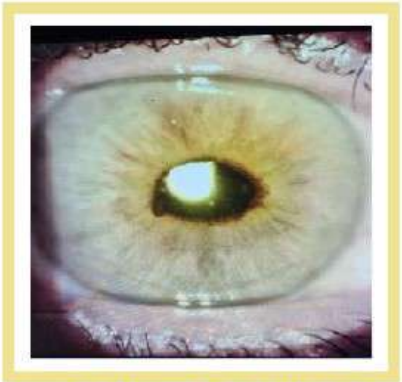


Figure 3 Round Pupil in Retro pupillary Iris claw lens

Figure 4 Showing Vitreous Wick Syndrome

Discussion

In our study rural patients comprised of 66.67% as compared to 33.33% of urban. This was attributed to presentation of rural patients in late stage of cataract pathogenesis like mature cataract, hyper mature cataract and traumatic cataract. Major age group belong to more than 60 year comprising of 61.90% (26). This age group had high prevalence of hyper mature senile cataracts, pseudo exfoliation syndrome, zonular weakness and lens subluxation that leads to complicated cataract surgery. Six (14.28%) patients belong to <30-year age group that presented with traumatic and congenital cataract. The major indication of iris claw implantation was intraoperative posterior capsule rupture i.e. 54.76%, which was in accordance with the studies done by Sezer Helvaci *et al*[13]and Maurice Schallenberg *et al.*[12]

The final visual outcome was better than preoperative visual acuity in 92.85% of patients. Three patients who had BCVA<6/60 were attributed to PBK (two), Cystoid macular edema (one) respectively. Jayamadhury et al reported similar improvement in BCVA in their study.[14] It was also in accordance with Kelkaret al.[15] Hence RPIC-IOL seems to be effective alternative for visual rehabilitation of aphakia in complicated cases.

Various studies have reported postoperative IOP elevation in 4.3%[16]–6%[12]. In our study, no case of pupillary block was observed during the follow-up period, which could be because of the fact that intraoperative PI was performed for all the eyes. Transient rise in IOP was noted in 14.28% of the eyes between day 1 and 1 week postoperatively, which was attributed to the retained viscoelastic, and was controlled with use of AGM. None of the eyes required long-term use of AGM.

Pupil distortion was noted in 15 patients, {Figure 2} which occurs due to asymmetrical fixation of haptic, tight fixation of haptic, or a difference of iris tissue volume that was clamped with both haptics.[17] To prevent pupillary distortion, iris-claw IOL should be fixed in the peripheral part of the iris and both the claws should clamp symmetrically on the iris with equal volume of iris tissue.[18] {Figure 3}. Anterior chamber reaction was noted in 10 patients, which responded to topical steroid therapy. Three patients had postoperative hyphaema, which also responded to conservative management. These were attributed to intraoperative iris manipulation. Hence gentle and skillful manipulation of tissue is important aspect of RPIC-IOL implantation.

Improper intraoperative enclavation of haptics or postoperative trauma can lead to the dislocation/subluxation of the iris-claw IOL into the vitreous cavity. In our study, 2 eyes (4.76%) had one haptic disenclavation, requiring re-surgery to enclave IOL haptic. Labeille et al reported 8.7% cases of dislocation at 3.3 months.[19] Long-term observations are needed to assess the stability of anchoring after implantation of RPIC-IOL. In our study, 2.38% of eye had CME;

it was due to vitreous wick syndrome {Figure 3}. This patient was referred to vitreo-retinal surgeon for further management The incidence of CME with RPIC-IOL has been reported to be 1.9% by Jare et al.[20]

The limitation of our study was small sample size and short follow-up and lack of endothelial cell count record. However the retropupillary position of the iris-claw IOL and its distance from the endothelium theoretically limits the risk of injury to corneal endothelium. [20] Studies with larger sample size and longer follow-up are needed to demonstrate the superiority of RPIC and probable long-term complications.

Conclusion

RPIC-IOL implantation is a viable option for management of aphakia in complicated cases. The technique is safe and easy to learn with good functional outcomes and less complications.

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CLINICAL STUDY

Central Corneal Thickness Used as A Variable to Compare and Assess Protective Action of Ripasudil Versus Travoprost Along with their Intraocular Pressure Lowering Ability in Primary Open Angle Glaucoma: A Comparative Study

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Abstract

Purpose: To evaluate the decrease in intraocular pressure and change in central corneal thickness in patients treated with ripasudil (0.4% w/v) versus travoprost (0.004% w/v) in patients suffering from primary open angle glaucoma.

Method and design: This 6 month, randomized, single center, parallel group, comparative, interventional study compared the efficacy of ripasudil and travoprost on intraocular pressure and their effects on the central corneal thickness. The study evaluated 40 patients suffering from primary open angle glaucoma (POAG) divided into two equal groups of 20 each.

Results: The baseline intraocular pressures were similar across the groups. Decrease in intraocular pressure (IOP) of 19.1% and 29.2% was observed for ripasudil and travoprost from the baseline on the last follow up (p value < 0.001). Also, a mean change of central corneal thickness (CCT) from baseline to last follow up was 505.65 microns to 505.05 microns in the ripasudil group and from 522.85 microns to 520 microns for travoprost group.

Conclusion: Travoprost is a more potent intraocular pressure lowering drug and the study underlines the protective effect of ripasudil however small on central corneal thickness.

Keywords: Ripasudil, travoprost, intraocular pressure, central corneal thickness, primary open angle glaucoma.

Introduction

Globally glaucoma is an important cause of irreversible vision loss.[1] 3.57% of the population over 40 years suffers from it and as the population ages its incidence is set to increase.[2]

To combat the disease we have come a long way but still no definitive treatment has been found. So, time and again we have come up with newer treatment modalities to fight it. In this study we compare travoprost, a prostaglandin, which increases uveo-scleral outflow[3] and ripasudil that acts via Rho kinase pathway to evaluate their effects in patients suffering from primary open angle glaucoma.[4]

Methods

Proper approval by ethical committee of the institute was taken and the ethical standards set by the Helsinki Declaration of 1964, as revised in 2013 were followed. The study was a single-centre, prospective, randomized, comparative and parallel-group study.

At the time of enrollment patients received information regarding the protocol and written informed consent for the study after which they were randomized by the random chit method.

For the examination of the patients slit-lamp examination, visual acuity testing, refraction, Goldmann applanation tonometry for IOP: All measurements were taken at each time point at least twice. If the measurements differed by 2 mmHg, a third measurement was taken: The mean of 2 or the median of 3 recordings was used for analysis, indirect ophthalmoscopy, pachymetry, gonioscopy, and optical coherence tomography for retinal nerve fiber layer (examination done by well-trained specialists).

A washout period was employed for patients being previously treated with some other IOP-lowering medications (b-blockers and prostaglandin analogs, 4 weeks or more; others, 2 weeks or more).

Follow up: Patients were evaluated at monthly follow-ups. Vision, IOP, CCT was assessed on every follow-up. And retinal nerve fibre layer optical coherence tomography was evaluated on first and last follow-up.

Inclusion criteria

Patients willing to give written informed consent, patients suffering from primary open angle glaucoma.

Exclusion criteria: Patients not willing for the study, angle-closure glaucoma or with narrow angles defined as grade 2 or less of the Shaffer classification by gonioscopy, patients with IOP levels of 30 mmHg or higher, secondary causes of elevated IOP, corneal abnormalities preventing reliable IOP measurement, previous filtration surgery, use of any glucocorticoid or ocular nonsteroidal anti-inflammatory agents, which inhibit cyclooxygenase and prostaglandin synthesis, having a single eye, life-threatening or debilitating disease, pregnancy.

Results

Fourty patients were randomly enrolled and divided equally into two groups: 20 patients were allotted ripasudil group (group A) and the other 20 to travoprost (group B). Both drugs were instilled in a once-a-day regimen. Patients baseline characteristics were documented on the first visit. t-test was used for statistical analysis. Mean age for group A and group B was 53 ± 16.94 years and 52.15 ± 15.97 years respectively (p=0.87). In group A there were 75% males and 25% females whereas in group B there were 65% males and 35% females (p=0.73).

Table 1: Intraocular lowering effect (Group A and Group B)

	Group A		Group B		Result (P value)
	Mean	SD	Mean	SD	
BASELINE	23.50	2.04	23.95	2.24	0.509 (NS)
FIRST FOLLOW UP	20.40	2.95	20.20	2.93	0.830 (NS)
SECOND FOLLOW UP	18.80	2.59	18.50	2.74	0.723 (NS)
THIRD FOLLOW UP	19.45	2.37	17.95	2.24	0.046 (S)
FOURTH FOLLOW UP	18.65	2.43	18.05	2.31	0.428 (NS)
FIFTH FOLLOW UP	19.20	1.96	16.50	2.06	0.0001 (S)
LAST FOLLOW UP	19.00	2.45	16.95	2.44	0.011 (S)

S = Significant; NS = Non-Significant; the follow ups were taken monthly till six months after a baseline follow up

Table 2: Central corneal thickness in Group A and B

	Group A			Group B		
	Mean	**SD**	**p value**	**Mean**	**SD**	**p value**
Baseline	505.65	40.82	-	522.85	31.29	-
Follow up 1	1.8	7.99	0.326 (NS)	1.6	6.04	0.251 (NS)
Follow up 2	2.55	6.88	0.114 (NS)	0.60	5.06	0.602 (NS)
Follow up 3	1.9	6.57	0.211 (NS)	2.75	5.06	0.025 (S)
Follow up 4	6.55	25.01	0.256 (NS)	2.05	3.9	0.030 (S)
Follow up 5	1	6.23	0.482 (NS)	1.75	5.34	0.159 (NS)
Follow up 6	0.60	6.62	0.690 (NS)	2.85	4.02	0.005 (S)

S = Significant; NS = Non-Significant; the follow ups were taken monthly till six months after a baseline follow up.

Now comparing IOP changes between two drugs

A mean baseline IOP of 23.50 ± 2.04 (mean ± SD) mmHg was noted in the ripasudil group and after treatment the IOP at first, second, third, fourth, fifth and sixth follow up reduced by 3.10 ± 1.97 (13% decrease from baseline),4.70 ± 1.84 (20% decrease from baseline), 4.05 ± 2.09 (17% decrease from baseline), 4.85 ±1.42 (20.6% decrease from baseline), 4.30 ± 1.42 (18.2% decrease from baseline), 4.50 ± 1.85 (19.1% decrease from baseline) (mean ± SD) mmHg respectively. Thus, achieving 13% to 20.6% decrease in IOP from the baseline. These results show high statistical significance (p=0.00) at all follow up points.

For travoprost group the mean baseline IOP was 23.95 ± 2.23 (mean ± SD) mmHg and at first, second, third, fourth, fifth and sixth follow ups IOP reduction was noted to be 3.75 ± 2.57 (15.6% decrease from baseline),5.45 ± 1.90 (22.7% decrease from baseline), 6 ± 2.42 (25% decrease from baseline), 5.90 ± 1.86 (24.6% decrease from baseline), 7.45 ± 2.23 (31.1% decrease from baseline), 7 ± 2.05 (29.2% decrease from baseline) (mean ± SD) mmHg. So, end IOP reduction achieved was between 15.6% to 31.1% from the baseline. These results too have high statistical significance (p=0.00).

Another variable that was documented for both the drugs was the central corneal thickness (CCT). As was seen for group A the mean baseline CCT was 505.65 ± 40.82 (mean ± SD) microns which had a mean decrease of 1.8 ± 7.99, 2.55 ± 6.88, 1.9 ± 6.57, 6.55 ± 25.01, 1 ± 6.23, 0.60 ± 6.62 microns at first, second, third, fourth, fifth and sixth follow ups respectively and shows no statistical significance (that is p value >0.05) at any time point whatsoever. Also, for group B which had a mean baseline value of 522.85 ± 31.29 (mean ± SD) microns decreased with serial follow ups to 1.6 ± 6.04, 0.60 ± 5.06, 2.75 ± 5.06, 2.05 ± 3.9, 1.75 ± 5.34, 2.85 ± 4.02 microns at first, second, third, fourth, fifth and sixth follow ups respectively wherein the first, second, third, fourth, fifth and sixth had a p value of 0.25, 0.60, 0.02, 0.03, 0.16, 0.005 hence showing a mixed significance for this variable.

Discussion

Fourty eyes of 40 patients were randomly and equally divided into groups. And then assessment for IOP changes and CCT was done during the study.

In our study for ripasudil group we obtained a 3.10 mmHg (13%) to 4.85 (20.6%) mmHg decrease in IOP which showed similarity to a 52 weeks long term study by Tanihara et al who reported a reduction in IOP of 2.6 mmHg (13.5%) at the trough and 3.7 mmHg (19.4%) at the peak.[5] Tanihara et al in another post-marketing surveillance study noted an IOP decrease in POAG patients of - 2.9±4.2 mmHg[6] which shows appreciation with the results of our study wherein an IOP decrease of the range of 3.10 to 4.85 mmHg was obtained. In a multicentric cohort study by Fukakuchi et al IOP reductions from baseline at 1, 3, and 6 months were - 19.4±25.1%, - 20.0±27.1%, and - 23.4±25.6% respectively which were similar to the decrease in IOP of about 20.6% which points to the similarity between the results.[7]

In the travoprost group an IOP reduction between 15.6% to 31.1% from baseline was obtained. Goldberg et al from a baseline IOP of 26 mmHg achieved a mean IOP reduction between 8.0 to 8.9 mmHg (about 30%) with travoprost 0.004% which correlates well with our study.[8] In a 12 months study by Netland et al after a baseline IOP between 25–26 mmHg the mean IOP decrease ranged from 17.7 to 19.1 mmHg with travoprost 0.004%.[9] Similarly we observed a mean IOP of 23.95 mmHg decrease ranged between 16.50 to 20.20 mmHg. Concluding that both the studies showed similar percentage decrease of around 30 percent. Fellman et al in their 6 month study achieved about a 25 percent or more percentage IOP reduction that ranged between 6.5 and 8.0 mmHg showing that the results were similar to our study.[10]

Also, the interdrug comparison among these drugs shows a visible difference between the IOP lowering potential. In the beginning the two drugs didn't show any statistical significance but as follow ups progress the mean difference of IOP reduction between the two drugs widens and becomes statistically significant where travoprost at 31.1% IOP reduction seemingly looks to be the

more potent drug over ripasudil at 20.6% decrease in IOP. Thus, proving travoprost-like drugs as the drug of first choice for many.

The neuroprotective effect of ripasudil have been studied and it has been considered as a drug for neuro-protection in glaucoma.[11] So as to evaluate this we have used CCT as a parameter in our study.

In the study, for group A the mean baseline CCT was 505.65 ± 40.82 (mean ± SD) microns which had a mean decrease of 1.8 ± 7.99, 2.55 ± 6.88, 1.9 ± 6.57, 6.55 ± 52.01, 1 ± 6.23, 0.60 ± 6.62 microns at first, second, third, fourth, fifth and sixth follow-ups respectively and shows no statistical significance at any time point whatsoever. For group B which had a mean baseline value of 522.85 ± 31.29 (mean ± SD) microns on serial follow-ups showed a decrease of 1.6 ± 6.04, 0.60 ± 5.06, 2.75 ± 5.06, 2.05 ± 3.9, 1.75 ± 5.34, 2.85 ± 4.02 microns at first, second, third, fourth, fifth and sixth follow-ups respectively wherein the first, second, third, fourth, fifth and sixth had a p value of 0.25, 0.60, 0.02, 0.03, 0.16, 0.005 hence showing a mixed significance for this variable. At the time of our study, there was no comparator study (or study known to us; so, this study being the only one to provide with quantitative data for this variable) for us to compare our results. But what was interesting to note was that the ripasudil group patients had no significant change in the CCT all through the study follow-ups but on the other hand the travoprost group did show a significant change however small during the study period.

This might point to the protective effect of ripasudil on CCT for which larger and more focused studies might be needed to appreciate the results better.

Four patients in our study had hyperemia as the only adverse effect in the ripasudil group. Vasodilatation of blood vessels from smooth muscle relaxation is known in case of Rho-kinase inhibitors which causes ocular hyperemia.[12] Ocular hyperemia in our study lasted for only about an hour or two and resolved on its own. Adverse effect as reported by other studies were not observed by us.[6]

Two patients had hyperemia and 1 patient had itching and burning sensation who was lost to follow up in the travoprost group. Peculiar adverse effects such as eyelash growth, iris hyper-pigmentation as noted in other studies were not observed by us.[13]

The drawbacks of the study were that the patients were not screened for racial differences, follow-ups could have been longer, number of patients in the study, we did not compare the IOP lowering effects of the drugs in fixed-dose combinations, and finally evaluation for placebo effect was not done.

Conclusion

The study shows that travoprost and ripasudil both have significant IOP lowering effect but travoprost is more efficacious. Also, ripasudil has a significant protective effect, however small, on the CCT whereas travoprost does not.

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CLINICAL STUDY

Importance of OCT in Explaining Visual Prognosis to Cataract Patients

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Visual impairment is one of the most tragic problems one can face. Multiple reasons can be responsible for such impairment, ranging from cataracts to grave retinal conditions. When these conditions are managed by an ophthalmologist, the visual prognosis needs to be deciphered and discussed with the patient. In case of a cataract, diminution of vision can be entirely due to the cataract or partially due to the cataract and partially due to other reasons, for example, RNFL defects in glaucoma patients or a macular hole[1].

Our tertiary centre (Pacific Institute of Medical Sciences, Umarda, Udaipur), by virtue of being located in southern Rajasthan, sees many patients of solar maculopathy/photic retinopathy. This is due to the extreme sun and harsh temperatures of this region. We do not have much data to show for it, but we also observed macular thinning and when asked about their medical history, a large number of those patients had tested COVID-19 positive at some point of time.

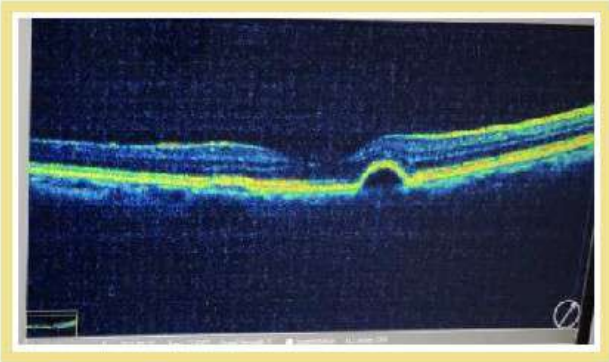
In the pre-operative evaluation of a cataract patient, among other investigations, the fundus is also evaluated, and is of significant importance when the vision loss does not correlate with the grade of the cataract. Some findings may be missed when evaluating the fundus on slit-lamp biomicroscope, or may be impossible to appreciate, in case of mature cataracts. In these cases, optical coherence tomography is a very useful tool in picking up any optic disc or macular abnormalities[2,3], which will help the ophthalmologist to better explain the visual prognosis of the specific patient's cataract surgery[4]. OCT is the most established method to evaluate the retinal anatomy [5] and can help the ophthalmologist not face any surprises post-op with regards to visual outcome.

In our medical college, we came across many such cataract patients for whom their vision loss was not explained by the grade of the cataract, and performing OCT gave us a perfect explanation for the same.

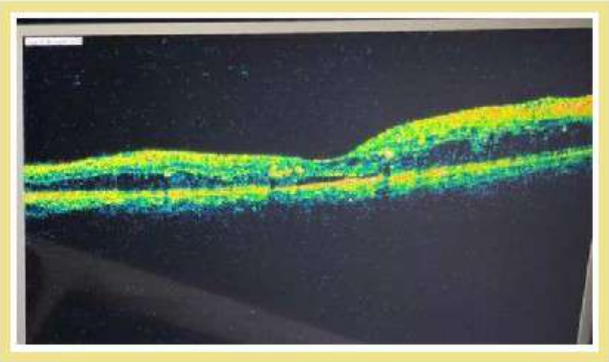
Patients and Methods

Candidates were all cataract patients. We performed a complete ocular examination for them which included slit-lamp biomicroscopy, intraocular pressure screening by non-contact tonometry (NCT), and a dilated fundus examination with 90D lens.

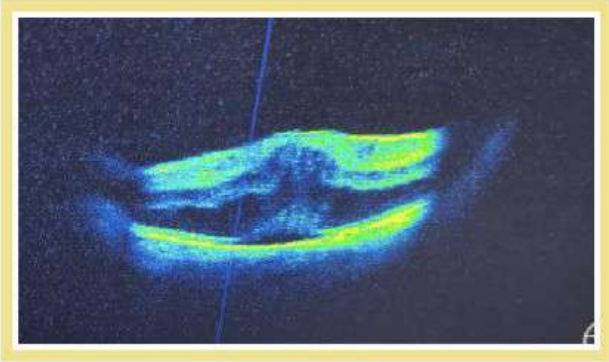
Then, we proceeded with performing OCT for these patients. These are some of the scans we recorded:



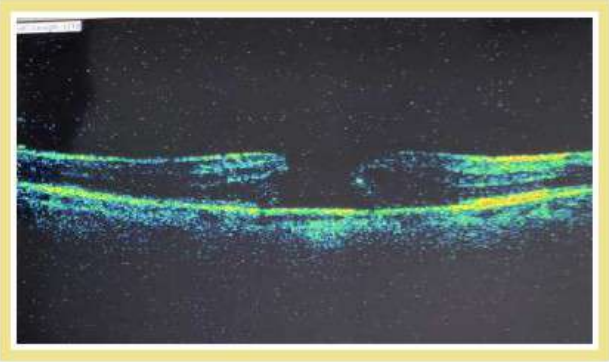
Pigment Epithelium Detachment



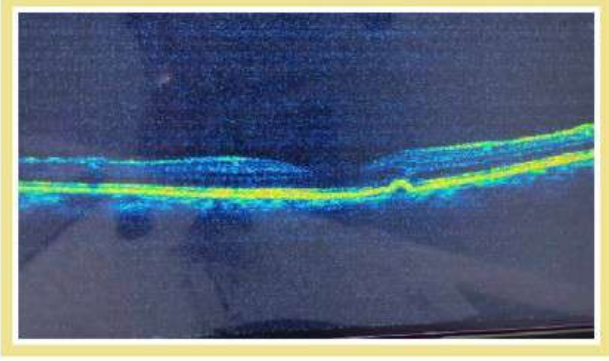
Intraretinal Edema (cystoid spaces)



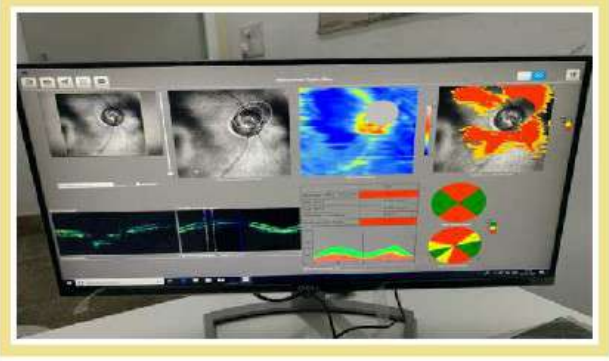
Cystoid Macular Edema (CME)



Full Thickness Macular Hole



Dry ARMD (PED)



Disc OCT

Conclusion

Nowadays, cataract surgery is not just a surgery to restore vision, but it is treated like a refractive surgery wherein patients expect the best possible correction for their scenario. After the patients were informed of their respective prognoses of surgeries, they were assured that the Ophthalmology team had been transparent with them and had conducted a thorough examination of their eyes, which immensely satisfied the patients. Fundus photographs are also a good tool to visualize the fundus, but OCT is far more superior in terms of explaining the condition quantitatively. OCT has the advantage of giving the ophthalmologist the confidence to explain the prognoses by talking in numbers and stating facts to the patient, which gives the patient reassurance as well. They gave their consent to be operated for their cataracts, and also accepted the visual outcomes they had, respectively. Some of these cases were treatable, and were managed accordingly by administering steroids, etc. But in cases which were irreversible, the explanation satisfied the patients and they were happy with 6/12 or even 6/24 vision. All these patients were happy in their post-operative follow-ups, not because they had 6/6 vision but because they knew the entire truth of what was happening with their eyes in entirety.

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CLINICAL STUDY

A Study to Determine Causes of Childhood Blindness and Ocular Morbidity Amongst Individuals Seeking Visual Disability Certificate in Western Rajasthan

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Abstract

Purpose- The objectives of this study were to determine the causes of childhood blindness and ocular morbidity amongst individuals seeking visual disability certificate in western Rajasthan, Jodhpur.

Methods- The observational study conducted on children less than or equal to 15years age who came seeking visual disability certificate in the Outpatient Department of Ophthalmology. All the patients were subjected to comprehensive ocular examinations and causes of blindness were categorized anatomically and etiologically.

Results-A total 200 children were undergone complete ophthalmic examination for visual disability certification. The visual impairment was maximum in the age group of 11 to 15 years. The main cause of avoidable VI in these children was a refractive error (75.0%).

Conclusion-Optic nerve abnormalities were the leading cause of blindness in children. Refractive errors are the most important cause of visual impairment in children and must be treated.

Introduction

Visual impairment (VI) has a profound impact in terms of limited educational, recreational, and social opportunities in children. About 1.4 million blind children under age of 16 are blind worldwide, about 75% of whom are from developing countries. The prevalence of blindness in children varies from 0.3 per thousand children in developed countries to 1.5 per thousand in developing countries. [1,2]

The certification of blindness or low vision is the process through which the social services for visually impaired in India is categorized according to severity and carried out by an official committee. This includes an ophthalmologist. According to the guidelines of the Ministry of Social Justice and Empowerment of the Indian government, the minimum degree of disability should be 40%for a person to be entitled to it concession or benefit.[3]

According to recent estimates by WHO, the major global causes of moderate to severe vision impairment are: uncorrected refractive errors 53%, cataract 25%, age related macular degeneration 53%, glaucoma 2%, diabetic retinopathy 1%. Leading causes of childhood blindness include xerophthalmia, congenital cataract, congenital glaucoma and optic atrophy due to meningitis, Retinopathy of prematurity and uncorrected refractive errors.[4]

Only few studies in India have analysed the applications of blindness certificates to know the causes of childhood blindness and its application in planning Eye Health Programme to reduce the blindness. No such study was conducted in our area, Jodhpur Western region of Rajasthan. So, our study is aimed to analyse the causes of blindness and visual impairment amongst children coming for visual handicap certification and its application in health planning to reduce the blindness.

Patients and Methods

A Cross-sectional study was conducted on children aged <16 years perusing certification for visual disability from March 2021-March 2022 for a period of one year in Department of ophthalmology, Dr S.N Medical College, Mathura Das Mathur Hospital, Jodhpur with approval of the Institutional Ethics Committee and Examination Board after written consent of the patients. The sample size was calculated to be minimum 180 subjects which was rounded off

to 200. Sample size was calculated with 95% confidence interval to verify an expected 16% proportion of retinitis pigmentosa among all cases of blindness as reported by Brijesh Patil et al. reported and 5% accepted in absolute terms allowable error. All the patients were subjected to comprehensive ocular examination which includes best corrected visual acuity using Snellen`s chart. Anterior segment examination using slit lamp, applanation tonometry for intraocular pressure. Posterior segment analysis with the help of direct ophthalmoscope, indirect ophthalmoscope and/or slit lamp biomicroscopy using +90D lens after dilating the pupil with mydriatics. Optical Coherence Tomography (OCT) and/or ERG/VEP and MRI, CT scan, Ultrasonography (USG) in selected cases. The study team also collected self-reported information about history of blindness, onset of blindness and family history of blindness including any known immediate or extended family member who were blind or had a visual impairment. The standard classification of visual impairment and blindness were followed as per International classification of disease (ICD 10). Total 200 cases i.e., visually impaired (VI) was divided further into blind (BL) and moderate to severe visual impairment (MSVI)

Results

In our study total 200 children were examined for their visual disability certification process in our hospital from March 2021-March 2022. Amongst 200 children 119 (59.5%) were aged between 11-15 years followed by 77 (38.5%) children in the age group of 6-10 years and 4 (2%) children between 3-5 years and none of them in the age group of 0-3 years. 108 (54%) were males whereas 92 (46%) were females (Table 1).

Table 1: Age and gender wise prevalence of blindness and visual impairment in children (PVA better eye) (n=200)

Age (years)	BL (<3/60) n = 15	MSVI (<6/18-3/60) n =185	VI (<6/18) n =200
<3	0	0	0
3-5	1 (6.67%)	3 (1.6%)	4 (2%)
6-10	6 (40%)	71 (38.3%)	77 (38.5%)
11-15	8(53.33%)	111 (60%)	119 (59.5%)
GENDER			
Male	8 (53.3%)	100 (54.0%)	108 (54%)
Female	7 (46.6%)	85 45.9%)	92 (46%)

Table 2: Anatomical causes of visual impairment among children based on presenting visual acuity (better eye) (0-15 years) (n=200)

Better eye: PVA	Causes	Male (%)	Female (%)	Total (%) (n = 200)
BL (<3/60)	Cornea	1 (12.5)	1 (14.2)	2 (13.3)
	Lens	1 (12.5)	2 (28.5)	3 (0.20)
	Retina	2 (25)	2 (28.5)	4 (26.6)
	Optic nerve	3 (37.5)	2 (28.5)	5 (33.3)
	Others* (Etiological-refractive error/ amblyopia)	1 (12.5)	0 (0)	1 (6.6)
	Total	8 (100)	7 (100)	15 (100)
MSVI (<6/18-3/60)	Whole globe	0 (0)	3 (3.5)	3 (1.6)
	Cornea	1 (1)	0 (0)	1 (0.5)
	Uvea	2 (2)	2 (2.3)	4 (2.1)
	Retina	17 (17)	6 (7.0)	23 (12.4)
	Optic nerve	2 (2)	0 (0)	2 (1.0)
	Others* (Etiological-refractive error/ amblyopia)	78 (78)	74 (87.05)	152 (82.1)
	Total	100 (100)	85 (100)	185 (100)
VI (<6/18) (BL+MSVI)	Whole globe	0 (0)	3 (3.2)	3 (1.5)
	Cornea	2 (1.85)	1 (1.0)	3 (1.5)
	Lens	1 (0.9)	2 (1.85)	3 (1.5)
	Uvea	2 (1.85)	2 (1.85)	4 (2.0)
	Retina	19 (17.6)	8 (7.4)	27 (13.5)
	Optic nerve	5 (4.6)	2 (1.85)	7 (3.5)
	Others* (Etiological-refractive error/ amblyopia)	79 (73.15)	74 (80.4)	153 (76.2)
	Total	108 (100)	92 (100)	200 (100)

A total of 15 children were blind, 8 (53.3%) male and 7 (46.6%) female and 185 children had moderate to severe visual impairment, of which 100 (54.0%) were male and 85 (45.9%) were female. In total were 185 visually impaired children with visual acuity <6/18 of these 108 (54%) were male and 92 (46%) were female. Amongst these 15 blind children a total of 5 (33.3) had optic nerve abnormalities (optic atrophy in 2 and glaucomatous optic nerve head cupping), 4 (26.6) with retinal (2 each with retinopathy of prematurity stage 5 and PHPV) and 3 (20.0) lenticular abnormalities (cataract), 2(13.3) had corneal (keratoconus) involvement and refractive error. In the remaining 185 children with MSVI, a total of 23 (12.4%) children had retinal (8 with retinal dystrophy, 18 with myopic chorioretinal changes) abnormalities, 4 (2.1%) had uveal (coloboma) abnormality, 3 (1.6%) each had whole globe (anophthalmos) abnormality or corneal (scar) or optic nerve (coloboma) abnormality remaining 152 (82.1%) children had no anatomical cause involved and were suffering from refractive error or amblyopia [Table 2].

On classifying these 200 VI children for etiological abnormalities, according to presenting visual acuity(PVA) in better eye, in15 children with blindness, 7 (3.5%) had perinatal (birth hypoxia) abnormality, 1 (0.5% each) had un-operated cataract and glaucoma, 3 (20% each) had hereditary (autosomal recessive), postnatal (trauma) abnormalities and refractive error respectively. Amongst the children diagnosed with MSVI, the major etiological cause was hereditary 8 (4.3%) (4 each with autosomal recessive and chromosomal anomalies) followed by 5 (2.7%) with undetermined cause. The remaining 172 (92.9% classified as others) had refractive error or amblyopia or idiopathic nystagmus [Table 3].

Table 3: Distribution of etiological causes of visual impairment among children aged <16 years (n=200)

Better eye: PVA	Causes	Male (%)	Female (%)	Total (%) (n = 200)
BL (<3/60)	Hereditary	1 (12.5)	2 (28.5)	3 (20)
	Perinatal	5 (62.5)	2 (28.5)	7 (46.66)
	Postnatal	1 (12.5)	0 (0)	1 (6.6)
	Undetermined etiology (cataract/ glaucoma)	1 (12.5) 0	1(14.28) 2 (28.5)	2 (13.3) 2(14.3)
	Refractive Error	8 (100)	7(100)	15 (100)
	Total			
MSVI (<6/18-3/60)	Hereditary	4 (4)	4 (47)	8 (4.3)
	Undetermined abnormality since birth	3 (3) 83(83)	2 (23.5) 65 (76.4)	5 (2.7) 148 (80)
	Refractive Error	8 (8	12 (14.1)	20 (10.8)
	Amblyopia	2 (2)	2 (23.5)	4 (21.6)
	Idiopathic nystagmus	100 (100)	85 (100)	185 (100)
	Total			
VI (<6/18) (BL+MSVI)	Hereditary	5 (4.6)	6 (6.5)	11 (5.5)
	Perinatal	5 (4.6)	2 (2.1)	7 (3.5)
	Postnatal	1 (0.9)	0	1 (0.5)
	Undetermined etiology (cataract)	1 (0.9) 0(0)	0 1(1.08)	1 (0.5) 1(0.5)
	Undetermined etiology (glaucoma)			
	Abnormality since birth	3(2.7)	2 (2.1)	5 (2.5)
	Refractive Error	83(76.8)	67(72.8)	150(75)
	Amblyopia	8(7.4)	12 (13)	20(10)
	Idiopathic nystagmus	2(1.8)	2(2.1)	4(2)
	Total	108(100)	92(100)	200 (100)

Discussion

Globally, 1.4 million children suffer from blindness and it is estimated that almost two-third of these live in developing countries. Overall, there are probably2,80,000-3,20,000 blind children in India.[1] This is a major Western Indian population-based study on childhood visual impairment.

In our study total 200 children were examined for their visual disability certification process in our hospital from March 2021-March 2022. Amongst 200 children 119 (59.5%) were aged between 11-15 years followed by 77 (38.5%) children in the age group of 6-10 years and 4 (2%) children between 3-5 years and none of them in the age group of 0-3 years. 108 (54%) were males whereas 92 (46%) were females.

A total of 15 children were blind, 8 (53.3%) male and 7 (46.6%) female and 185 children had moderate to severe visual impairment, of which 100 (54.0%) were male and 85 (45.9%) were female. In total were 185 visually impaired children with visual acuity <6/18 of these 108 (54%) were male and 92 (46%) were female. A study done by Wadhwani M *et al* (2021) suggested that a greater number of males were visually impaired as compared to females.[5] Most of the cases were belongs to age group 11-15 years which was 49.19% followed by age group 6-10 years i.e., 40.3%. similar demographic pattern was found in study done by Kemmanu et al[6].

In our study amongst 15 blind children a total of 5 (33.3) had optic nerve abnormalities (optic atrophy in 2 and glaucomatous optic nerve head cupping), 4 (26.6) with retinal (2 each with retinopathy of prematurity stage 5 and PHPV) and 3 (20.0) lenticular abnormalities (cataract), 2(13.3) had corneal (keratoconus) involvement and refractive error. In the remaining 185 children with MSVI, a total of 23 (12.4%) children had retinal (8 with retinal dystrophy, 18 with myopic chorioretinal changes) abnormalities, 4 (2.1%) had uveal (coloboma) abnormality, 3 (1.6%) each had whole globe (anophthalmos) abnormality or corneal (scar) or optic nerve (coloboma) abnormality remaining 152 (82.1%) children had no anatomical cause involved and were suffering from refractive error or amblyopia.

The main anatomical cause of blindness was optic nerve abnormalities in this study. This is similar to another population-based study conducted by Nirmalanet al. [7] but differed from Kemmanuet al. [6] and Dorairaj[8] et al., as in

their studies the main anatomical site for blindness was posterior segment abnormalities and lenticular abnormalities respectively [Table 2].[7,10] The main anatomical cause for visual impairment in this study was posterior segment abnormalities, this is the similar to the findings reported by Kemmanu et al. and Dorairaj et al [6,8].

On classifying these 200 VI children for etiological abnormalities, according to presenting visual acuity (PVA) in better eye, in 15 children with blindness, 7 (3.5%) had perinatal (birth hypoxia) abnormality, 1 (0.5% each) had unoperated cataract and glaucoma, 3 (20% each) had hereditary (autosomal recessive), postnatal (trauma) abnormalities and refractive error respectively. Amongst the children diagnosed with MSVI, the major etiological cause was hereditary 8 (4.3%) (4 each with autosomal recessive and chromosomal anomalies) followed by 5 (2.7%) with undetermined cause. The remaining 172 (92.9% classified as others) had refractive error or amblyopia or idiopathic nystagmus [Table 3].

In 2000, Hornby SJ et al reported that 291 students under 16 years were examined and after refraction 267 (91.7%) were classified as diseased severely visually impaired or blind. The most common anatomical sites of SVI/BL were retina in 31.1 % of children cornea in 24.3% and the whole globe in20.2%. Etiology was unknown in38.2% and childhood cause in 24%. 114 children (39.2%) had functional visual impairment (i.e., visual acuity <6/18 for light perception with navigational vision. In this study there were 150 children with refractive error and 20 children with amblyopia but amblyopia is not only due to refractive error as its causes are overlapping with other diseases like cataract and squint.

Conclusion

In summary, ametropia is still the most common cause of visual impairment and is easily avoidable with timely warranty providing these refractive error services to the children. A timely approach to cataract and squint surgery is required to reduce the visual impairment due to amblyopia. Removing barriers to the use of services for children with accessibility for visually impaired with provision of certificates for the visually impaired.

Financial support and sponsorship: NIL.

Conflicts of interest: There are no conflicts of interest.

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CASE REPORT

Pars Planitis in a Young Adolescence with Tuberculosis

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Abstract

A 26 year old male presented with complaints of sudden painless decreased vision in right eye (RE) with best-corrected visual acuity(BCVA) of 6/60 who was presented with no significant history of any systemic illness, no history of previous surgery, no history of any long term drug medication and no significant past history. Thorough right eye on slit lamp examination revealed AC reaction : 3+ with koeppe iris nodules. On fundus examination optic disc appears normal, membrane seen in vitreous with snow ball appearance in the periphery.

Introduction

Pars planitis is a specific type of intermediate uveitis that is idiopathic in nature; characterized as a vitritis with inflammatory condensates (snowballs) and pars plana fibrovascular exudation (snow banking). Pars planitis is a chronic condition that may reoccur for many years. Pars planitis usually occurs in children and young adults. A decrease in vision and an increase in floaters may be the only symptoms. Systemic associations of intermediate uveitis include sarcoidosis, multiple sclerosis (MS) and Lyme disease. Lesser associations include Behcet's disease, syphilis, tuberculosis, familial exudative vitreoretinopathy, toxoplasmosis, candidiasis, Eales' disease, Vogt Koyanagi-Harada syndrome, inflammatory bowel disease, cat-scratch disease and ocular lymphoma.

Case Report

A 26 year old male presented with chief complaints of sudden painless decreased vision with blurring of vision associated with floaters in right eye(RE) since 3 days. Best corrected visual acuity(BCVA) was 6/60 in right eye. No diplopia, photophobia, redness and watering was noticed. No history of ocular trauma, ocular surgery, any long term drug medication, systemic illness. On slit lamp examination, right eye(RE) showed keratic precipitates

(KP's) anterior chamber was of normal in depth, AC reaction:3+ , koeppe iris nodules , pupil small ,miotic and sluggish reactive to light , IOP 18mmHg (by NCT), EOM normal. Fundus examination of right eye(RE) revealed faint glow, media hazy, disc seems appears normal, membrane seen in vitreous with snowball appearance in periphery. On slit lamp examination, left eye(LE) showed anterior segment normal . IOP 11.0mmHg(by NCT), best corrected visual acuity(BCVA) was 6/6 partial, EOM normal. Fundus examination of right eye revealed glow present, optic disc and vessels normal with foveal reflex(FR) seen.

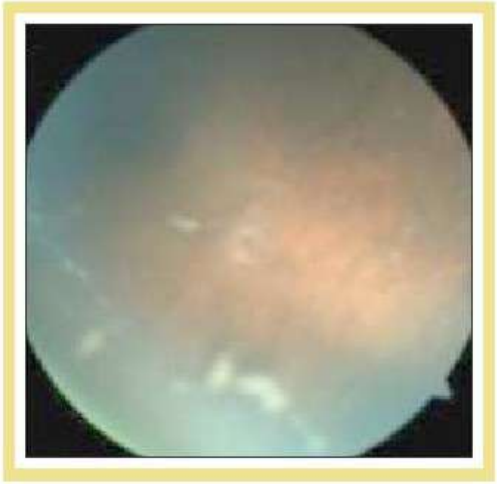

Figure 1 – Right eye (RE) fundus showing membrane seen in vitreous with snowball appearance in periphery.

In our patient ,on indirect ophthalmoscopy macular edema was absent so fundus fluorescein angiography or OCT was not done.

Investigations was done to rule out the cause of pars planitis in 26 year old male was Mantoux skin test: positive, VDRL/PRP rapid test: non reactive , radiographic chest X-Ray finding with upper lobe involvement with hilar lymphadenopathy, serum CRP: non reactive, TORCH profile: negative.

Discussion

Clinical work up for suspected pars planitis includes a complete history and review of systems with attention to diseases, infections, skin rashes and eye trauma. Complete ocular examination with anterior segment evaluation, intraocular pressure and indirect ophthalmoscopy and scleral depression of the ora serrata should be performed to look for snow banking. OCT or fluorescein angiography is useful in determining if macular edema is present and will aid in treatment plans. Additional testing to be considered includes CBC with differential, syphilis serology (FTA-ABS and RPR or VDRL), TORCH profile,

mantoux skin test, serum CRP, rheumatoid factor, chest radiography and lymes titre. If additional findings suggest multiple sclerosis(MS), an MRI with fat suppression of the brain and orbits, with and without gadolinium, should be considered especially if the patient is over the age of 25. Pars planitis has a higher prevalence with multiple sclerosis(MS) (3% to 27%) versus the general population (0.02% to 0.08%). Immunogenic studies have shown that the predisposition of multiple sclerosis(MS) and pars planitis may be associated with the HLA-DR15 allele. The most common ocular manifestations with multiple sclerosis (MS) are optic neuritis, diplopia, nystagmus and uveitis. Patient symptoms may include vision loss, pain on eye movement, double vision, oscillopsia, numbness or weakness in one or more limbs, tingling sensation or pain. The patient may also experience electric-shock sensations that occur with certain head movements (Lhermitte's sign), tremor, lack of coordination, fatigue, dizziness and a visual heat intolerance (Uhthoff's sign). If a patient presents with pars planitis these symptoms should be discussed with the patient and if they have neurologic signs of multiple sclerosis (MS) a referral to a neurologist is warranted. If multiple sclerosis (MS) is diagnosed, the term intermediate uveitis should be applied.

So on the basis of above findings the cause of pars planitis in our patient is tuberculosis.

Management

The treatment regimen of topical steroids(prednisolone acetate 1% QID) and cycloplegic (atropine sulphate 1% TDS) given to the patient and monthly follow up done. At 6th follow up patient vision improve to 6/9P in worst with BCVA.

Treatment of pars planitis is usually not indicated if visual acuity is 6/12 (20/40) or better and there are no signs of macular edema. The treatment regimen includes topical steroids (prednisolone acetate 1% qh-q6h) and a cycloplegic agent for any anterior chamber reaction. A posterior sub-tenon's injection of triamcinolone (40 mg/mL) or methylprednisolone acetate (40 mg/mL) is given when macular edema is present or if severe posterior inflammation is present. Oral non-steroidal anti-inflammatory drugs (NSAIDS) have been used in conjunction with corticosteroid injections for maintenance therapy. Topical NSAIDS may be added in patients with macular edema. Oral steroids (prednisone 40 to 60 mg daily) may be used for four to six weeks if injections are not possible or in severe bilateral cases.

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CASE REPORT

Ocular Manifestations of Wilson's Disease: A Case Report

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Introduction

Wilson's disease is an inborn error of copper metabolism also known as hepatolenticular degeneration which results from defective ATP7B gene inherited in an autosomal recessive pattern but can also be sporadic. The disease consists of copper accumulation in tissues like liver, eyes, and brain.

Its clinical consequences may vary from asymptomatic state to fulminant hepatic failure, chronic liver disease with neurologic and psychiatric manifestations. The disease is caused by homozygous or compound heterozygous mutations (the presence of two different mutant alleles) in the *ATP7B* gene that encodes a transmembrane copper-transporting ATP-ase that mediates the excretion of copper into bile and delivers copper for the functional synthesis of ceruloplasmin (the major copper-containing protein in the blood)[2]. This leads to copper accumulation in the blood leading to overload in hepatocytes. It is the free copper in the blood that determines copper intoxication and not the ceruloplasmin- bound copper. Treatment includes low copper diet, chelating agents such as D- penicillamine, hepatic transplantation for fulminant hepatic failure. Routine monitoring of serum copper and ceruloplasmin are indicated along with liver enzymes, INR, CBC with diff and urinalysis while on chelators. Also a yearly physical with 24-hour urinary copper should be performed while on medication.[3]

Case Report

A 25 year old female patient referred to ophthalmology OPD in JLN medical college Ajmer with complaints of hyperactive abnormal body movements, trembling of hands and feet for past 8- 10 years. Patient did not have any significant history of fever and hospitalization. Tremors – sudden onset gradually progressive since the age of 13 – 14 years for which she was taking treatment outside. The girl was the eldest child amongst 7 siblings from a non-consanguineous marriage. The other siblings were normal. The girl appeared to have delayed physical development and was underweight according to her age.

She had expressionless mask like face. On examination Pallor was present but there was no evidence of icterus, edema or lymphadenopathy. The liver was palpable 2 fingers firm with smooth margins. Spleen was also palpable 2 fingers inferior to left costal margin but there was no evidence of ascites. Neurological examination – Conscious, oriented, cooperative responding to commands, mental development was normal according to the age. Sensory system was normal. Tone of muscles was normal in all 4 limbs. Chorea abnormal body movements of entire body was observed, coarse resting tremors of both Upper limbs and Lower limbs and occasional intentional tremors in both hands. Girl could walk without support. Superficial and deep tendon reflexes were normal.

Ocular examination

	Right Eye	**Left Eye**
Lid and adnexa	Normal	Normal
Conjunctiva	Normal	Normal
Cornea	KF ring 1-2 mm in width superiorly 1-2 mm inferiorly. situated peripherally involving DM and appearing golden brown in colour.	KF ring 3-4 mm in width superiorly 1-2 mm inferiorly. situated peripherally involving DM and appearing golden brown in colour.
Anterior chamber and Pupil	Normal content and depth. Round regular reactive , no APD	Normal content and depth. Round regular reactive , no APD
Iris	Normal colour and texture	Normal colour and texture
Lens	Clear	Clear
EOM	Normal	Normal
UCVA Near vision Colour vision	6/6 N/6 Normal	6/6 N/6 Normal
Fundus	WNL	WNL

Figure 1 – Kayer Fleischer ring seen in Right and Left eye

Gonioscopy–Golden brown pigments on Trabecular meshwork which extended up to Schwalbe's line but Intraocular pressure was normal.

Radiological investigations

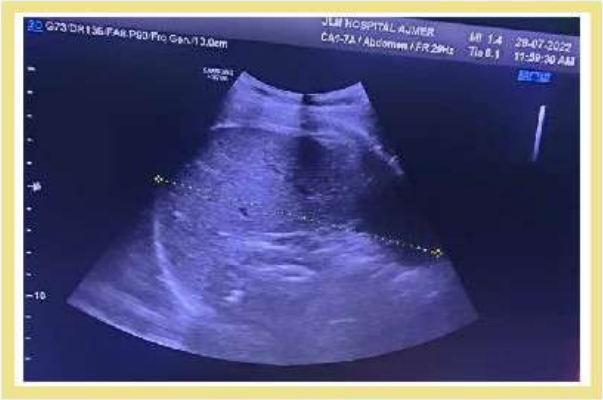


Figure 2 Splenomegaly altered echo texture

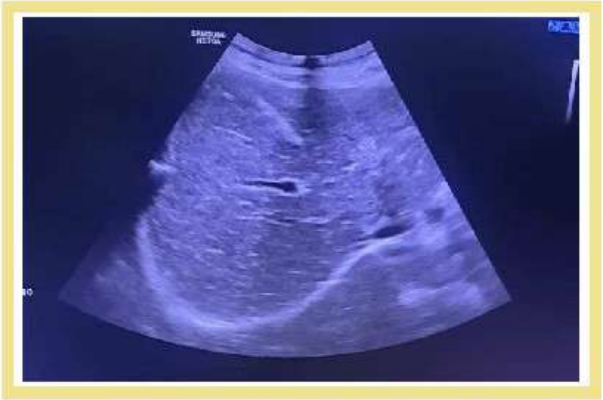


Figure 3 – echogenic liver parenchyma with

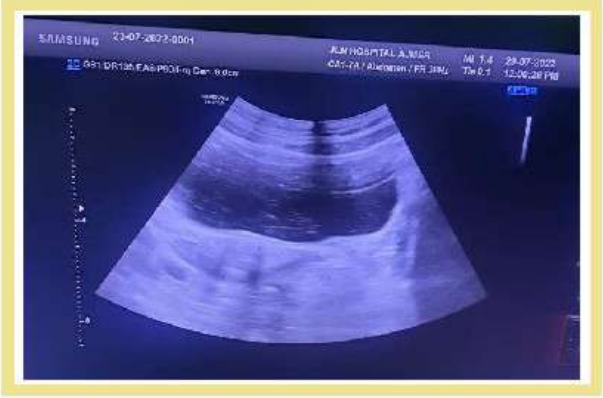
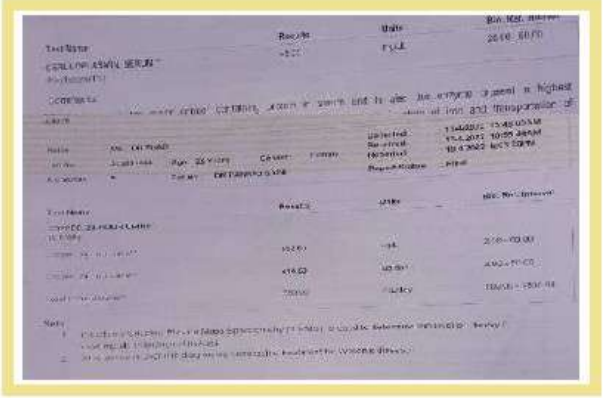


Figure 4 – Sediments in urinary bladder



Lab investigations

Diagnosis

The presence of KF ring with characteristic choreiform movements, splenomegaly and hepatomegaly, reduced serum ceruloplasmin and increased 24 hour urine copper pointed towards the diagnosis of Wilson's disease.

Discussion

The WHO estimates the prevalence of Wilsons disease as 1/ 10,000 to 1/30,000, prevalence of China – 5.8/100,000. There is no community based study for incidence and prevalence of Wilson's disease in India. Mean age of presentation in Indian population is 13.2 years[4]. Male preponderance have been observed in many of Indian literature but international literature reports equal incidence of Wilson's disease in males and females. The clinical spectrum of disease includes tremors, speech abnormalities, dystonia, high mental function abnormalities, seizures, cerebral signs and ocular signs such as Kayser – Fleischer ring , sunflower cataract and signs of hepatic dysfunction , out of which our patient presented with tremors and KF ring . Ocular manifestations may be the 1st presenting symptoms of Wilson's disease which must be recognized to prevent fatal outcomes. KF ring is an essential criteria for diagnosis of Wilson's disease and slit lamp examination is the gold standard for its detection.

KF ring was described by German ophthalmologist Bernhard Kayser in 1902 and Bruno Fleischer in 1903 . KF ring is due to copper deposition in the descments membrane of the cornea at the limbus, starts from Schwalbes line and extends up to 5 mm on the corneal surface. Starts from the superior pole as an arc from the 10- to the 2 o'clock position, followed by an arc in the inferior pole, and then encircles the entire cornea. Free copper loosely bound to albumin enters the aqueous humor and then enters the descements membrane. KF ring is seen in 95% of the patients with neurological manifestations and 65% with liver dysfunction. KF rings is also present in intraocular copper bodies (chalcosis) and primary biliary cirrhosis. The ring resolves in the reverse order either completely or partially following penicillamine therapy and after liver transplant. The density of copper deposits in the cornea at the diagnosis and during the follow up of Wilson's disease could determine the severity of the disease and response under chelator treatment.

Sunflower cataracts are rarely observed these cataracts have limited effect on patients visual acuity as these are not true cataracts and are caused by reversible copper deposition under the anterior capsule of lens .The objective of treatment is to prevent appearance of symptoms in asymptomatic subjects, prevent clinical deterioration in affected subjects, and can also be life-saving in cases of acute-on-chronic hepatitis. Treatment is based on the removal of copper excess by chelating agents such as penicillamine, trientine, or

tetrathiomolybdate or by blocking the intestinal copper absorption with zinc salts, with the ultimate goal of normalizing free plasmatic copper.

Dietary restriction of copper rich foods such as chocolate, nuts, mushrooms, crustaceans, soy, and gelatin should be followed. Also the use of cooking utensils containing copper is discouraged, and for tap water coming from copper pipes to be sufficiently safe for consumption, it must be left running for a few minutes.

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CASE REPORT

Post Fever Bilateral Retinal Vasculitis and Macular Edema in an Immunocompetent Individual: A Case Report

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Abstract

We describe a case of dengue fever–associated bilateral retinal vasculitis and macular edema in an immunocompetent individual. A 55-year-old Indian man had presented with sudden onset diminution of vision in the both eyes for the past 22 days. Fundus showed multiple intra-retinal hemorrhages and a patches of retinitis along the superotemporal and infero-temporal arcade in right eye and along superotemporal arcade involving macula in left eye. Optical coherence tomography and fluorescein angiography were indicative of macular edema. He was treated with oral steroids along with supportive treatments. A near complete anatomical and functional recovery was noted. Dengue associated eye disease has a spectrum of ocular manifestations, ranging from nonspecific symptoms to severe sight-threatening ocular involvement. Our case depicts the significance of awareness of the ophthalmic complications of dengue fever among both ophthalmologists and physicians.

Introduction

Dengue fever, an arboviral infection, is the most common mosquito-borne viral disease in humans. Dengue infection is now endemic in many countries, particularly in the Southeast Asia, Indian subcontinent, and the Americas.[1]Among them, Asia alone represents 70% of the disease burden.[2]Recently, there has been a spike in the number of reported cases of dengue and associated ocular complications from all over the world, including India.[3,4] This may be attributed to the rapid resurgence of the principal vector, due to overcrowding and urbanization along with the hot and humid climate of the tropical countries.[5]

Ophthalmic manifestation of dengue is rare but diverse. Usually the posterior segment of the eye is involved in the form of maculopathy, macular edema, optic neuropathy, vasculitis, and vein occlusion.[6] We report a rare ocular

complication of dengue fever as bilateral retinal vasculitis with macular edema and was treated successfully with oral steroids and supportive treatments.

Case Report

A 55-year-old male patient presented to the eye outpatient department with the complaints of sudden blurring of vision in both the eyes for the last 22 days. On examination, the best-corrected visual acuity (BCVA) in the right eye was FC 2 mt and FC 5 mt in the left eye. Applanation tonometry recorded an intraocular pressure (IOP) of 12 mmHg in both the eyes. Slit lamp examination showed normal anterior segment in both the eyes. Fundus examination of both the eyes showed multiple intra-retinal hemorrhages, dilated and tortous vessels and patches of retinitis along the superotemporal and inferotemporal arcade along with macular edema(Fig.1 A and B). Fluorescein angiography (FA) was carried out and it revealed dilated, tortous vessels and capillary non perfusion area in both the eyes (Fig.2 A and B). He had a history of, serology confirmed, dengue fever 45 days back and was a febrile at presentation. He did not have any petechial rash over his body, nor any other systemic manifestation. His blood counts were within normal limits with a platelet count of 258,000/mm3 at the time of presentation. The total leukocyte count was also within normal limits. Erythrocyte Sedimentaion Rate was 20mm/hour. He was diagnosed with classic dengue fever by his physician using IgM positive serology. Other possible diagnosis were ruled out with the help of a normal C-reactive protein, serum ACE levels, antinuclear antibodies, negative VDRL test, negative serology for toxoplasmosis, HIV, Herpes virus, Cytomegalovirus, hepatitis, Rheumatoid factor, Mantoux of 12 mm induration, normal chest X-ray and a negative WIDAL test. Thus, a diagnosis of post Dengue fever vasculitis with macular edema was made. The patient was started on oral steroids 60mg daily. On follow up after 15 days, the vision improved to 6/12 in both the eyes, with almost complete resolution of macular edema on OCT.

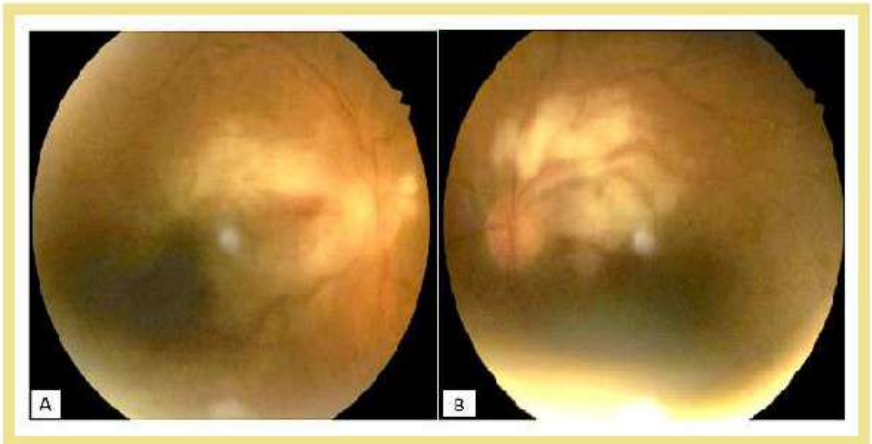

Figure 1 (A & B): (A) Fundus picture of right eye at presentation; (B) Fundus picture of left eye at presentation.

At 1 month of follow-up, BCVA was 6/9 in both eyes, and vascular tortuosity and inflammation had resolved near completely (figure 3).The steroids were tapered gradually thereafter.

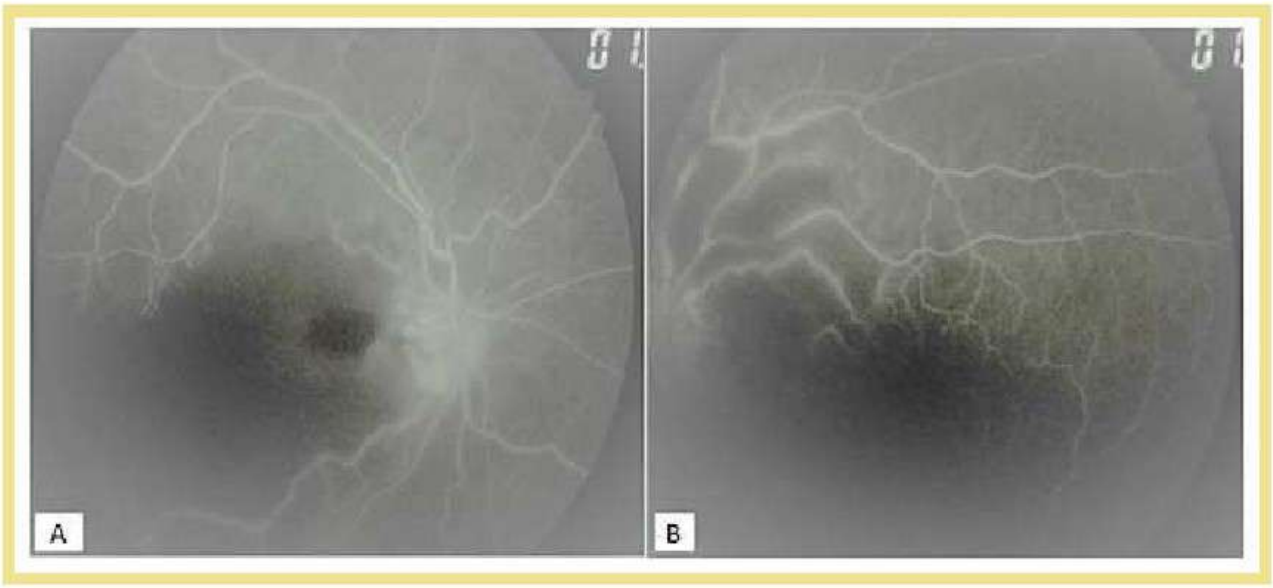

Figure 2 (A & B): (A) FA picture of right eye; (B) FA picture of left eye at the time of presentation

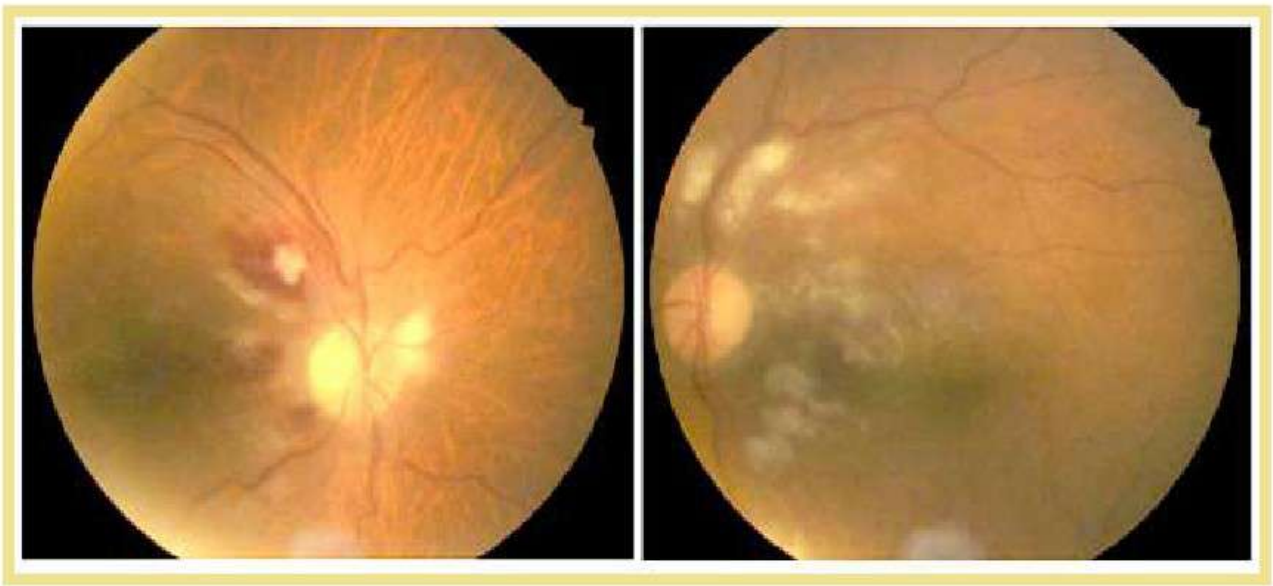

Figure 3: Fundus photographs showing resolution of retinopathy in both eyes.

Discussion

Dengue fever, an arboviral infection, is transmitted by infected female mosquitoes Aedes aegypti and Aedes albopictus.[7] It causes a wide spectrum of disease ranging from mild self remitting acute flu-like illness to a potentially fatal Dengue haemorrhagic fever/ shock syndrome. Infection with one serotype provides lifelong immunity against subsequent infection by the same serotype but only partial immunity against infection with other serotypes.[8]The diagnosis is usually done by IgM and IgG titres that is not positive until at least 5 days after fever.

Dengue fever related ocular involvement is rare and occurs in less than 10% of patients with symptomatic dengue virus infection.[9]Ophthalmic manifestations in dengue can vary from subtle signs such as subconjunctival hemorrhage, uveitis to more serious complications such as maculopathy, foveolitis, macular edema, retinal vascular occlusion, optic neuropathy and choroiditis.[10]The ocular complications usually manifest when they are at the nadir of thrombocytopenia.[11]The correlation of thrombocytopenia and ocular complications has also been explained to occur due to endothelial dysfunction, immune complex deposition, or retinal capillary ischaemia.[9]Average onset time of dengue retinitis was seven days (range1-28 days) after the onset of fever.[9] This delay is due to the immune mediated pathophysiology of dengue related eye disease.[13]

Treatment of dengue eye disease has largely been conservative, with spontaneous recovery.[12-14]The patients who have significant ocular impairment may be considered for oral or intravenous steroid treatment. Various dengue serotypes may cause infection in the same patient later in life and do not provide cross immunity.

Conclusion

We report this case to highlight the fact that it is of paramount importance for physicians and ophthalmologists to be aware of the extended spectrum of the dengue eye disease for timely diagnosis and educate the patients about possible ocular symptoms long after the dengue has been treated. Early diagnosis and management may help in complete recovery of vision in such eyes.

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CASE REPORT

Early Presentation of Advanced Diabetic Retinopathy: A Diagnostic Dilemma

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A 46-year-old male presented with complaints of decreased vision in both eyes. Decrease in vision was sudden, painless in LE with BCVA of HM. Decrease in vision was gradual, painless and progressive in RE with BCVA of CF ½ mt. He was diagnosed with diabetes 1 year back and has been taking oral hypoglycaemic agents regularly and had moderate glycemic control. Thorough eye examination revealed macular hole with traction retinal detachment in superotemporal quadrant in RE and combined rhegmatogenous and traction retinal detachment in LE. He did not undergo any ophthalmic examination earlier and directly presented with advanced diabetic eye disease.

Introduction

India being, rapidly becoming Diabetic capital of the world, cases of Diabetic Retinopathy are also rising. Duration of diabetes is the biggest risk factor in the incidence of retinopathy. Incidence of retinopathy is high in juvenile onset diabetes compared to adult onset. Regular ophthalmic examination helps in early diagnosis and treatment of diabetic retinopathy, thus preventing risk of severe visual loss.

Case Report

A 46 year old male presented with chief complaints of decreased vision in both eyes. BCVA was counting fingers-1/2 mt in RE and hand movements in LE. Decrease in vision was sudden and painless in left eye 2 months back. In right eye, decreased vision was gradual, painless and progressive in nature since 1 month. Floaters present in left eye.

No diplopia, coloured halos, photophobia, redness, watering was noticed.

Patient had history of using herbal eye drops 1 month back.

No history of ocular trauma, ocular surgery, infection

He was diagnosed with type-2 diabetes mellitus 1 year back and has been using oral hypoglycaemic agents regularly since then, had moderate glycemic control. He did not undergo any ophthalmic examination in the last 1 year.

On slit lamp examination, BE showed early nasal pterygium, clear cornea, AC normal depth, iris normal pattern, pupil normal size, sluggishly reacting to light and greyish white lens IOP in RE and LE 13- AND 10-mm Hg respectively.

Fundus examination of right eye revealed optic disc normal in shape, size and margin with CDR of 0.3:1. Old vascular occlusion with macular hole with traction retinal detachment in superotemporal quadrant. (fig 1)

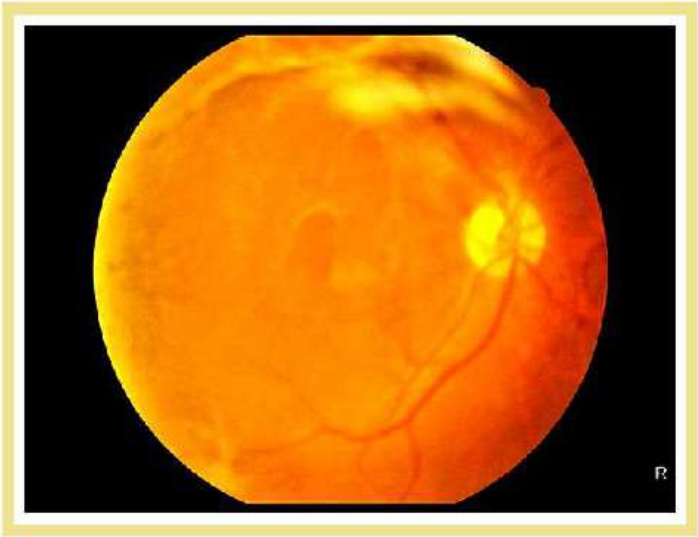

Figure 1- RE fundus showing old vascular occlusion with macular hole with traction retinal detachment in supero-temporal quadrant.

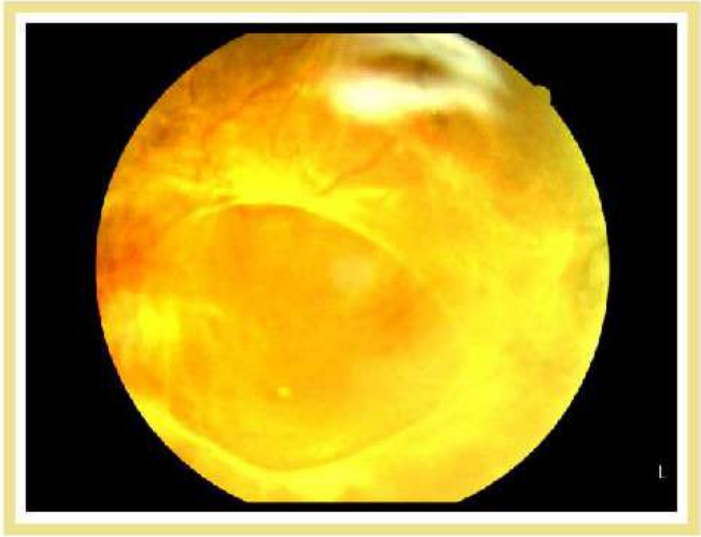

Figure 2- LE fundus showing combined rhegmatogenous and traction RD with fibrovascular proliferation.

LE fundus revealed optic disc normal in shape, size and margin with CDR of 0.3:1. Combined rhegmatogenous and traction retinal detachment with fibrovascular proliferation. (Figure 2).

B scan also revealed RE supero-temporal traction RD and LE combined rhegmatogenous and traction RD (Figures 3 and 4).

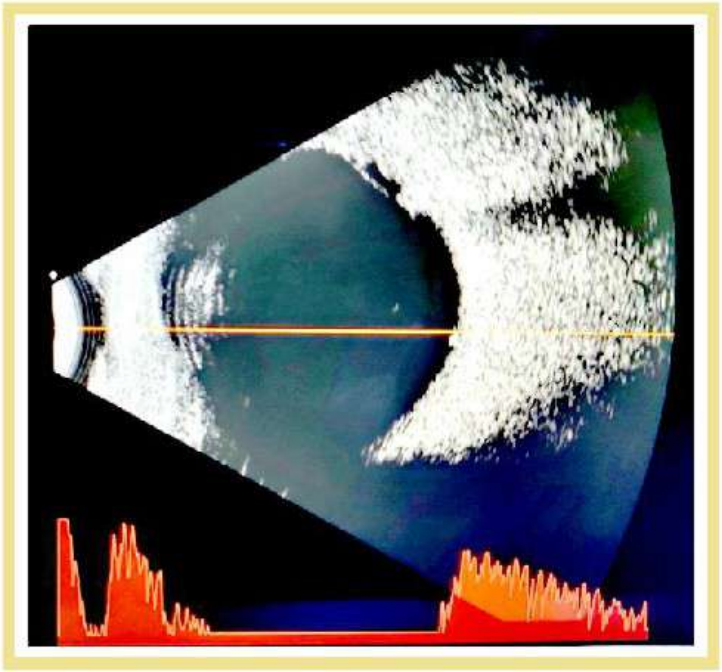

Figure 3- RE Bscan showing traction RD in supero temporal quadrant.

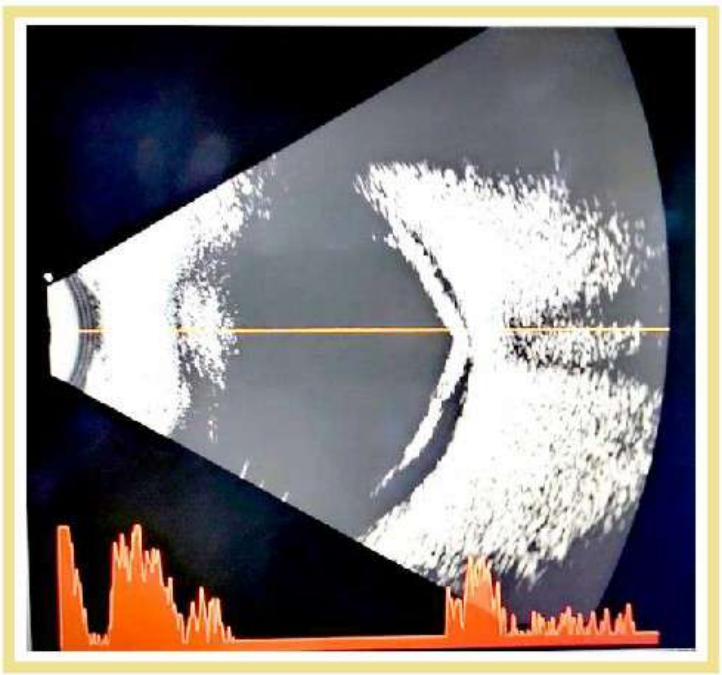

Figure 4- LE B scan showing Combined RD.

Discussion

Diabetes can impact the eye and vision by affecting many structures of eye ranging from cornea to retina. Diabetic retinopathy is most visible and important change that takes place in eyes due to diabetes. Longer duration of diabetes increases risk and severity of retinopathy. Nearly 100% of juvenile diabetics develop retinopathy after 20 years of suffering from diabetes. Poor blood sugar level control also impacts the development of retinopathy. Severity of retinopathy is also associated with high triglyceride levels.

Non proliferative DR shows early changes such as microaneurysms, intraretinal haemorrhages, venous beading, hard exudates, IRMAs.

Proliferative DR is characterised with neovascularisation, vitreous haemorrhage, fibrous bands and progressing to retinal detachment causing severe visual loss.

Ophthalmologic treatment is indicated when neovascularization develops, carrying the risk of vitreous haemorrhage and traction retinal detachment. Panretinal photocoagulation improves the balance of retinal oxygen demand and supply and therefore reduces the neovascular stimulus. In case of diabetic macular edema, focal retinal photocoagulation or intravitreal drug injections can be given to stabilize capillary permeability in order to preserve vision. In advanced cases, treatment via pars-plana-vitrectomy has to be evaluated.

Incidence of advanced disease is greatly reduced due to anti-VEGFs and vitrectomy, thus emphasising on providing better treatment to patients at early stages. Emerging technologies with retinal imaging tools and artificial intelligence have increased access to care for diabetic people.

Diabetic retinopathy is a severely debilitating disease which can be prevented with strict control of blood sugar levels, complete ophthalmic examination as soon as patient is diagnosed with type-2 DM and after 3 years in type-1 DM as per new recommendations. Regular eye check-ups will help in early diagnosis and treatment and prevention of severe visual loss.

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CASE REPORT

Presentation and Management of Squamous Cell Carcinoma of Eyelid

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Abstract

Squamous cell carcinoma (SCC) is an invasive epithelial malignancy showing keratinocytic differentiation. It is the second most common neoplasm of the eyelids, comprising 5-10% of all eyelid malignancies. We report a case of 70 year old female patient presented to the eye clinic with itching, sticky discharge and swelling over the left upper lid (LUL). Excisional biopsy of the mass was suggestive of poorly differentiated SCC. Management consisted of complete tumour excision and full thickness defect of the eyelid was reconstructed with Cutler-Beard technique.

Introduction

Squamous cell carcinoma (SCC) is an invasive epithelial malignancy showing keratinocytic differentiation. It is the second most common malignant neoplasm of the eyelids,[1]comprising 5-10% of all eyelid malignancies. The incidence for eyelid SCC has been reported to be between 0.09 and 2.42 cases per 100000 population.[2] Extrinsic risk factors include ultraviolet light/actinic damage and exposure to arsenic, hydrocarbons, radiation, or immunosuppressive drugs.[3] Intrinsic risk factors include albinism, pre-existing chronic skin lesions and genetic skin disorders such as xeroderma-pigmentosum and epidermodysplasia verruciformis. Early diagnosis and management of the tumour and regular follow-up can largely influence the survival rate of the patient.

Case Report

A 70 year old female presented with itching, sticky discharge and swelling over the (LUL) for 2 months. Swelling was gradual in nature, progressive, and painless with no limitation of eye movement.

Figure 1 showing irregular lesion in left upper eyelid.

There was history of trauma to the wooden stick 3 months back. No history of pain, burn and chronic exposure to sunlight. No history of skin cancer amongst the other members of the family.

General examination: patient was well nourished, average built, well oriented to time, place and person. Systemic examination was within normal limits.

Ocular examination:

On examination, the lesion was irregular, no hyperkeratotic changes seen. On palpation, the mass was approximately 40×24×27mm in size. Shape was irregular and firm in consistency. The overlying skin was partly excoriated and partly nodular, extending up to underlying conjunctiva. The contour of the lid margin was lost with loss of eyelashes. Other ocular structures were within normal limits. No associated preauricular and submandibular lymphadenopathy was noted.

Investigation

A MRI orbit shows lobulated heterogeneous signal intensity mass lesion appearing showing hyperintense on T2 and STIR and hypointense on T1W sequence is seen involving left upper eyelid abutting the anterior chamber anterior aspect of lower eye led with ill-defined margins with associated adjacent fat stranding and measuring approx. 38×22×24mm in size-suggestive of neoplastic etiology. Figure 2 to 5 showing MRI imaging of extension of the tumour in different compartments of eye.

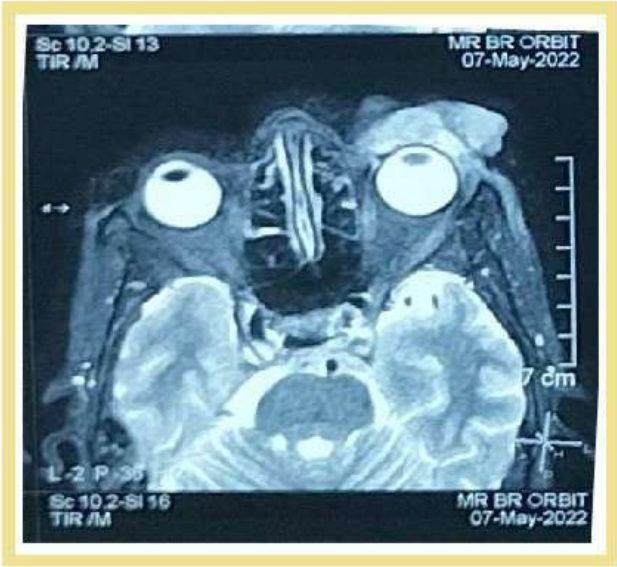

Figure 2

Figure 3

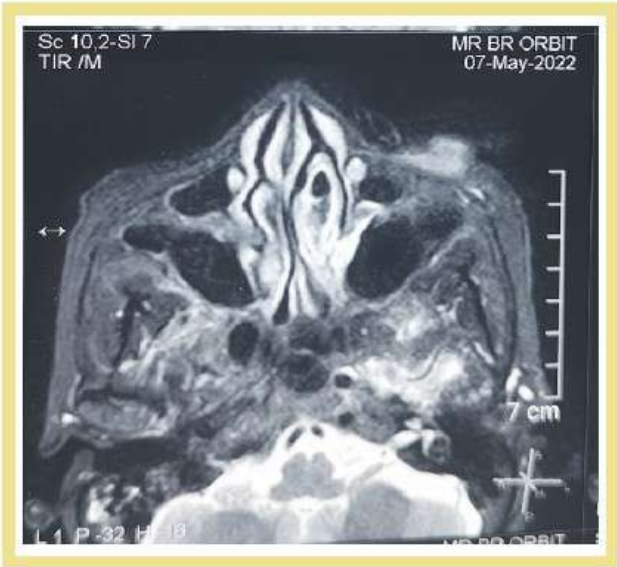

Figure 4

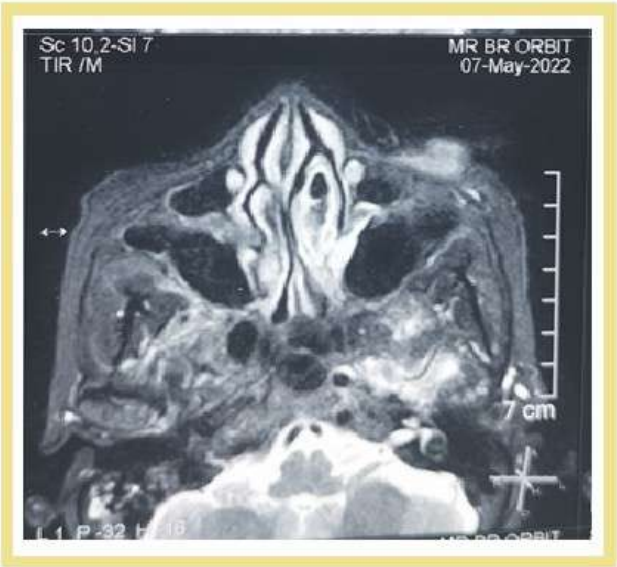

Figure 5

An excisional biopsy of the mass showed neoplastic cells, high nucleocytoplasmic ratio, hyperchromatic nuclei and prominent nucleoli suggestive of poorly differentiated squamous cell carcinoma. CT brain and orbit was done to rule out metastasis. All blood investigations were done in order to perform the surgery.

Treatment

Patient's written consent was taken before the surgery. She underwent wide excision of the mass along with full thickness excision of upper eyelid under general anaesthesia. The resected mass was sent for histopathological examination for confirmation of the type of tumour. The full thickness defect of the upper eyelid was reconstructed with Cutler-Beard technique. At first, a full thickness lower eyelid flap was divided into anterior (skin & muscle) and posterior (conjunctiva) and sutured with upper eyelid defect.

Figure 6 showing closure of left upper eyelid defect.

After 6 weeks the eyelids were opened. On follow-up no perineural invasion, no lymph node and no distant metastasis were noted. The patient was doing well both functionally and cosmetically.

Figure 7 showing normal left upper eyelid.

Discussion

Eyelid SCC is a relatively uncommon but potentially fatal disease. It is responsible for considerable morbidity; however, if detected early and treated adequately, the prognosis is generally excellent and death and disability can be reduced.[4] The clinical presentation varies and histological examination is required for accurate diagnosis. Eyelid skin cancers occur most often on the lower lid but may also occur on the upper lid, medial or lateral canthal area, eyebrow, or adjacent eyelid skin.Soysal HG et al. in a retrospective study reported that tumors with well-differentiated and moderately differentiated histology were less likely to metastasize than those with poorly differentiated histology.[5]Perineural spread is an adverse prognostic sign, which requires consideration of postoperative prophylactic radiotherapy. Orbital invasion is a rare complication, but if recognised early can be treated effectively with exenteration. Regardless, the type of management, all patients with eyelid SCC should be advised for lifelong follow-up. Prevention by minimising sun exposure, remains of prime importance in minimising the morbidity and mortality of this disease.

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CASE REPORT

A Rare Conjunctivitis with Extreme Discomfort: A Case Report

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History

A 35year old male patient presented to the emergency OP with intense pain, watering and redness in both eyes since last three days. He also complained of sticky feeling in both eyes. He had taken treatment elsewhere for last two days (a steroid antibiotic eye drop), but had felt no relief. His systemic history was unremarkable, and he denied having taken any systemic medication or suffering from any drug allergy.

On Examination

His visual acuity was 6/9 right eye, 6/9p left eye respectively with no improvement on pinhole. Purulent discharge, marked conjunctival congestion with mild chemosis were noted as shown in figure 1 (A-C). On everting upper and lower lids a whitish membrane was present involving the forniceal and palpebral conjunctiva. Cornea was clear. Pupils were round regular and reactive. Anterior chamber was quiet. His pre auricular lymph nodes were enlarged.

Figure 1 (A-C). Showing Purulent discharge, marked conjunctival congestion with mild chemosis.

Management

Conjunctival swab was taken after instillation of topical anaesthetic agent, which was sent for microbiological evaluation. Prior steroid drops were stopped. The membrane was peeled off with the help was swab stick and this was repeated daily till it stopped forming (Figure 2). Started him on Moxifloxacin eye drop 6times a day Tobramycin eye drop 6times a day Homatropine eye drop thrice a day (for ciliary spasm), Carboxymethylcellulose eye drop 6 times a day Ciprofloxacin eye ointment bed time. Tablet ciprofloxacin 500mg twice a day for 5 days Tablet Serratopeptidase diclofenac twice a day Vitamin C supplements Frequency of eye drops were tapered, after treatment for 5days. Complete resolution was noted.

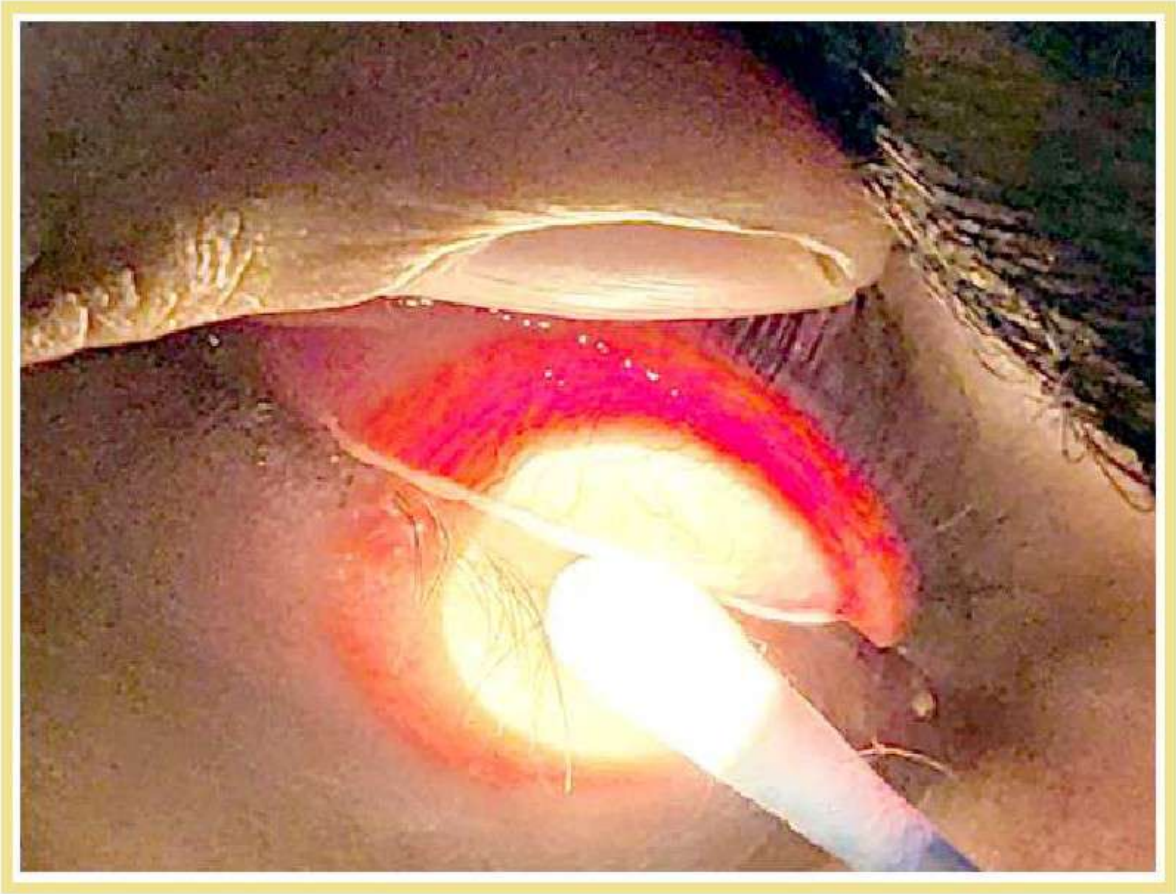

Figure 2. Peeling of the membrane with cotton tip applicator.

Discussion

Pseudomembranous conjunctivitis is an inflammatory condition of the conjunctiva, characterized by conjunctival congestion, mucopurulent discharge and pseudomembrane formation. Pseudomembrane consist of fibrin rich coagulated exudate adherent to the inflamed palpebral conjunctival epithelium. It can be easily peeled off with a swab stick , leaving behind underlying

epithelium. Removing pseudomembranehas shown to dramatically reduced symptoms and shorten the normal course of infection. Causative organism are Streptococcus pyogens, Cornyebacterium depterium, Niesseria gonorrohea, Adenovirus, Ligneous conjunctivitis (rare). Our patient was most probably a case of adenoviral conjunctivitis (seasonal outbreak; enlarged Preauricular lymph nodes) superimposed with bacterial infection. Conjunctival culture revealed Streptococci.

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CASE REPORT

The Dark Side of Headache! A Case Report of Headache with Bilateral Papilledema with Lateral Sigmoid Sinus Thrombosis

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Department of Ophthalmology, J.L.N. Medical college and Hospital, Ajmer, Rajasthan
E-mail: ayushigupta011@gmail.com

Introduction

Headache is a pain around the head, pain being above the eyes or the ears, behind the head(occipital), or the back of the upper neck. All headaches are considered primary or secondary headaches. Primary headaches are not associated with other diseases, examples are migraine headaches, tension headaches, and cluster headaches. Secondary headaches are caused by other diseases the associated disease may be minor or major.

The World Health Organization(WHO) estimates that over half of the global population had at least one attack of headache during the past year and within the adult population up to 47% have a general headache disorder. The WHO has stated that headaches are under-treated, under-recognized and under-reported [1].With the increase in the number of patients with headache more and more patients are being referred to ophthalmology clinics to rule out secondary causes of headache –such as refractive errors, raised intraocular pressure, papilledema which will in turn help to rule out the intracranial causes of headache. *Here we present a case of a 42- year -old male presenting with sudden onset headache and gradual loss of vision in one eye. With no prior history of any other disease.*

Case Report

A 42- year- old male was referred to eye OPD for fundus evaluation. The patient had a history of sudden onset severe generalized persistent throbbing headache since 1-2 months which was acute in onset and not relieved by oral analgesics, associated with vomiting with a progressive diminution of vision in the Right eye from the last 10 days. No history of seizures, trauma, or head injury Past

History – Not a known case of Diabetes, Hypertension, Bronchial Asthma or any blood disorders and was not on any medication.

Patient firstly presented to an ophthalmologist in July 2022 with complaints of headache where his vision was recorded OD=6/6 OS=6/6. He was prescribed analgesics for pain relief. The patient did not have any improvement in his symptoms for 1 week and noticed his vision deteriorating in one eye so he went to another ophthalmologist where his vision was recorded as OD=6/12 OS=6/6. Fundus showed early signs of papilledema in both eyes (Right eye > Left eye). The CT scan Head and Orbit report was normal. He was then referred to a neuro-physician where he was completely investigated. His hemogram showed mild leukocytosis and raised blood sugar levels, HbA1c level - 6.2 which is in the pre-diabetic range. There was no improvement in his symptoms so he presented to Neuro physician at JLN hospital Ajmer and was referred to us for fundus evaluation.

Systemic examination – Conscious, oriented, responding to commands. No evidence of Pallor, icterus, or cyanosis. The patient was slightly overweight. General systemic examination was normal.

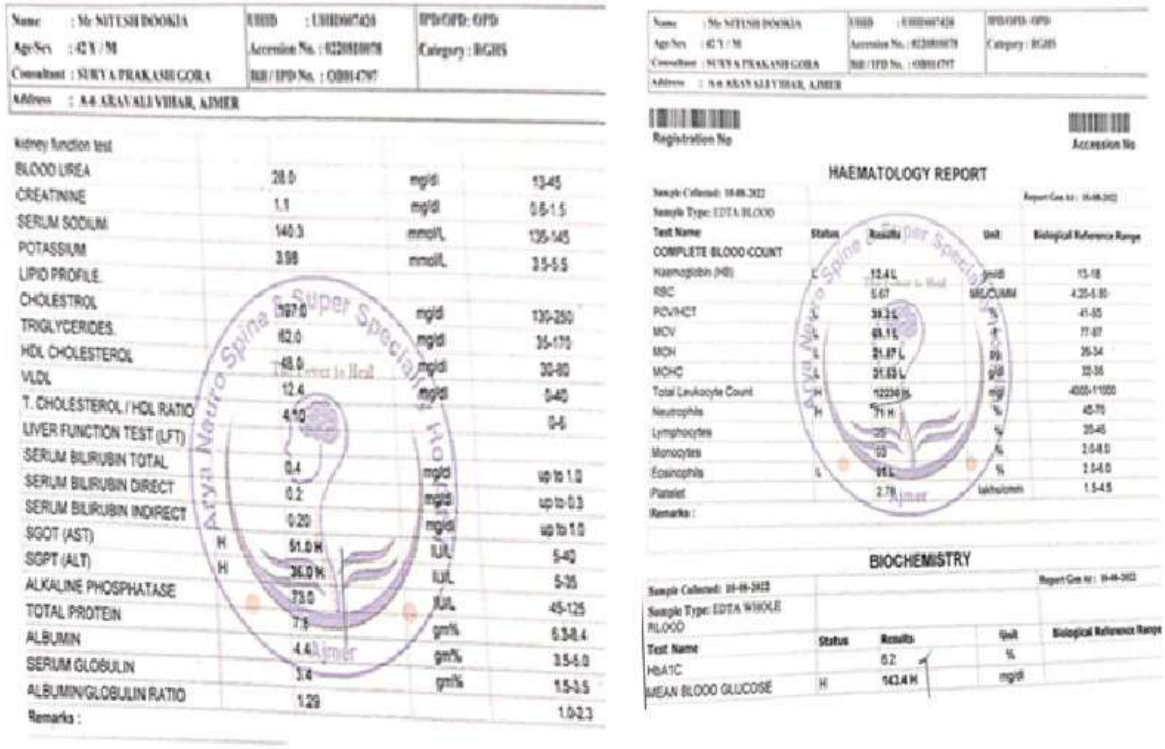
Ophthalmic Examination

	Right Eye	**Left Eye**
Lid and adnexa	Normal	Normal
Conjunctiva	Congested	Normal
Cornea	Clear	Clear
Anterior chamber and Pupil	Normal content and depth. Round regular Relative Afferent Pupillary Defect present	Normal content and depth. Round regular reactive, no APD
Iris	Normal colour and texture	Normal colour and texture
Lens	Clear	Clear
EOM	Normal	Normal
Fundus	Media clear Disc margins blurred Complete elevation of cup, circumferential Halo, obscuration of major vessels on disc Vessels tortuous, splinter hemorrhages present around the disc	Media clear Disc margins blurred Complete elevation of cup, circumferential Halo, obscuration of major vessels on disc Vessels tortuous
UCVA	PL + PR accurate	6/12

Investigations

Blood test showed no abnormal biochemical abnormalities except mild leukocytosis was present. CT scan and MRI of Head and orbit were normal and Lumbar puncture demonstrated an elevated CSF pressure (320 mm of H2O)with raised protein in CSF (229 mg/dl), with normal sugar and chloride levels. ell count was less than 5 mm^3 , cell types were predominately lymphocytes . To further investigate MR venography was performed which showed a narrowed left sigmoid sinus (Partial thrombosis of left sigmoid sinus).

Patient was started on Inj. Manito i/v 100cc 8 hourly , Inj. LMWH 0.6 cc s/c 12 hourly ,Tab Acetazolamide 500 mg TDS , Tab. Warfare 5 mg 1 OD, Tab Telmisartan + Clinidipine 1 OD and BP , PR and sugar charting .



Name : Mr NITUSH DOOKIA	UHID : UHID007420	IPD/OPD: OPD
Age/Sex : 42 Y / M	Accession No. : 022080078	Category : RGHS
Consultant : SURYA PRAKASH GORA	Bill / IPD No. : OBH4797	
Address : A-6 ARAVALI VIHAR, AJMER		

Kidney function test			
BLOOD UREA	28.0	mg/dl	13-45
CREATININE	1.1	mg/dl	0.6-1.5
SERUM SODIUM	140.3	mmol/L	135-145
POTASSIUM	3.98	mmol/L	3.5-5.5
LIPID PROFILE			
CHOLESTROL	197.0	mg/dl	130-200
TRIGLYCERIDES	62.0	mg/dl	35-170
HDL CHOLESTEROL	48.0	mg/dl	30-80
VLDL	12.4	mg/dl	0-40
T. CHOLESTEROL / HDL RATIO	4.10		0-5
LIVER FUNCTION TEST (LFT)			
SERUM BILIRUBIN TOTAL	0.4	mg/dl	up to 1.0
SERUM BILIRUBIN DIRECT	0.2	mg/dl	up to 0.3
SERUM BILIRUBIN INDIRECT	0.20	mg/dl	up to 1.0
SGOT (AST)	H 51.0 H	IU/L	5-40
SGPT (ALT)	H 36.0 H	IU/L	5-35
ALKALINE PHOSPHATASE	73.0	IU/L	45-125
TOTAL PROTEIN	7.8	gm%	6.3-8.4
ALBUMIN	4.4	gm%	3.5-5.0
SERUM GLOBULIN	3.4	gm%	1.5-3.5
ALBUMIN/GLOBULIN RATIO	1.29		1.0-2.3
Remarks :			

Registration No — Accession No

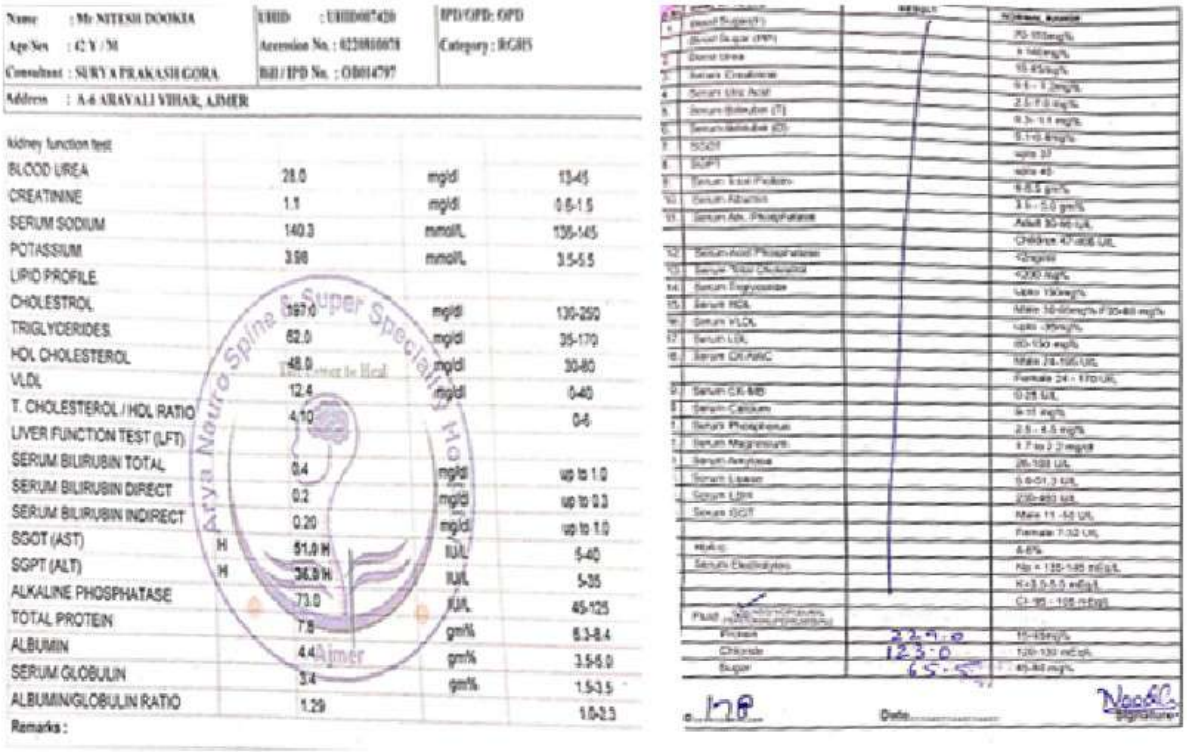
HAEMATOLOGY REPORT

Test Name	Status	Results	Unit	Biological Reference Range
COMPLETE BLOOD COUNT				
Haemoglobin (Hb)	L	12.4 L	gm/dl	13-18
RBC		5.67	MIL/CUMM	4.25-5.80
PCV/HCT	L	39.2 L	%	41-53
MCV	L	69.1 L	fl	77-97
MCH	L	21.87 L	pg	26-34
MCHC	L	31.63 L	g/dl	32-36
Total Leukocyte Count	H	12234.56	/cmm	4000-11000
Neutrophils	H	71 H	%	40-70
Lymphocytes		20	%	20-40
Monocytes		03	%	2.0-8.0
Eosinophils	L	01 L	%	2.0-8.0
Platelet		2.78	lakhs/cmm	1.5-4.5
Remarks :				

BIOCHEMISTRY

Test Name	Status	Results	Unit	Biological Reference Range
HbA1C		6.2	%	
MEAN BLOOD GLUCOSE	H	143.4 H	mg/dl	

Figure 1 – Normal biochemical report



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Age/Sex	: 42 Y / M	Accession No.	: 0220810078	Category : RGHS
Consultant	: SURYA PRAKASH GORA	Bill / IPD No.	: OB014797	
Address	: A-6 ARAVALI VIHAR, AJMER			

kidney function test			
BLOOD UREA	28.0	mg/dl	13-45
CREATININE	1.1	mg/dl	0.6-1.5
SERUM SODIUM	140.3	mmol/L	135-145
POTASSIUM	3.98	mmol/L	3.5-5.5
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CHOLESTROL	197.0	mg/dl	130-250
TRIGLYCERIDES	82.0	mg/dl	35-170
HDL CHOLESTEROL	48.0	mg/dl	30-80
VLDL	12.4	mg/dl	0-40
T. CHOLESTEROL / HDL RATIO	4.10		0-6
LIVER FUNCTION TEST (LFT)			
SERUM BILIRUBIN TOTAL	0.4	mg/dl	up to 1.0
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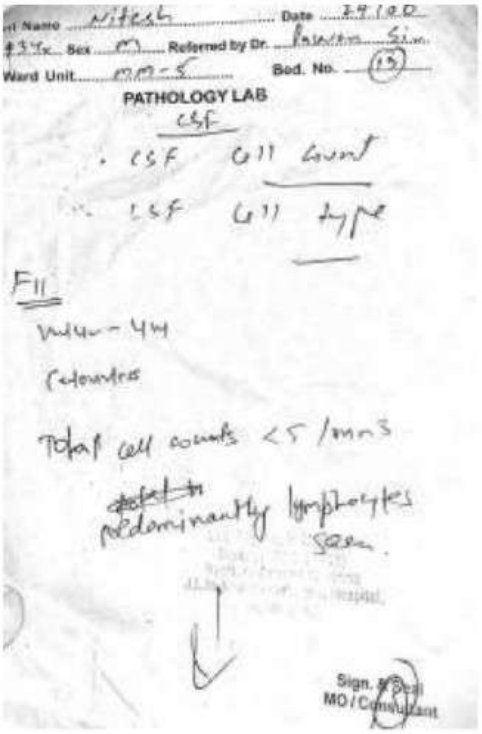
SERUM GLOBULIN	3.4	gm%	1.5-3.5
ALBUMIN/GLOBULIN RATIO	1.29		1.0-2.3
Remarks :			

Figure 2 – Hematological report showing Leukocytosis



Name Nitesh Date 29.10.22
Age/Sex M Referred by Dr. Paliwal Sir
Ward Unit MM-5 Bed. No. 13
PATHOLOGY LAB
CSF
CSF Cell Count
CSF Cell type
FH
WBC - Nil
Colour
Total cell counts < 5 /mm3
predominantly lymphocytes
Sign. & Seal
MO / Consultant

Figure 3 – Lumber puncture – Raised CSF protein with normal chloride and sugar levels and predominant cells.

Radiological Investigations

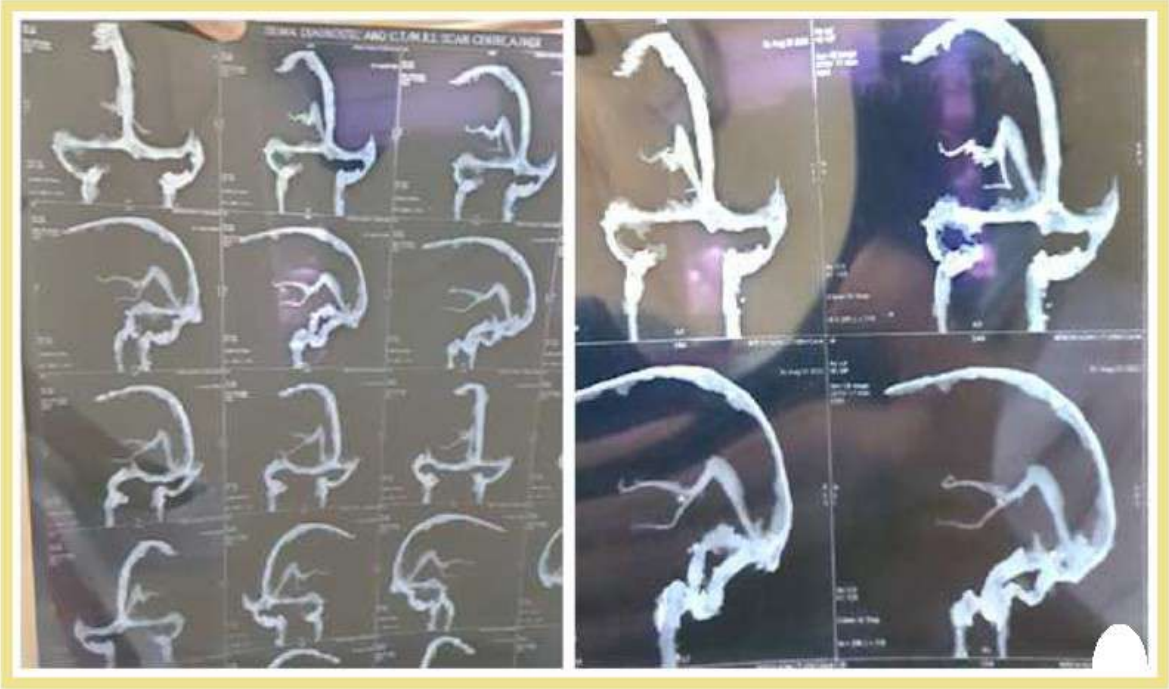


Figure 4 – MR Venography showing Partial Left Sigmoid Sinus Thrombosis.

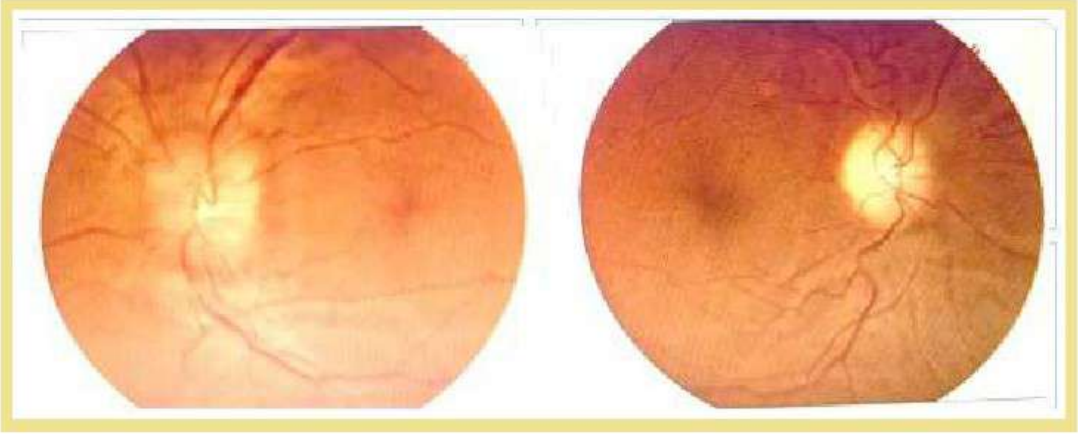
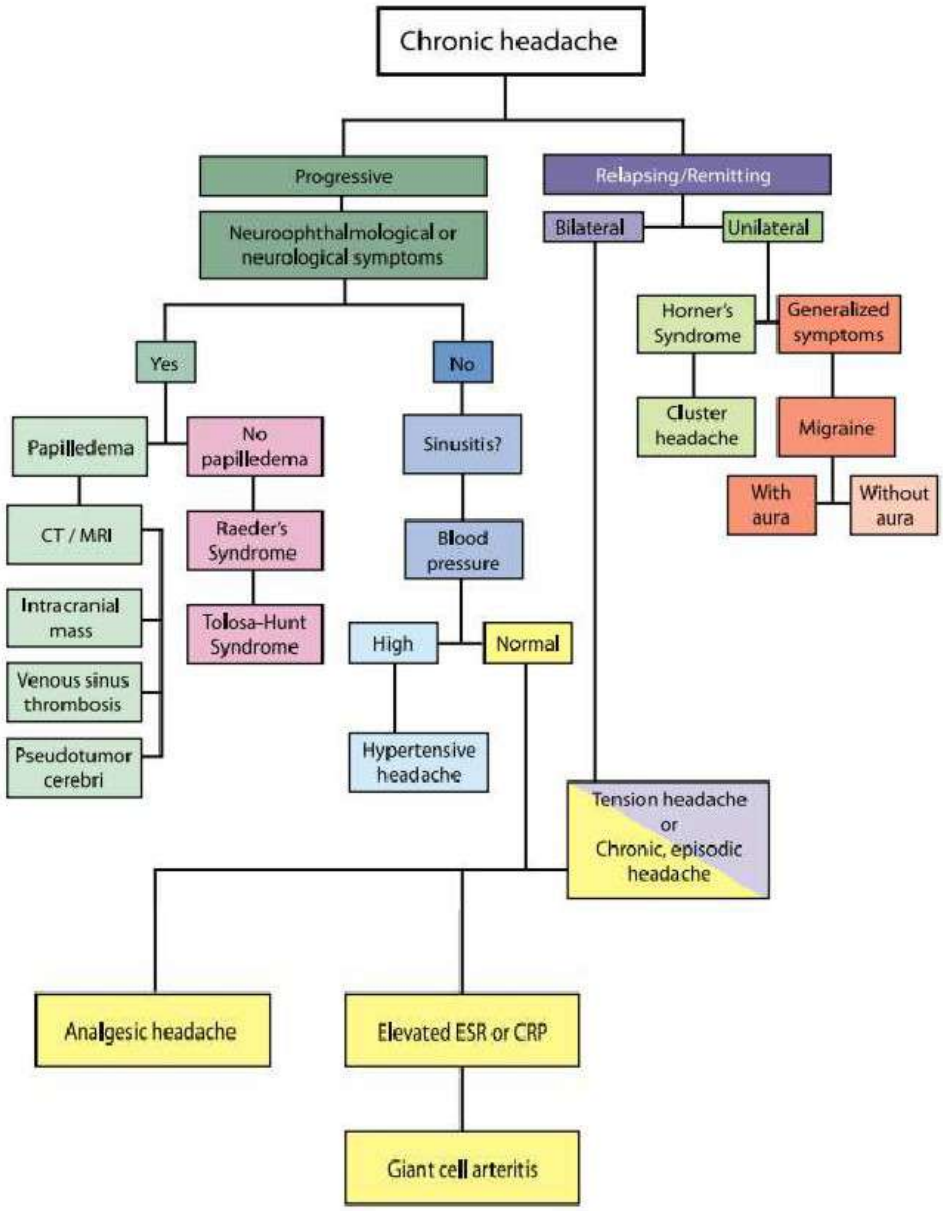


Figure 5 – Fundus Photograph showing Papilledema of both eyes.

Diagnosis

The presence of papilledema and visual impairments with chronic headache directed us towards the diagnosis of lateral sigmoid sinus thrombosis which was diagnosed by MR Venography.



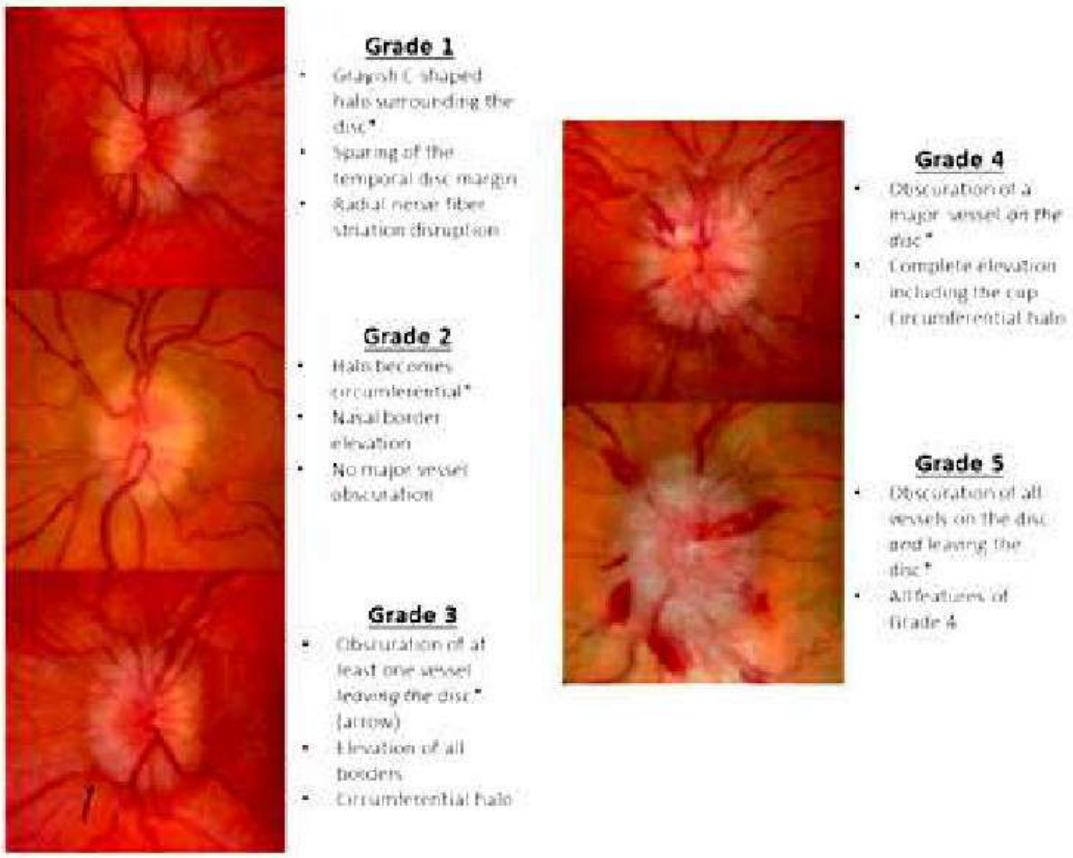
This flow chart by H. Wietholter, H. Wilhelm [3] helps us to simplify the various causes of headache.

Discussion

Proper assessment of a patient of headache when presenting in an ophthalmology clinic should include pupillary reaction followed by UCVA (Uncorrected visual acuity), Intraocular pressure, slit lamp examination , BCVA and dilated fundus examination.

The most important elements of the history of headache include the frequency, location, duration of episodes, intensity of pain, the course taken during episodes, instigating factors, and a family history of headache.

Papilledema is a swelling of optic nerve head secondary to elevated intracranial pressure(ICP). It is usually bilateral. Causes of elevated intracranial pressure include obstruction in the ventricular system by congenital or acquired lesions, space occupying intracranial lesions, subarachnoid haemorrhage , cerebral trauma , cerebral venous sinus thrombosis and idiopathic intracranial hypertension .



Modified Frisén scale for grading of optic disc edema and papilledema. [5]

Chronic cerebral venous sinus occlusion (CVSO), either complete or partial, can cause chronically elevated ICP, and can produce visual impairment as the only clinical symptom[4], which was seen in our patient. CVSO (partial or

complete) affects the dural venous sinuses that drain blood from the brain, and is usually caused by either venous thrombosis or stenosis. Cerebral venous sinus thrombosis (CVST) most commonly affects the transverse sinus (86% of cases), followed by the superior sagittal sinus (62%), straight sinus (18%), then least commonly, the cortical veins (17%) [6]. Risk factors for CVST include thrombophilia, chronic inflammatory diseases, use of hormonal contraception, infections such as meningitis, mastoiditis and sinusitis, and invasive procedures in the head and neck area [7, 8]. Symptoms of CVST include headache, visual impairment, symptoms of stroke (such as unilateral limb and facial weakness), and seizures. However, neurological symptoms are absent in a notable proportion of patients, and these patients may present later with impaired visual acuity due to chronic, advancing papilledema [9,10].

Imaging of patients of CVSO using unenhanced CT MRI may be totally normal, hence contrast enhanced MR venography may be required to image the cerebral veins for thrombus or stenosis and lumbar puncture is needed to measure the opening pressure and test for constituent changes in CSF.

Conclusion

It is very essential to evaluate each and every patient of headache so that we don't miss out the subtle signs of raised ICP which may be visible to an ophthalmologist and may be the only presenting sign of a very serious disease. Timely referral to the neuro-physician can save the vision and other morbidity of the patient .

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CASE REPORT

Persistent Pupillary Membrane with Bergmeister's Papilla: A Rare Case Report

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Abstract:

We report a 17 year old female with unilateral Persistent Pupillary Membrane (P.P.M) associated with Bergmeister's papilla (B.P) , which was an incidental finding and a contributing factor for her presenting symptom of diminished vision in that eye. Isolated cases of Persistent Pupillary Membrane are frequently encountered congenital anomaly of the iris while B.P is a rare congenital ocular anomaly of the optic disc.

Introduction

PPM is a common congenital anomaly that appears as fine iris strands along the pupil, which are remnants of anterior tunica vasculosa lentis that supplies nutrition to the lens in the first six months of fetal life.[1]. The membrane disappears completely by the eighth month of gestation. A failure of cellular activities that result in regression of pupillary membrane leads to PPM.[2]. PPM can be associated with amblyopia, anterior polar cataract, coloboma, strabismus [3].

Bergmeister's papilla (BP), also known as epipapillary veil, is named after an Austrian ophthalmologist, O. Bergmeister(1845–1918).[4] It is a rare congenital anomaly of the optic disc, characterized by the persistence of residues of the hyaloid artery. It starts regressing during fetal life itself, and disappears completely at birth. The residue of its anterior portion on the posterior lens capsule is called Mittendorf's dot, while the posterior residue on the optic disc is called Bergmeister's papilla. [5].

Case Report

A 17-year-old young female came to the Department of Ophthalmology, of a tertiary healthcare centre. She underwent a complete ophthalmological examination, for unilateral visual deterioration in left eye which she noticed since the last five years. There was no history of any systemic illness and the general physical and systemic examination was unremarkable. The uncorrected visual acuity (UCVA) in right eye was 6/6 and that of left eye was reduced to perception of light(P.L) and projection of rays(P.R) inaccurate in nasal quadrant. The pupil in both eyes were equal in size, round and regular in shape, reactive to light in right eye and sluggishly reactive to light in left eye.

For diagnosis slit-lamp examination, fundoscopy and OCT was done.

Anterior segment examination of right eye was within normal limits. Anterior segment examination of the left eye showed hypertropia and exotropia in primary gaze. Extra-ocular movements were normal in all directions of gaze. Slit lamp examination of left eye showed presence of persistent pupillary membrane and anterior capsular cataract [Figure1]. The membrane was attached to the iris anteriorly seen as strands of iris tissue running from the collarette and to the anterior lens capsule posteriorly, having an iridolenticular attachment.

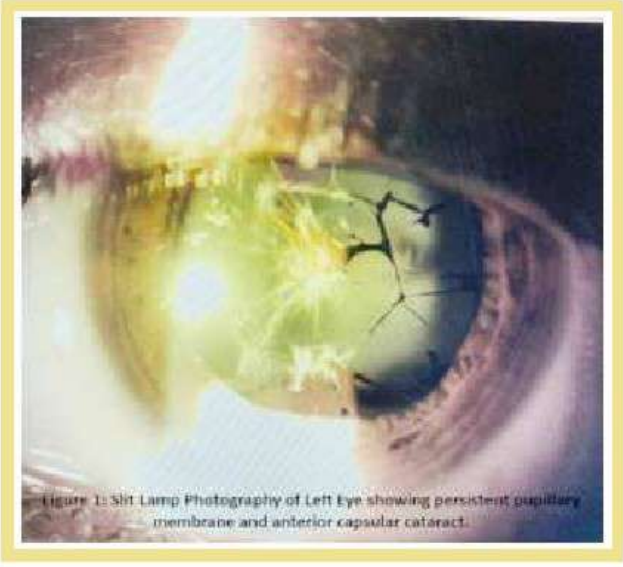
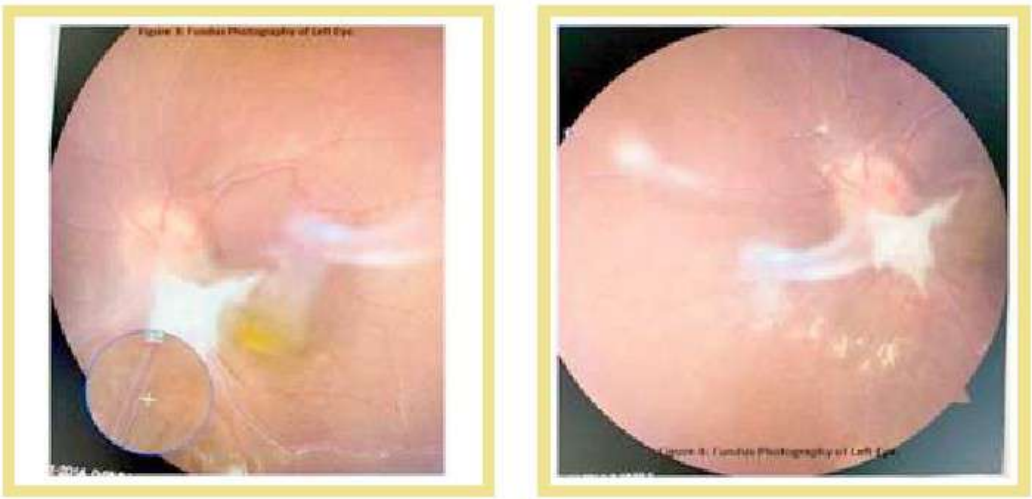


Figure 1: Slit Lamp Photography of Left Eye showing persistent pupillary membrane and anterior capsular cataract.

Fundoscopic examination of right eye was normal. [Figure 2].

Figure 2: Fundus Photography of Right eye.

Fundoscopic examination of the left eye revealed a vitreous thickening in the inferior sector of the optic disc, seen as a greyish white glial tissue on the optic nerve head (ONH). Sheathing of the infero-nasal & infero-temporal retinal venous vessels were seen along with few areas of hard exudates seen on the inferior quadrant. This glial tissue also led to dragging of optic disc and macula towards each other and this pulling force on the optic disc has led to striae like formation on the macula. [Figure 3 and 4]. OCT of the left eye confirmed the presence of hyper-reflective mass over as a cap on the ONH extending into the vitreous cavity. The intraocular pressure in both eyes were 14mm Hg measured by Goldmann applanation tonometer.



Figures 3 and 4: Fundus Photography of Left Eye

Fundoscopic examination of right eye was normal [Figure 2]. Fundoscopic examination of the left eye revealed a vitreous thickening in the inferior sector of the optic disc, seen as a greyish white glial tissue on the optic nerve head (ONH). Sheathing of the infero-nasal & infero-temporal retinal venous vessels were seen along with few areas of hard exudates seen on the inferior quadrant. This glial tissue also led to dragging of optic disc and macula towards each other and this pulling force on the optic disc has led to striae like formation on the macula [Figure 3 and 4]. OCT of the left eye confirmed the presence of hyper-reflective mass over as a cap on the ONH extending into the vitreous cavity. The intraocular pressure in both eyes were 14mm Hg measured by Goldmann applanation tonometer.

Discussion and Review of Literature

This case report of PPM with BP was diagnosed clinically and confirmed by OCT. In a study, the overall prevalence of BP was found to be 0.8%.[6]Another study using spectral domain OCT as a tool documented that BP was present in the majority of young, normal eyes, even though it could not be observed on fundoscopy.[7]

B.P is an incidental finding with minimal visual consequences, when present in isolation. The visual acuity may range from 6/6 to <6/60. The probable cause of detoriation of vision in left eye of our patient could be due to multiple reasons like PPM, anterior capsular cataract, deprivational amblyopia and maculopathy. P.R is inaccurate probably due to traction on the optic disc

A review study done by S Vaishali et al revealed that severe cases of BP can be associated with, pigmentary changes, amblyopia, posterior polar cataract, microphthalmia, persistence of primitive vitreous, vitreo-macular traction, vitreous haemorrhage and sometimes tractional retinal detachment due to contraction of the residual fibrovascular tissue [8]. The presence of these peri-papillary folds and surrounding retina could also be suggestive of chorio-retinal folds which shows alternating areas of hyper and hypo- hypo-fluorescence on FFA. Therefore, in all cases of suspected BP, careful monitoring of eventual vitreous thickening in the Peripupillary areas, by SD-OCT, FFA, OCT-A and doppler ultrasound is of considerable importance.

PPM can be unilateral or bilateral and can be variable in appearance, size, configuration, and density. It is usually seen as thin lacy strands of iris tissue running from the collarette, but a bilateral total persistent membrane is a rare occurrence and is associated with vision deprivation. In our case slit lamp examination revealed a similar pattern of lacy strands of iris tissue with anterior capsular cataract in her left eye. According to Duke-Elder's classification PPM is of 3 sub-types: Type 1 membranes that are attached solely to the iris, type 2 membranes are Iridolenticular adhesions. In a sub-variant of type 2, pigmented dendritic iris stromal melanocytes, singly and in clumps, are

situated aberrantly on the anterior lens capsule. These pigmented stars of the anterior lens capsule are often called "chicken tracks" and type 3 membranes, which are attached to the cornea, typically occur in Axenfeld-Rieger syndrome. Based on our slit lamp findings, our case had Type 2 PPM.

According to a study done by Shashidhar et al. most cases with PPMs are not significant enough to have visual complaints and so might go undetected. Pupillary apertures of less than 1.5 mm may obscure the light to reach the retina[9], which affects the visual acuity due to decreased retinal illumination and diffraction. In our case, probably due to the presence of a thick and large membrane along with cataract the patient had poor vision in her left eye.

In a study done by Brownstein S and his team in 1976, a 920g male infant was born with features of Potter's syndrome having B/L renal agenesis with multiple ocular anomalies. Ocular abnormalities included absence of keratocytes in the inner central corneal stroma, cataract with retention of cell nuclei in the nucleus of the lens, hypoplasia of the ganglion cell and nerve fiber layers of the retina, and absence of nerve bundles in the optic nerve. Other ocular findings including microphthalmos, fetal chamber angle, persistent pupillary membrane , retinal avascularity, and prominent Bergmeister's papilla that may have been related to the prematurity of the child[10].

As in our patient, ocular findings revealed PPM with BP with poor visual acuity and retinal findings of foveal drag on the optic disc and traction on the disc due to fibrous tissue, the patient was referred to a vitreo-retinal specialist and was asked for regular follow-up. Similar cases presenting remnant hyaloid artery should be monitored over time being a possible cause of future visual deterioration.

Conclusion

This case report of PPM with BP is a rare entity and has so far only been reported once in a baby with Potter's syndrome few decades back who had many other associated ocular and systemic anomalies. Search engines like pubmed and scopus were utilised using the keywords Persistent Pupillary Membrane with Bergmeister's papilla. Although several isolated cases of either PPM or BP have been reported in literature, this case report is a rare one from the perspective that failure of regression of both the embryological elements-tunica vasculosa lentis and hyaloid artery were present simultaneously.

Declaration of Patient Consent:

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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CASE REPORT

Congenital Ectopic Lentis

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Congenital Ectopic lentis is familial disorder in which displacement of crystalline lens from patellar fossa in the anterior chamber ,vitreous cavity or on retinal surface. This condition can be associated with many systemic and ocular disorder. Here we are describing a case of congenital ectopic lentis, which is associated with aniridia, AAK (aniridia associated keratopathy) ,glaucoma and foveal hypoplasia.

Case Report

An 18 year old girl presented with complaint of gradual diminution of vision in both eye since early childhood and photophobia . Left eye was more affected as compared to right eye. Her best corrected visual acuity was 3/60 in right eye and only perception of light in left eye. Intraocular pressure (IOP) with Goldman applanation tonometer was 34 mmhg and 40 mmhg in right and left eye, respectively. Complete evaluation of both eye was done ,inferiorly scleral show is in excess in both eye (Figure 1). Slit lamp examination done for evaluation of anterior segment ,which revealed, complete absence of iris tissue in both eye which was confirmed on gonioscopy ,corneal changes of aniridia associated keratopathy (AAK) ,peripheral superficial and deep corneal vascularisation and lens was subluxated superiorly which was hyper mature (Figure 2A, 2B).

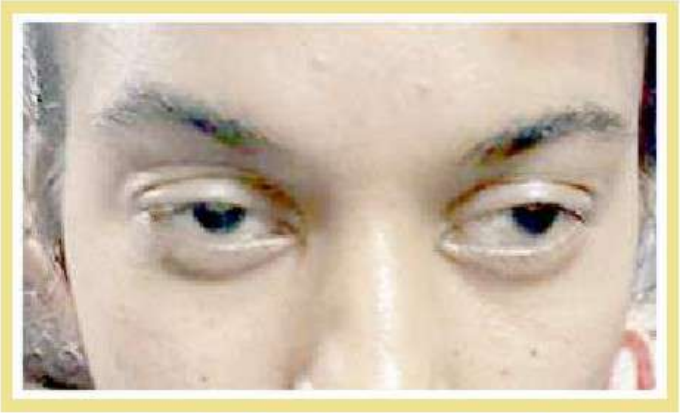

Figure 1: Diffuse light photography showing excess inferior scleral show in both eyes

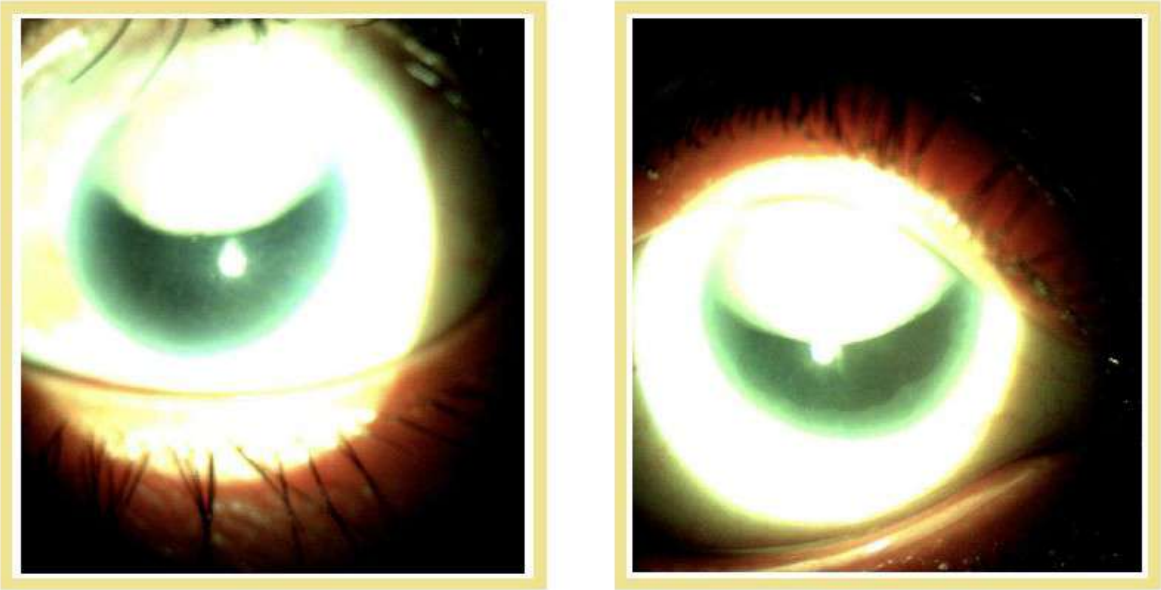

Figure 2 A and 2B : slit lamp photography showing aniridia and superior subluxation of lens with aniridia associated keratopathy.

Both eye had normal corneal diameter with superior lens subluxation with broken zonules inferiorly and raised intraocular pressure. Fundus evaluation revealed, cup disc ration 0.7:1 in right eye and glaucomatous optic atrophy in left eye (Figure 3) and foveal hypoplasia in both eyes. Detailed systemic examination done by physician and paediatrician which include skeletal examination, cardiovascular examination, ultrasound abdomen, homocysteine level in blood, vitamin B12 level ,liver function test ,renal function test etc, was unremarkable. Family history was not significant.

Figure 3: Glaucomatous optic atrophy with foveal hypoplasia in left eye

Left eye was having glaucomatous optic atrophy, visual prognosis in both eye was explained to patient and her family. This patient was managed medically and surgically as well, in right eye pars plana vitrectomy and lensectomy was performed and post operative rehabilitation with use of spectacles was advised.

Anti glaucoma medication started and patient was advised for regular IOP monitoring and follow up.

Diagnosis

Bilateral congenital ectopic lens with aniridia and foveal hypoplasia associated with glaucoma.

Discussion

Congenital ectopic lentis refer to the familial displacement of crystalline lens from patellar fossa.(1) When lens is partially displaced it is called as subluxated. It may be associated with systemic disorder. So it is important to evaluate the patient completely and find out the associated etiology for appropriate management. Patient should be asked about any other cardiovascular and skeletal abnormalities. Detailed family history is also required. Inheritance can be autosomal dominant and autosomal recessive. Patients usually present with defective vision, photophobia , refractive errors, loss of accommodation and diplopia .

Ectopic lentis can be associated with many systemic disorders. These are, Marfan syndrome, which is most common cause of congenital ectopic lentis (2). Lens dislocation in Marfan syndrome is usually bilateral and occurs most often in supra-temporal direction but other directions are also possible.(3) After Marfan syndrome, Homocystinuria is the second most common cause of congenital ectopic lentis. It is an autosomal recessive metabolic disorder. Cystathionine b synthase is deficient in homocystinuria. Here displacements of lens is bilateral like in Marfan syndrome .(4)
Other systemic diseases associated with ectopic lentis are Ehlers - Danlos syndrome, Sturge - Weber syndrome, Pierre Robin syndrome, Sprengel deformity, WAGR syndrome. (5)

Ectopic lens can be associated with many other ocular conditions like aniridia, AAK, congenital glaucoma, pseudo exfoliation syndrome, retinitis pigments, intraocular tumour, megalocornea , hyper mature cataract ,high myopia , buphthalmos, ectopic lentis et pupillae .(6) Congenital anridia is either partial or complete hypoplasia of iris. In most of the cases , there is mutation in PAX6 gene, due to deletion on chromosome 11p13. It can be associated with WAGR syndrome (Wilm's tumour, Aniridia, Genitourinary abnormalities, Retardation). Many cases presented with aniridia associated keratopathy (AAK). (7) Ectopic lentis can be associated with glaucoma, so patient should be advised for regular IOP monitoring and treatment. It may be due to anatomical changes in drainage angle or trabecular mesh work or schlemm's canal.

Once diagnosis is made , patient should be treated accordingly. For that , there should be co-management with paediatrician. Genetic counselling should be done if indicated. Displaced crystalline lens are managed surgically, pars plana

lenesectomy/ vitrectomy with or without intraocular lens implantation. Lensectomy is indicated in case of mature and hyper mature lens induced glaucoma, lens induced uveitis, lens in anterior chamber and touching corneal endothelium. In case of an aniridia scleral fixated IOL can be implanted. If patient is left aphakic contact lens or spectacles can be prescribed. (8) For raised IOP anti glaucoma medication to be prescribed and regular follow-up to be advised to the patient. All the patient of ectopic lentis require detailed counselling regarding pathology, long-term visual outcome and prognosis of the condition. The patient and his family should also understand the complication associated with ectopic lentis.(9)

Conclusion

Congenital ectopic lentis is familial disorder. Patients present with defective vision, loss of accommodation, photophobia, diplopia, pain etc. Detailed ocular and systemic examination should be done. Counselling of the patient should be done regarding complication and prognosis . Management depends on the visual acuity, extent of the subluxation, other associated ocular and systemic pathology.

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CASE REPORT

Corneal Neurotization: A Novel Approach for Treating Neurotropic Keratitis

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Introduction

Insensate cornea is a difficult and challenging problem to manage throughout the world. Sensations are required to initiate blink reflex, maintain integrity of corneal epithelium and limbal stem cell function[1]. Corneal sensations are provided by ophthalmic division of trigeminal nerve. Reduced corneal sensation results in reduced reflex tearing, increase risk of corneal surface injuries and poor healing[2]. All these factors lead to epithelial defects that ulcerate and perforate if not treated appropriately. Lack of corneal sensation leads to a clinical condition known as neurotrophic keratopathy. Patients who had facial palsy along with insensate cornea are at even greater risk for corneal disease. Facial nerve palsy results in lid laxity and the inability to completely close the eyelids leading to chronic exposure, dry eye, loss of corneal clarity and keratitis. Treatment options are limited and most of them target to protect the cornea, instead of addressing the corneal denervation leading to recalcitrant progression and vision loss. Reinnervation of the cornea can be achieved by transfer of a healthy donor nerve into the cornea.[4,5,6] Corneal reinnervation/neurotization restores the corneal sensation and corneal healing thus preventing vision loss. Here we present a case of insensate cornea with lagopthalmos, managed successfully with corneal neurotization and ipsilateral temporalis transfer for eye closure.

Case Report

A 22-year-old male presented with lagopthalmos associated with irritation and burning in right eye that was unresponsive to medical management. On presentation, his best corrected visual acuity in right eye was 6/12. He had absent corneal sensation and blink in all four quadrants tested with a wisp of

cotton. On Slit lamp examination, he had macular type corneal opacity in inferior quadrant (Figure 1A). Corneal Rose Bengal staining was suggestive of epithelial breakdown with epithelial deficits (Figure 1B).

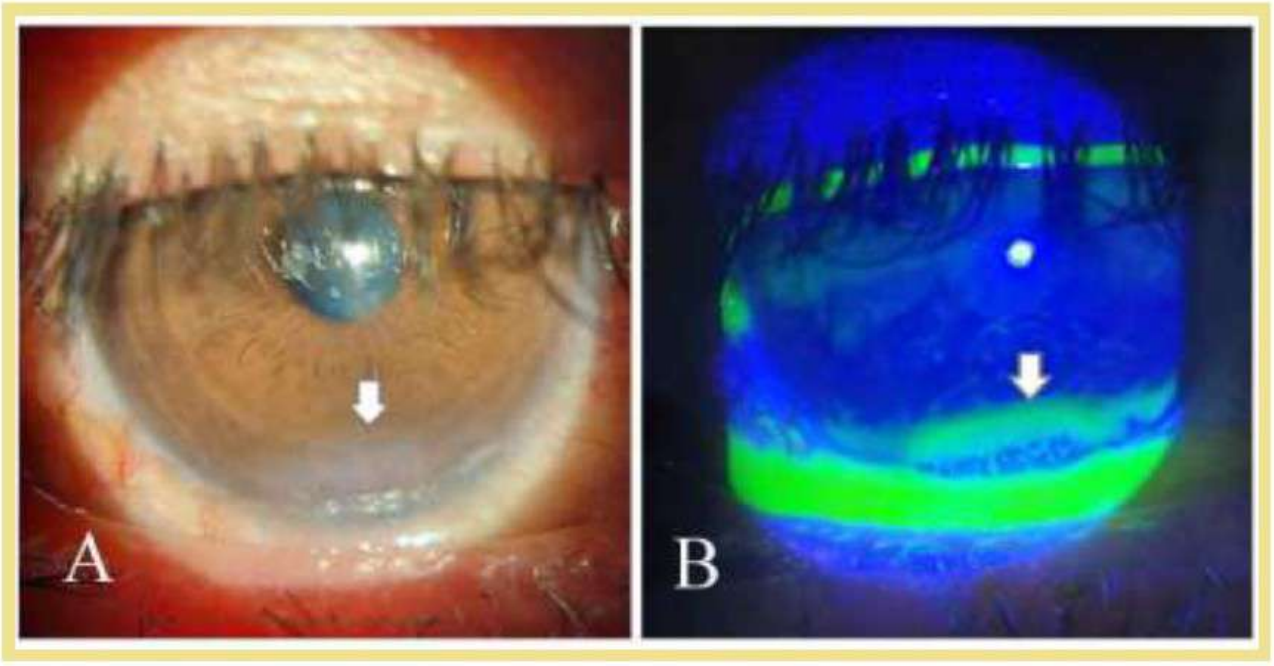

Figure 1 (A) Corneal opacity in inferior quadrant on slit lamp examination. (B) Epithelial breakdown with epithelial deficits on Corneal Rose Bengal staining. External exam revealed right sided lagopthalmos along with other features of facial nerve palsy on right side of face

(Figure 2). Sensations were intact over entire face except right cornea Figure 2 (A,B) – Incomplete Right Eye Closure.

After evaluation, he was planned for corneal neurotization by ipsilateral supraorbital nerve transfer using sural nerve graft and temporalis transfer for right eye closure.

Surgical Technique: Corneal Neurotization

Right sub brow skin incision was made and dissection was carried down to the periosteum of orbital rim. Supraorbital nerve is identified at supraorbital notch and its deep branch was dissected. 8cm sural graft was harvested from right leg (Figure 3).

A tunnel was made from the sub brow incision to the superior medial fornix and the sural nerve graft was passed through the tunnel into upper fornix (Figure 4A). Perilimbal conjunctival and Tenon's layer incisions were made at 12, 3, and 9 o' clock, 5 mm away from the limbus to accommodate the nerve fascicles.

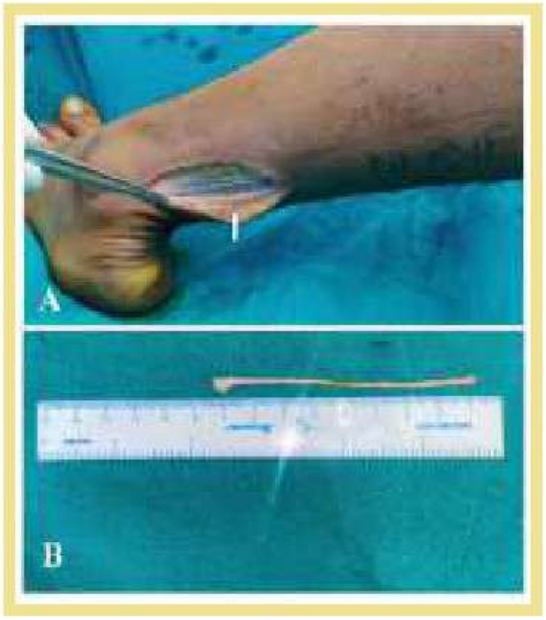

Figure 3 (A,B) Harvested Sural Nerve Graft. Blunt dissection was done to create a sub tenon space from 9'o clock to 3'o clock and from 12'o clock position to upper fornix. Sural nerve graft was brought out at 12'o clock perilimbal incision. The sural nerve is separated into 2 fascicles (Figure 4B).

Both fascicles were passed around the limbus in the Sub Tenon's space and sutured to the sclera at the limbus with 10-0 nylon sutures at 9'o and 3'o

clock position (Figure 4C). The conjunctiva was closed to cover the nerve-corneal union. The deep branch of the supraorbital nerve was coaptated with sural nerve graft with 9-0 nylon in end-to-end fashion (Figure 4D).

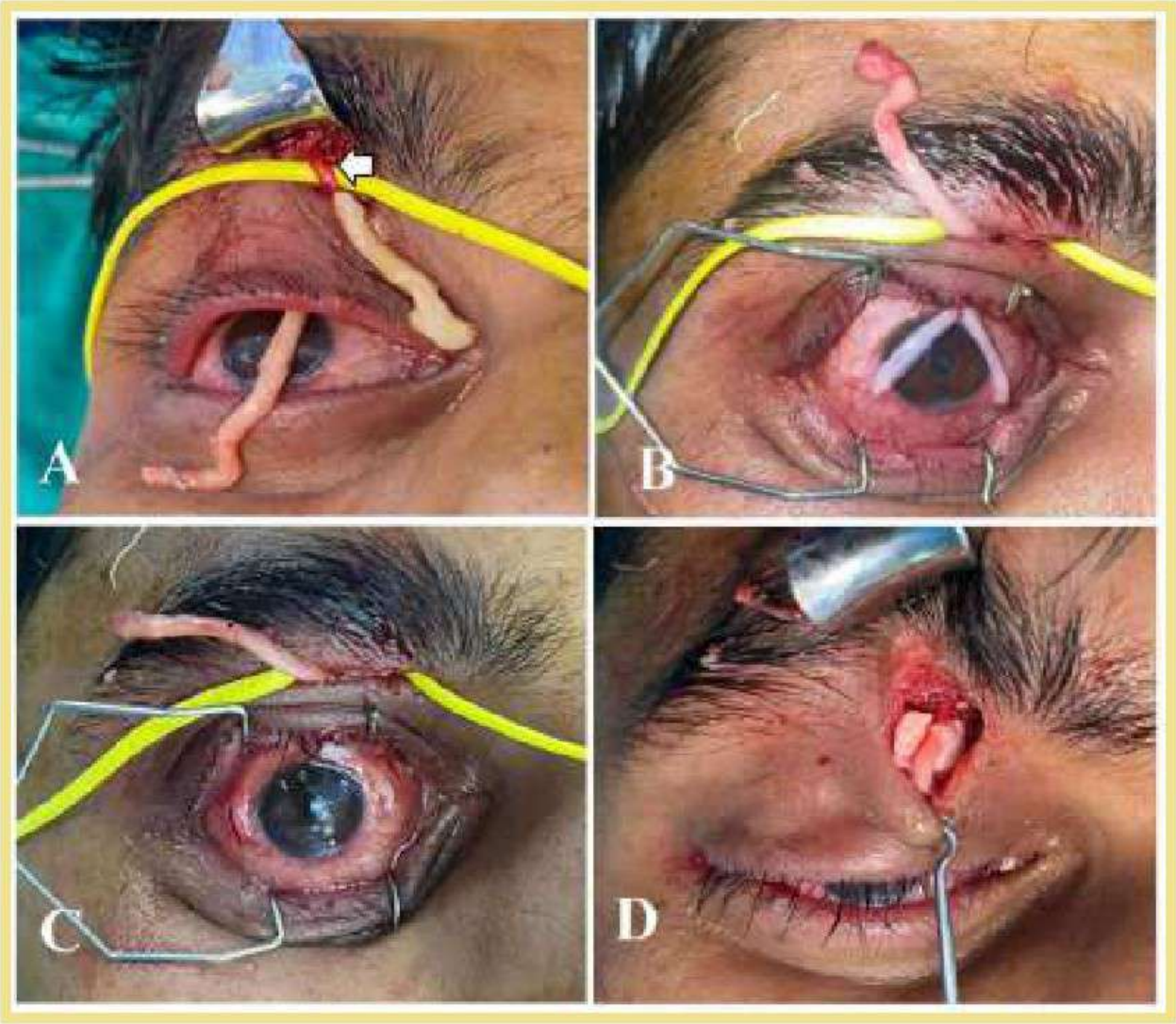

Figure 4 (A) Arrow indicate deep branch of supraorbital nerve. (B) Two separated fascicles of sural nerve at 12'O clock position (C) Sural nerve fascicles suture to sclera at 3'O and 9'O clock position. (D) Sural nerve graft coaptated with supraorbital nerve in end-end fashion.

Temporalis Transfer

A curvilinear incision was made in temporal hairline as shown in figure 5A. Deep temporalis fascia. A 15mm wide strip of deep temporal fascia along with 20 mm tongue of temporalis muscle is harvested keeping fascia attached at the upper end. Temporalis fascia is then divided into 2 strips.

Another curvilinear incision was made at lateral margin of orbit temporalis muscle along with two fascial slings brought out from the lateral orbital incision via subcutaneous tunnel 9Figure 5B). Third incision was made at medial canthus and medial canthal tendon was dissected. The upper fascial strip was passed through upper eye lid in a plane above the tarsal plate and approx. 5mm above the lid margin and brought out at medial canthal incision. Similarly lower strip was passed from the lower lid. The two strips were sutured with each other and to the medial canthal tendon, creating a overlap of lower lid by upper lip by 2mm (Figure 5C).

Figure 5 (A) Temporal hairline incision to harvest temporalis muscle with deep temporal fascia. (B) Two temporal fascial slings brought out at lateral orbital margin. (C) Both slips passed through eyelid and sutured to medial canthal tendon.

There were no intra op and post op complications. At 4 months follow up, there was partial resolution of corneal stromal opacification and greatly improved corneal sensation by testing with a wisp of cotton in all 4 quadrants (Figure 6). Right eye closure was near complete (Figure 7).

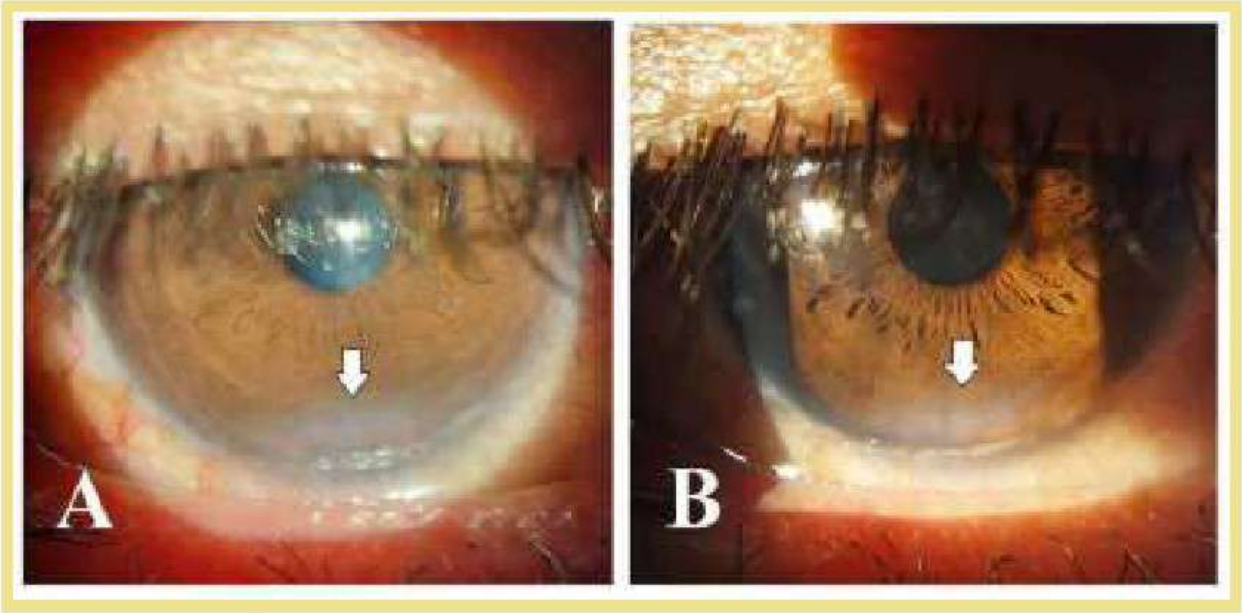

Figure 6 (A) – Preop Corneal opacity in inferior quadrant on slit lamp examination. (B) Partial resolution of opacity at 4 months.

Figure 7 (A) Incomplete right eye closure prior to surgery. (B) Near complete eye closure at 4 months.

Discussion

The insensate cornea is a devastating condition that affect the quality of life and leads to permanent blindness. It remains a challenge to treat insensate cornea. Majority of treatments provide only temporary relief and do not address the underlying cause of denervation.

The first report of corneal neurotization was described by Terzis et al.[5] in 2009, where direct neurotization of the cornea was done using the contralateral supraorbital and supratrochlear nerve. The average time to restore corneal sensations was 2.8 years.[5]

Elbaz et al. reported the use of sural nerve segment for end-to-side coaptation with contralateral supratrochlear nerve.[4] Marked improvement in corneal sensation was noted at 6 months.[4]

In contrast to previous reports, our patient had congenital palsy of long ciliary nerves and facial nerve leading to corneal anesthesia and lagopthalmos respectively. Sensations in ipsilateral supraorbital and supratrochlear nerve territory were normal, so we utilized his ipsilateral supraorbital nerve to neurotize the cornea using sural nerve interpositional graft.

In addition, we did temporalis transfer for right eye closure. This procedure corrects the lid gap and also helps in restoration of corneal sensation by reversing the exposure keratitis.

Conclusions

Corneal neurotization is a novel technique to restores the corneal sensation. This method showed excellent results in corneal reinnervation, healing of corneal epithelium, vision improvement and quality of life improvement.

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CASE REPORT

Nocardia Keratitis: A Case Report

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Introduction

Nocardia species can be found as saprophytes in soil, dust and water worldwide, Keratitis caused by it is not common in clinical scenario and therefore poses a challenging diagnosis. Several case series have demonstrated that patients with Nocardia keratitis typically have good visual outcomes with timely appropriate diagnosis and therapy.[1,2]

Case Report

An Eighteen year male, presented to our outpatient clinic in June2022 with complaints of pain, redness, photophobia and blurring of vision in his left eye. He gave history of fall of some sand particles in his left eye after which he started to have these complaints. He had no history of contact lens use in past or any previous ocular surgery. With these complaints he visited to some local practitioner and got prescribed topical drops constituting ciprofloxacin and dexamethasone . At this time he was having mild symptoms ,but after use of eye drops , his symptoms got increased. So, he visited to our outpatient department for the same. During examination his Visual acuity of left eye was found to be 6/ 36 improving to 6/9 with pinhole. On Slit lamp biomicroscopy examination ring shaped corneal infiltrate involving anterior stromal layers of size 5*5 mm at inferotemporal location in periphery ,sparing visual axis , with central epithelial defect was found . The pattern was wreath like ,so he was suspected to have nocardia infection. Corneal scrapings were taken from the edges of the infiltrate and sent to laboratory for gram stain, KOH stain , Acid fast stain and culture. Anterior Chamber was quiet, no signs of inflammation was seen. Digital intraocular pressure was also normal. Fundus examination was found to be within normal limits .Visual Acuity in right eye was 6/6 and ocular examination was within normal limits. While awaiting reports, Amikacin1% eye drops 1 hourly, ciprofloxacin 0.3% eye drops 6times a day and atropine eye ointment 1% three times a day was initiated on the basis of

clinical signs. With above maintained topical therapy patient was reviewed every week thereafter. At every visit ocular examination including BCVA and slit lamp examination with fluorescein staining was done. He showed improvement at every visit ,and patient was symptomatically better. On 7th day , the epithelial defect started to approximate and on day 15 it was completely healed by a fusion line . After approximately two months with the same treatment , eye quietened with nebulo-macular scarring peripherally, with no vascularization. At the final visit, best corrected visual acuity was 6/6 and the young patient was happy.

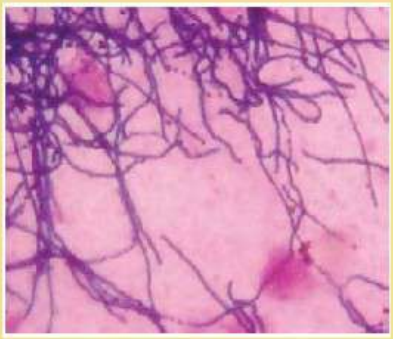

Fig. 1 Gram staining showed beaded gram-positive, branched filaments

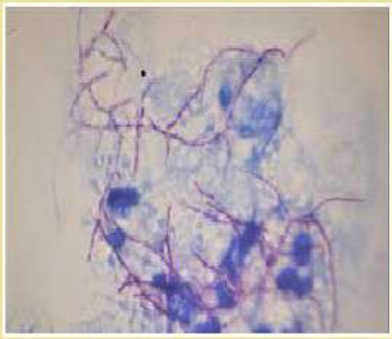

Fig 2 .Corneal scrapings stained with 1% acid fast (**Ziehl-Neelsen**)stain showing acid fast filaments

Fig 3 .Blood agar showing heavy growth of small white dry colonies

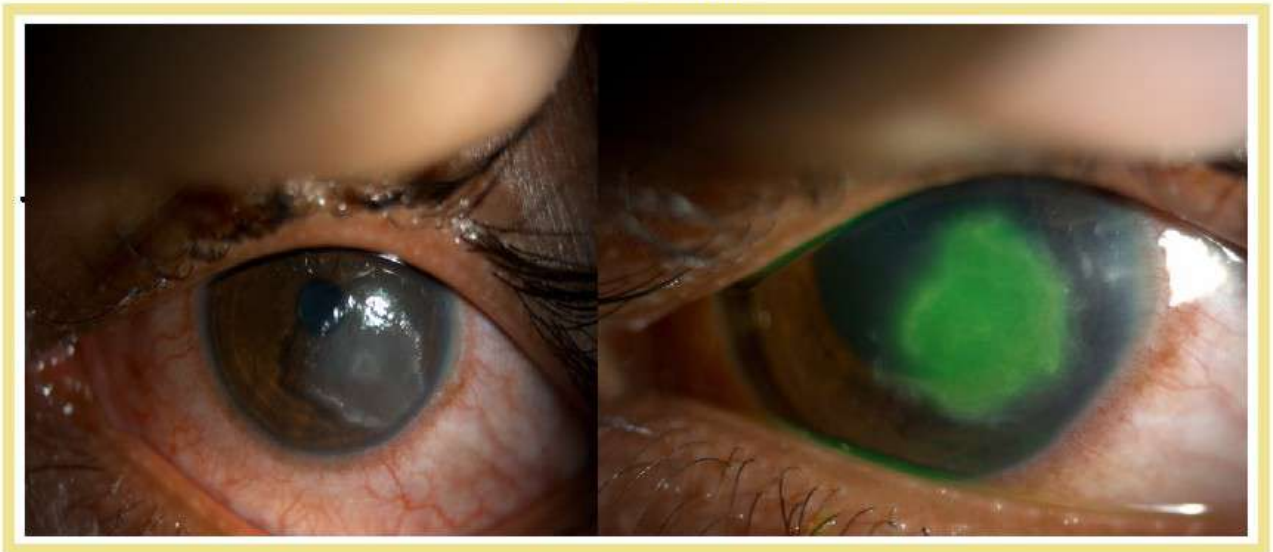

Fig 4 Slit lamp image of left eye showing ring infiltrate with wreath pattern and fluorescein stain delineating epithelial defect (Day 1)

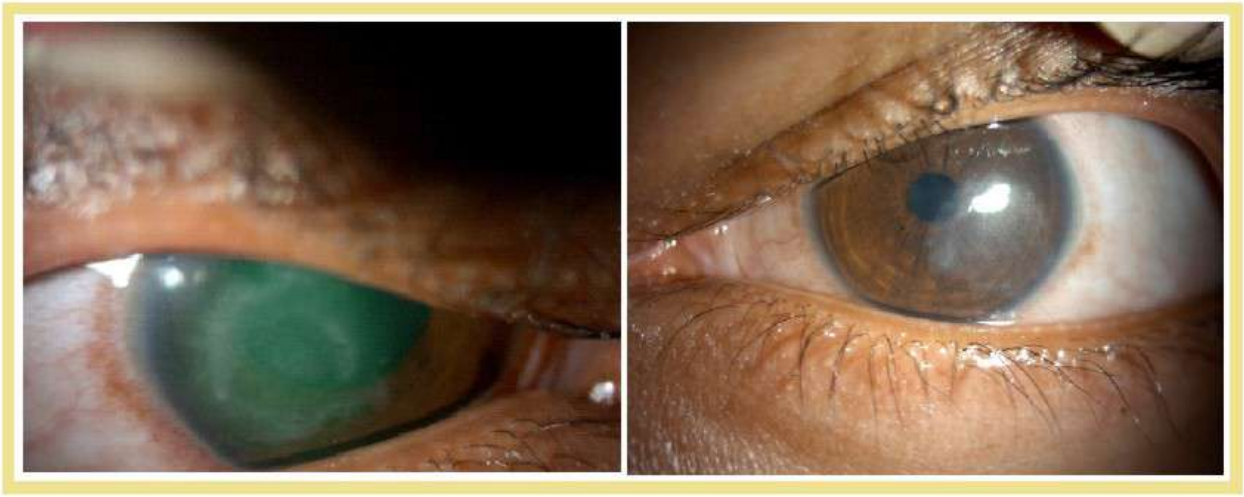

Fig 5 Slit lamp image of left eye after initiation of treatment- Reduced infiltrate and scarring started (1 month)

Fig 6 Slit lamp image of left eye after healing at the last visit displaying minimal nebular scar .

Discussion

Nocardia are aerobic, branching, beaded filamentous bacilli with Gram stain variability and are often acid-fast positive. Although relatively uncommon, Nocardia are an important cause of keratitis.[1-5]. A study from Hyderabad in South India revealed that among 689 bacterial isolates from cases of keratitis examined between January 1991 and December 1998, Nocardia species constituted 1.7%. [6.] Nocardia species keratitis is known to present a challenging diagnosis, especially in those regions where clinicians are not familiar with the typical appearance on examination: patchy white and pin head infiltrates in the anterior stroma, often arranging in a wreath pattern[2,9].The usual predisposing factors are trauma(vegetative matter, dirt), corticosteroid use and contact lens wear. However when promptly identified and treated with topical antibiotic amikacin, visual outcomes are good.[1,8]

This particular case of Nocardia keratitis displayed similar anterior stromal infiltration with pinhead edges in the course of the disease following soil entry

into eye and responded well to topical amikacin and ciprofloxacin. The use of topical steroids in bacterial corneal ulcers is controversial.

Case reports have suggested that topical corticosteroids may result in recurrence of the infection and prolonged healing time[7].In our case, we found that use of corticosteroids would have aggravated the course of infectious disease.

In light of review of literature[1-11] and our own experience with this specific case, we believe that Nocardia species Keratitis should be kept in mind in the presumptive diagnosis of a ring infiltrate. Microbiological diagnosis obviously aids in clinching the disease. Corticosteroids should not be applied in the treatment of Nocardia species Keratitis. The course of disease is however prolonged.

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CASE REPORT

An Interesting Ocular Finding, Leading to the Systemic Diagnosis

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The 32 years old female patient came to eye OPD for regular eye check-up, and found to have Vortex keratopathy and the findings further helped in diagnosing the rare enzymatic deficiency disorder- Fabry Disease. Cornea verticillata consists of bilateral whorl-like opacities in the superficial corneal layers, mostly in the inferior corneal area. In Fabry Disease, it is usually present in young adults, with history of long term fever and renal involvement. They show some resemblance to the corneal opacities found after chronic administration of drugs such as chloroquine, Tamoxifen, Amiodarone, Diclofenac, Antacids, Chlorpromazine and Retinoids. Cornea verticillata have been described in almost all patients with Fabry disease, both in hemizygous males and heterozygous females and they are usually considered to be the most reliable ophthalmological marker of Fabry disease.

Introduction

Ophthalmological manifestations are usually found in Fabry disease, affecting ocular structures. They are important because some manifestations act as markers of the disease, with diagnostic and prognostic implications even though they don't usually cause any visual impairment or other ocular symptoms. Eye is an external organ, easily investigated with minimally invasive technologies, ocular abnormalities may also provide a useful means of monitoring the natural history of the disease and patients' response to enzyme replacement therapy (ERT).

Case Report

A 34 years old female came with complain of blurring of vision in both eyes since 1 month, associated with headache off and on. She also gave a history of fever since 10 days. Not associated with pain, redness or watering. She has no history of glasses used or ocular surgery. There was no other significant systemic history. Clinical findings are shown in Figures 1- 3.

	RE	LE
VA (unaided)	6/12p, N10	6/12p, N12
BCVA	1.25/-1.75X180(6/6)	-1.00/-0.25X180(6/6)
IOP	14	16
PUPIL	RR SLUGGISHLY REACTIVE	RR SLUGGISHLY REACTIVE

Whorl like whitish epithelial streaks present on cornea inferior to pupillary axis, extending from 9 to 11'o clock in basal epithelial layer. No flare, cells or pigments. Whorl like whitish epithelial streaks present on cornea inferior to pupillary axis, extending from 1 to 4 'o clock in basal epithelial layer. No flare, cells or pigments (Figure 1).

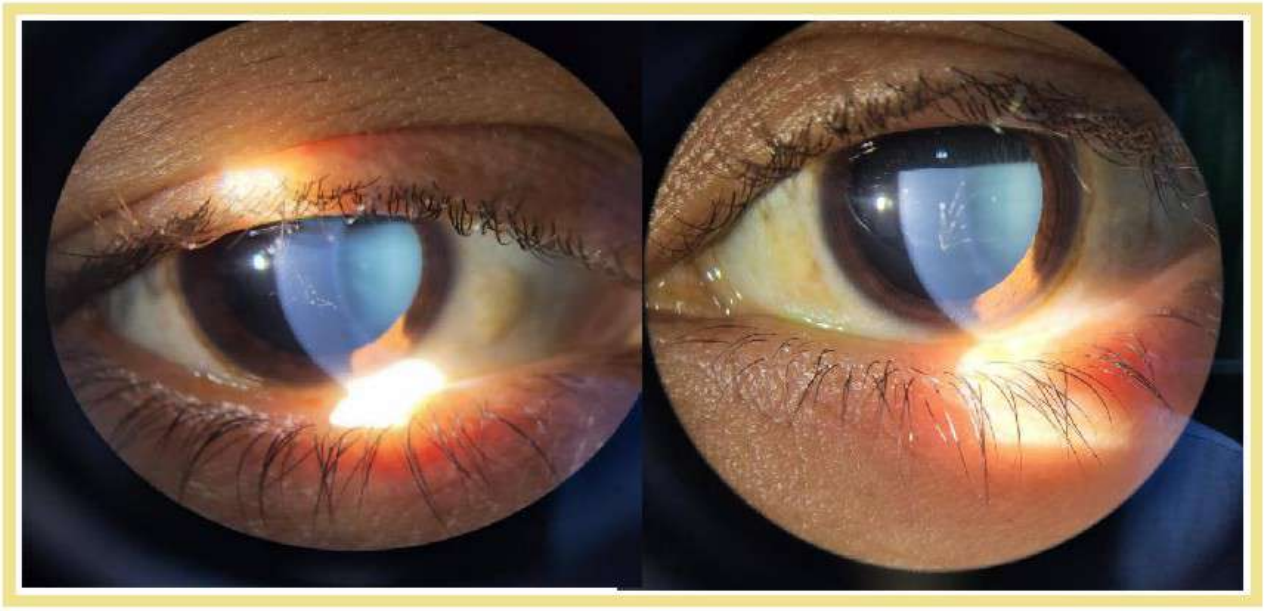

Figure 1 RE **Figure 2 LE**

Media : hazy Disc: Blurred margins, congested, edematous Posterior pole: Diffuse RP atrophy, Few dot haemorrhages temporally FR: present Vessels: tortuosity of vessels in corkscrew pattern with looping of vessels (Figure 3).

Figure 3.

After examination, a differential diagnosis of Fabry disease, hypertension, Multiple myeloma, or drug use like antacids/retinoids was made and the patient was sent to medicine department for further workup, where her RFTs were abnormal (Creatinine: 6 Urea: 40) And the final diagnosis of Fabry Disease was made.

Discussion

Eye abnormalities in Fabry disease result from the deficient activity of the lysosomal hydrolase, α-galactosidase A. This deficiency leads to a progressive deposition of glycosphingolipids in some ocular structures [2-4]. The most specific ocular manifestations of Fabry disease are:

- conjunctival vascular abnormalities
- corneal opacities (cornea verticillata)
- lens opacities
- retinal vasculature

Conjunctival Vessel Abnormalities

The most characteristic ophthalmological manifestations are increased vessel tortuosity, venous vascular aneurysmal dilation and 'sludging' of the blood in the small blood vessels. These changes can be seen in any conjunctival area, but they are most commonly located in the inferior bulbar conjunctiva.

Corneal opacities

Cornea verticillata consist of bilateral whorl-like opacities located in the superficial corneal layers, most commonly in the inferior corneal area. These opacities are typically cream coloured, ranging from whitish to golden-brown. They are termed cornea verticillata because the deposits are distributed in a vortex pattern. In the early stages, the opacities may form fine horizontal lines, but they later develop into curving lines, radiating from a point below the centre of the cornea and forming small whorls, before becoming almost straight at the periphery.

Corneal pathology has been investigated both in hemizygous and heterozygous patients with Fabry disease [5-9]. The most relevant finding is the presence of intra-epithelial deposits consisting of dense laminated cytoplasmic inclusions, both membrane-bound and lying freely in the cytoplasm. In a histopathological study of the cornea of a woman with Fabry disease, Weingeist and Blodi described subepithelial ridges composed of re-duplicated basement membrane and amorphous electron-dense material between the basement membrane and Bowman's layer. They suggested that the diffuse accumulation of sphingolipids in the corneal epithelium might be responsible for the diffuse corneal haze,

while the whorl-like pattern might be determined by a series of subepithelial ridges [10]. However, these ridges have not been consistently reported in either heterozygous women or hemizygous men [11].
Differential diagnosis of Vortex Keratopathy (Cornea Verticillata):
Drugs: Tamoxifen, Amiodarone, Diclofenac, Antacids, Chlorpromazine, Retinoids. Deficiency of Galactosidase A- FABRY DISEASE, Multiple Myeloma, Neurotrophic Keratitis, Lisch Corneal Dystrophy

Lens opacities

Two specific types of lens opacities have been reported in patients with Fabry disease. Anterior capsular and subcapsular opacities are generally bilateral and wedge-shaped. Posterior subcapsular cataracts are rare but very specific for the disease (and hence are called Fabry cataracts).

Retinal and choroidal vessel abnormalities

Retinal and choroidal vessel abnormalities are mainly represented by an increased tortuosity of the retinal vessels (sometimes with a 'corkscrew' appearance) associated with segmental venous dilation, arteriolar narrowing and arteriovenous nicking (localized constriction).
Retinal vessel tortuosity can be easily detected by simple ophthalmoscopy of the posterior segment of the eye. However, it is better appreciated using fluorescein angiography.

Conclusions

A review of published literature and analysis of the FOS database suggest the following conclusions regarding ocular manifestations of Fabry disease.

- The presence of cornea verticillata is highly sensitive and specific for Fabry disease in both male and female patients. Cornea verticillata can therefore be considered a useful ophthalmological marker for Fabry disease, although it should be borne in mind that corneal opacities may also occur in patients taking certain drugs.
- In some cases, cornea verticillata can be an isolated occurrence, without the presence of other eye abnormalities.
- Tortuous vessels are common in Fabry disease, but are relatively non-specific for the disease.
- Posterior subcapsular cataracts with a spoke-like appearance are rare but, when present, may suggest a diagnosis of Fabry disease.
- The presence of vessel tortuosity appears to be associated with a more rapid progression of Fabry disease.
- The study of eye abnormalities can aid diagnosis but does not significantly improve the accuracy of monitoring progression of the disease and its response to treatment. Technological developments are required before eye signs can be quantified and used successfully to monitor patients affected by Fabry disease.

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OPHTHALMOLOGY PRACTICE PEARLS

Alarming Rise in Consumer Cases/Medical Malpractice Claims: How to Safeguard Medical Professionals?

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India is witnessing an alarming rise in cases of medical negligence filed in consumer cases against hospitals and medical professionals.[1-3] A heavy compensation was ordered in some of these cases. In a judgment given in October 2013 on medical negligence, the Supreme Court awarded compensation amounting to Rs. 11 Crore to a victim, which was to be paid by the doctors and the private hospital deemed responsible for the wrongful death of a patient. This landmark decision was by far the largest compensation award in the history of Indian medical negligence litigation. The National Consumer Disputes Redressal Commission (NCDRC), New Delhi, on August 26, 2022, awarded an exemplary compensation of Rs. 1 Crore to the parents of a 6-year-old child. The child was admitted for a squint eye correction surgery and died while undergoing squint surgery at an eye hospital in Chennai. In July 2015, the Supreme Court ordered a compensation of Rs 1.7 Crore to a girl who lost vision soon after birth due to medical negligence by doctors of a government hospital in Tamil Nadu.

The medical profession is considered to be one of the noblest professions in the world. While Indian medical infrastructure is being noticed and praised on the global map, on the contrary, the doctor-patient relationship is deteriorating, our internal medical setup is facing extensive problems with medical litigation fast becoming one of the most serious of all issues. Doctors are no longer regarded as infallible and beyond questioning. We live in a culture in which displeased patients have increasingly turned to litigation as a means of obtaining redress from perceived deficiencies in the quality of care received from their physicians. Consumer cases are increasing not only for the medical branches dealing with life and death (emergency medical branches) or dealing with two lives (such as obstetrics and gynaecology) but doctors working OPD/day care set up and diagnostic branches like ophthalmology, dermatology, dental surgery, radiology are also increasingly facing consumer cases/litigations.

The best way to handle consumer cases/medico-legal issues is by preventing them by 5 Cs: Checklist, Cost, Consent, Counselling and Complications Management.

Despite the best possible care, best intentions and best medical practices, some complications are bound to occur, and at these times, effective communication with the patients and attendants is the key to avoid these complications from becoming lawsuits. While communicating with these patients, we need to be honest and sympathetic, but not overly defensive. It helps to clearly admit that a problem has occurred rather than being evasive. However, the responsibility doesn't end with good communication, we need to do our best to ensure that the complications are handled well, or are referred to the right place. Support the patient at this time, by explaining the attendants, helping to take the patients elsewhere etc. The right attitude and communication at this crucial time can make a huge difference to the reaction of the patients and avoid litigation despite unfavourable outcome.

Timely referral to an expert is important for managing difficult situation or any specific disease. Never criticize or disapprove of treatment or surgery done by your professional colleague in front of patients or relatives as it can provoke them to file malpractice lawsuits. The increased cost of service delivery has ultimately led the consumers to have higher expectations from the medical providers. Combined with the increased awareness and the availability of means to vocalize their grievances, patients can highlight cases of negligence even for the smallest deficiency in the service.

"Checklists", "Cost", "Consent", "Counselling" and "Complications Management":

The WHO Surgical Safety Checklist is a prominent example of a surgical checklist intended to ensure safe surgery and minimize complications. Train your entire team to follow the checklists and protocols. Examine each and every patient very carefully. Ask for the previous medical records and never forget to take complete history of systemic illness, drug allergy, previous surgery or trauma, etc.

It is important to counsel and explain each and every patient about the surgery, cost, outcome after surgery, need for follow-up, and possible complications. The preoperative stage entails taking valid informed consent (video consent in all high-risk cases) of the patient for executing the proposed treatment, taking and recording the history of the patient, carrying out a proper examination, diagnosis, and investigations, pre-anaesthetic check-up, detailed counselling, complete systemic investigations (and clearance for surgery) and then proceeding with treatment.

Always take the help of an anaesthesiologist for monitoring vital parameters after taking patients in the operation theatre. The surgeon and entire team should be vigilant to minimize the complications encountered during the surgery in the operation theatre, accidents, drug reactions, and mishaps experienced while operating, (for example surgery in the wrong eye/wrong side, implanting wrong IOL/implant), death during operation, and other similar incidents. Always document all operative notes, follow-up advice, detailed instructions about using the medications/eye drops, and communication about the postoperative complications, etc. Several doctors use abbreviations and short forms instead of detailed notes, and this needs to be avoided, especially in the instructions for the patients. *Always explain the prognosis/complications/adverse outcomes in simple words using language spoken by patients. It may be helpful to write about the treatment details/prognosis/outcome in the Hindi/local language on the discharge/follow up records so patient (and his relatives) can read and understand clearly.*

Team Training & Periodical Checking

Periodical training/checking your staff members and OT team is a must to ensure they follow the checklist and protocols to minimize any error(s) when the patient is taken for the surgery. Double-check the patient records, investigation reports, consent signed by the patient, site of operation, and medical records related to systemic illness, etc. For ophthalmic practice (for example), before taking the patient to the operation theatre, check the intraocular lens type and its power, carefully inspect the irrigating solution for any floating particles, always cross-check the date of expiry of drugs and devices. Train your OT team to always follow the practice to minimize postoperative endophthalmitis such as application of adhesive drapes, pre-operative cleaning of the eye and peri-ocular area with 5% povidone-iodine solution, and instillation of one drop of povidone-iodine solution after completion of intraocular surgery. Avoid unnecessary conversation (including jokes, irrelevant talk, scolding your staff, etc) in the operation theatre premises in presence of patient (or relatives) scheduled for any procedure or surgery. Exercising utmost care while performing any surgery under local or spinal anesthesia is important as the patient is actively listening to all conversations and may (wrongly) correlate negligence in case of lack of desired results.

Professional Liability Insurance

Doctors must cover themselves under professional liability insurance. One may take the help of medical societies for bulk purchase and to reduce the premium. A group of doctors can always negotiate better terms with the insurer than any individual. If the Insurance company is being changed, one should always insist on retroactive cover.

In summary, the number of cases against medical professionals for malpractice is increasing because of the increased internet awareness ('Dr. Google') among the patients. While very few cases may be legitimate and based on clinical negligence exercised by the doctors, most doctors are wrongfully accused because of the lack of public understanding. The medical professionals must communicate empathetically, emphasize diligent service delivery and also maintain proper records about the patient history, consent, and treatment. This practice will bring down the incidents of malpractice and will protect the doctors from allegations and fake lawsuits. It is imperative to take substantial measures to ensure due diligence while performing surgical procedures, follow the provided guidelines, and take all necessary measures before performing any surgery in the hospital. Following surgical checklists, protocols, proper documentation (maintaining medical records), taking informed consent, communication about the outcome of the procedure or treatment, timely referral of the patient (in case of any complication), and obtaining adequate professional liability insurance are a few important tips to minimize the risk of litigation against ophthalmologists and medical professionals.

There is an urgent need to evaluate the manner in which India chooses to address medical negligence/errors. In addition to the fear of defensive medicine, increasing insurance premiums and rise in costs for patients, it is time we are aware of the inequity that the present system perpetuates. Systemic deficiencies such as a very heavy malpractice/litigation costs, delayed and protracted litigation, as well as dependence on judicial discretion, do not provide effective justice to victims and could harm medical professionals and hospitals as well. In a developing country like India where there is an abysmal investment in health, the paucity of trained human resources, a huge gap between urban and rural health care, and poor political/administrative will to improve the health sector, it would be wise to implement a no-fault liability system within the public health sector and also to have caps on the types compensation after research and discussion. The government needs to act and invest in health care (at least 5 percent of GDP) before it is too late.

India needs to overhaul the present system of addressing medical negligence using all of the above-mentioned solutions effectively. It is time for Indian Medical Association (IMA), All India Ophthalmologica Society and other medical/ophthalmic societies to work together to sensitize the politician to exclude the medical services from consumer protection act.

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OPHTHALMOLOGY PRACTICE PEARLS

Why Must We Own Everything?

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No, this article is not going to be on Zen or Buddhist philosophy or even on communism. This is a practical piece for those who wish to survive in the big bad world of private practice. At every conference, when you wander around the Trade Exhibition area, you feast your eyes on all types of diagnostic gadgets, each more high tech and obviously more expensive than the other. There are newer perimeters (since they cost so much, they are now called visual field analysers), some even hand held and portable! Then there are nerve fibre analysers, OCT machines, Electrophysiology equipment like VEP, EOG and ERG (with multifocal ERG thrown in for free as a package deal). For those interested in retina, there are all sorts of fundus cameras, mydriatic and non mydriatic, OCT-Angio machines, Ultrasound machines. For the cataract and refractive surgeons, there are various tools to accurately calculate the astigmatism, both corneal and otherwise, higher order aberrations, sophisticated A scan machines for more accurate calculation of IOL power for their premium patients demanding premium IOLs. The cornea surgeons, not to be left behind can drool over fancy ocular surface analysers with built in infra red Meibography, osmolarity machines, various combinations of pachymeters with topography machines, Schiempflug imaging machines, anterior segment OCT machines, ultrasonic Bio microscopy machines or UBMs. By no means is this an exhaustive list – it is only meant to exhaust the reader who has reached thus far!

For the average private practitioner, it is of course impossible for him to acquire even a small fraction of these in his lifetime, so he stares enviously at his colleagues. The ophthalmologist in his lane owns one more machine than him; the ophthalmologist in the next lane owns 2 more machines than him! Hence he decides to take one more bank loan to buy yet one more "white elephant", knowing full well that almost all diagnostic machines never pay for themselves in 5 years. He hopes that seeing a clinic "well stocked' with gadgets will convince the walk in patient to trust his eye surgery to him, as that is where the 'moolah' lies.

Unfortunately this mathematical equation seldom plays out as he expects, making him spend many a sleepless night wondering how he will repay the bank loan and causing him to increase the dose of his antihypertensive pills. His family holidays are cancelled or indefinitely postponed, causing much friction between the spouses and even affecting his relations with his 2 young children.

Is there any way out of this maze or "chakravyuha"? There is no easy answer. First, the individual ophthalmologist must change his mind set. (1) He must realise the futility of trying to keep up with his neighbour, who is always trying to be one-up on him. Perhaps the neighbour has a 5 year head-start on him or has a friendlier bank manager who sanctions bigger loans or has a rich father in law or all of the above! Once the mindset is altered, solutions are possible. However, this need **"collegiality"**. (2) What does this term mean? The dictionary defines it as "companionship and cooperation between colleagues who share responsibility"

1. Companionship: He has to stop looking at his other eye surgeons in his lane or town as competitors but as colleagues who are all trying to improve their standard of living by offering better service to their patients.

2. Cooperation: He has to meet his colleagues in the lane or town and sit and discuss with them how they can pool the diagnostic equipment they have. Perhaps they could open a neutral diagnostic centre where a technician is employed or a junior doctor who can operate these gadgets and churn out reports. They could even pool in their money and purchase some more gadgets which they all believe they will be able to use. Each diagnostic machine will pay for itself if 5 or 10 ophthalmologists are using it instead of one! Also the expensive maintenance contracts will not seem so expensive if the cost is shared. The neutral centre could also allow referred patients from doctors who have not invested in the centre, perhaps charging such patients a little more. The responsibility of upkeep of the centre is shared by all the investor doctors! (3)

3. There are other options as well. In Goa, an eye surgeon stopped his own surgical practice and invested in various diagnostic equipments. He gets referrals from across Goa for Visual field analysis or OCT or ERG etc. The referring doctors are secure in the knowledge that they will not lose their patient and he uses the return on investment to purchase more equipment every year.

4. In Mumbai, a radiologist who was a pioneer in ophthalmic ultrasound, set up his own diagnostic centre. He now has OCT, UBM, Optical Biometry with the latest formulae for calculating IOL power for premium

IOLs, the only state of the art colour vision testing machine in the city etc. Many of us happily refer our patients to him. He sees dozens of patients every day and his reports are extremely accurate.

5. What about Surgery? Here again, in Mumbai as with most metro cities, opening your own nursing home or day care centre is a huge investment. Besides, we need a COU (Change of user) certificate, a pollution control board certificate, a nursing home certificate, a fire compliance certificate etc – to add to the misery, many of these need to be periodically renewed. Space is also expensive, so most end up with a single operating theatre and a single room where the post op patient is kept for 10 minutes and 'pushed' to leave to make room for the next patient! What if several colleagues get together and start a neutral centre where they can book their operating slots with a full time manager, have 2-3 OT. They can have their own fine instruments and share the Phaco and microscopes. At my own nursing home, there are 2 OT and 7 eye surgeons using the space. Of course, since they joined me after their own nursing homes closed due to reasons beyond the scope of this article, they have brought their own Phaco machines and microscopes with them. Luckily my N. Home has space to house all the equipment.

In conclusion, let us all not try to be the "complete clinic". Judicious use of our resources and maintenance of collegiality with our colleagues will go a long way in improving our bank balance and reducing our stress levels.

This article is only meant to serve as food for thought. You are free to implement what you find applicable in your set up or town. If none of these is relevant to you, my apologies for wasting your time!

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RESIDENT/YOUNG OPHTHALMOLOGIST CORNER

The Road to Perfectionism in the Surgical Field:

How Resident/Young Ophthalmologist Can Become Perfect Ophthalmic Surgeon?

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"The mind of Aesculapius, the eye of an eagle, the heart of a lion, and the hands of a woman."

-The 15th-century England's attributes of an ideal surgeon.

"Good surgeons know how to operate, better ones when to operate, and the best when not to operate. It takes wisdom, experience, strength, and courage not to intervene. The minute that a surgeon cuts the skin or a physician prescribes a drug, harm is done."

-Anonymous

Ophthalmology is one of the exciting sub-specialties of medical science with continued change and advancement in the field of investigations (tools) and micro-surgical techniques. Surgical training and transfer of surgical skills of new techniques to ophthalmology residents and newer generations of ophthalmologists is the need of the hour. Unfortunately, surgical training of ophthalmology residents in India and several developing countries in most of the residency programs has not kept pace with these advances. A transformation of the mindset, focus and curriculum of ophthalmic surgery alone can prepare the new generation of ophthalmology residents to be competent world leaders in this field. In the fast paced hi-tech world of modern ophthalmology, this is the least that we owe our trainees.

Medical profession is a field where one needs to consistently aim for perfection in order to build their competitive edge and retain optimum perfection. Surgery, ophthalmology and medicine all require a lifetime of learning, and learning requires repetition with aim of perfection. Entering into the surgical field (ophthalmology) is another critical stage where your life is on the verge of transformation as you make a career choice that is for the rest of your life. Moreover, ophthalmic surgery is a field that demands nothing less than perfection as even the most little mistake can lead compromised vision or even blindness.[i] During recent years most of the ophthalmic residents are not getting sufficient surgical cases for learning/mastering the eye surgery in most of the training institutes in India. *In this article, the authors share some pearls for learning and mastering eye surgery to become a perfect ophthalmic surgeon.*

What are the qualities of perfect surgeon? How resident or young ophthalmologist can learn these qualities? Why do we need to become perfect surgeons – especially when it comes to eye surgery and ophthalmology? Ophthalmologic surgeries are very delicate, highly crucial in nature. Any minor mistake can lead the patient to irrevocable damage in form of blindness or other serious visual impairment. This makes eye surgeries riskier than any other and require perfect surgeons on the list to execute surgeries and minimize the damage risk.

How to become a Perfect Ophthalmic Surgeon?

Perfection requires a surgeon to have a number of qualities to become best at what they do. Having these qualities make them a surgeon who is not only professionally competent, but personality-wise too. To become a competent surgeon in ophthalmology, the resident/young ophthalmologist needs to have following skills and qualities:

How to Learn and Master Surgical Skills: Wet Lab Training and Surgical Simulators

The surgical skills can be learned and refined by watching surgical videos on YouTube and using wet-lab training, training on surgical simulators before operating on real patients (Figure 1 A & B). This is the first quality that is extremely necessary to make you a perfect surgeon. You cannot expect to become an impeccable surgeon without having the mandatory set of skills require for your field of practice. Only if you are well-practiced and well-aware of the true skills of ophthalmology only then will you succeed in the field. With skills comes the precision of knowing how and when to utilize these skills in which situation.[ii] So, with your skills, you have to be highly precise too in using those skills. EyeSi Surgical offers an immersive environment for training of surgical steps. Through the simulator´s OR microscope trainees see the virtual surgical field in stereo and high resolution while operating with lifelike surgical

instruments. Just as in real surgery, discreet instrument movements are required to avoid undue wound stress, loss of viscoelastic, or diminished red reflex. The highly realistic simulation of interaction with tissue in real-time increases trainees' surgical experience – without risk for real patients.

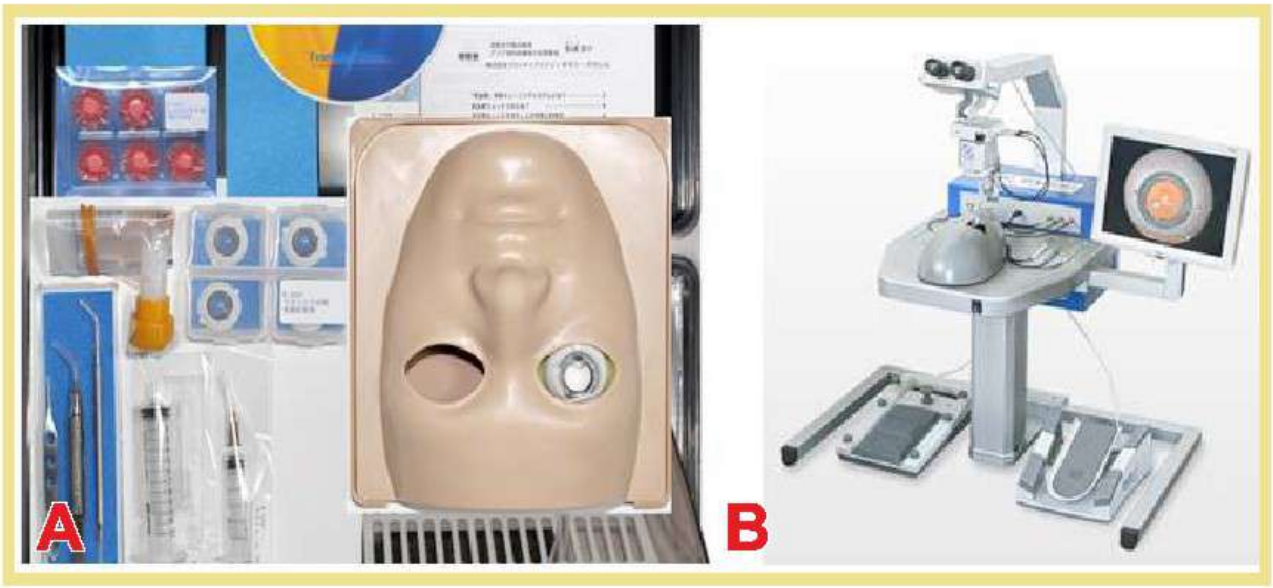

Figure 1. A: Kitaro Kit for dry and wet lab training for learning phacoemulsification and other eyue surgeries. B: The EyeSi surgical simulator is helpful in developing manual dexterity, hand-eye coordination, and comfort operating through a microscope.

The Art of Patient Satisfaction

Now this is something that is rare among surgeons yet highly sought after. Not every surgeon is as good at satisfying patients as they are with performing surgical procedures. This requires the surgeon to have highly proficient communication skills that can help them convey their message effectually to the patients and address any concerns they may have pertinent to their condition or the surgical procedure. Eye is a sensitive organ and vision a sheer blessing –to gain the trust of the patients, ophthalmologic surgeons need to be convincing in their communication to satisfy them.

Know the Advances and Innovations of Your Field

In the medical field, you can never be knowledgeable enough to say that you know everything and there is need for nothing more. The learning process for doctors is continuous, and a surgeon aiming for perfection knows the

importance of this process. They continuously work on increasing their knowledge base to ensure that optimum care service is delivered to the patients.[iii] Ophthalmology is witnessing inventions and innovations in the surgical procedures and discoveries of ophthalmologic conditions rather frequently. To become a perfect ophthalmologic surgeon you have to keep up with these advancements and discoveries to ensure you stay abreast of the research and deliver accordingly.

Becoming the perfect surgeon requires you to practice consistently and work relentlessly on refining your skills and knowledge to make yourself as updated as possible. Being an ophthalmologist, you need to further refine your skills because there is practically no (very minimal) room for mistakes. As you learn and perform delicate eye surgery as resident-in-training, you put your patients at risk. This is a tough truth and we all have dealt with it while training. The question is how to limit (minimize) that risk and add value to the patient who has agreed to let you operate on them. In most residency programs the resident get chance to perform surgery comes in the last year. However, it is better to start practicing surgery from the beginning of residency, which includes getting both hands going under the operating microscope in your practice lab, minor room or operating room. Resident should practice suturing, phacoemulsification on animal eyes or model eyes (Kitaro Kit or other surgical simulator) as this is helpful for hand eye coordination under the operating microscope. Practice using both right and left hands and assist the surgery (as much as you can) while keenly observing all minute details. During conference, never miss opportunity to participate in organized wet lab and simulation sessions seriously. Structured learning limits your risk to the patients. *Here are some useful tips that can help make you a more responsible and ultimately perfect ophthalmic surgeon:*

Carry Out a Pre-Op Preparation

A comprehensive pre-operative examination and investigations are very important before surgery as there is nothing worse than going into a surgery ill-prepared. It ultimately impedes your confidence level and curbs your from performing optimally. It is important that you take substantial time before the surgical procedure and give attention to the case you are about to perform. Talk to the patient, take their consent and listen carefully to what they have to say.[iv] This will enable you to know what thought process they are going through and how you can acquire their trust and satisfaction subsequently. Besides studying your case and talking to patients, the details of the operating room is another important thing you need to be watchful for. Having all your surgical items and other adjuncts (for example capsular dyes, new phaco tip, new blade, chondroitin sulfate based viscoelastic solution etc for doing a

phacomulsification of a hard cataract) in place and conducting your own survey of the room helps you prepare better while doing surgery.

Be Observant While Assisting Surgery

The best quality you can cultivate in yourself during your medical school and training years is that of observation and information absorption while in class room or in the operating room assisting surgery. These two skills are going to get you to the level of perfectionism. When you observe things and acquire information from them, it ultimately makes you more vigilant in your doing and careful in how you execute your procedures. So have a sharp eye for details so that you know what to be careful of and what mistakes you need to avoid.

Practice Makes Perfect

To nail your ophthalmic career, you need to develop a thorough habit of reading, discussing with your mentors, colleagues, watching surgical videos and practicing it a lot in wet lab or in the operation theater. Reading articles about ophthalmology, recently published cases and research studies about discoveries and trials can help expand your knowledge base and compel you towards critical thinking.[v] Never miss opportunity to participate in eye camps or assisting emergency ophthalmic surgery (for example repair of corneal trauma) during the odd hours.
Learn to use operating microscope and other surgical instruments and practice the art of suturing. Learn to respect delicate ocular tissues and never go for the short cut by following the dictum "do no harm". All this exercise will help you to refine your skills as a surgeon and makes you more abreast of what is happening in the field so that you can mold your practice accordingly and offer the optimum care.

Find a Mentor, Give Respect to Get Respect

There are many great teachers who are willing to mentor young ophthalmologists. You have to give respect to your peers, subordinates and patients to earn back their respect. A perfect resident is not the one who is perfect in the surgical room. He is perfect in all aspects outside the surgical room, too. As a matter of fact, you can only perform optimally in the surgical room if you are able to develop trust of those whom you work with and those you work for. So, bear this reciprocal method all the time – you are going to get what you give out.

Minimize Error by Following the Surgical Safety Checklist

The surgical safety checklist was developed after extensive consultation aiming to decrease errors and adverse events, and increase teamwork and

communication during the surgery. The surgical safety checklist has gone to show significant reduction in errors and is now used by majority of surgeons worldwide.

How Left-Handed Surgeon Can Learn and Master Eye Microsurgery?

It is a rather common question to ask whether left-handed people can become a perfect surgeon or not. Well, the answer is yes, they definitely can. Being perfect is not a skill or a certain art that needs to be learned. It is actually a matter of practice – a whole lot of a practice. The process may not be as easier for the left-handed people because the apparatuses and tools are designed keeping in mind right-hand users majorly, but nothing is impossible to do if there is consistent practice behind it. Any art can be mastered when you keep doing it consistently. Left-handed surgeons, however, have to face more difficulties as patients are not much comfortable with their laterality. [vi] One of the author (Dr. Suresh K. Pandey) is the left-handed surgeon. While working as an ophthalmologist, the author found it difficult to learn eye surgery but fortunately, he had the opportunity to learn eye surgery from a well-known left-handed (ambidextrous) eye surgeon from Sydney, Australia, Dr. E. John Milverton. Dr. Milverton shared his experience telling the author that he was left-handed but teachers in Australia tried to convert him into a right-handed person. The author was inspired from Dr. Milverton to became an ambidextrous surgeon by increase use of his right (non dominant hand). During past 25 years the author had successfully performed more than one lakh cataract surgery and IOL implantation and also participated in more than 20 ophthalmology conferences successfully performing live surgery. The author learned very important tip of training your non-dominant hand. While doing residency, it is very important for trainee ophthalmic surgeons to improve the hand-eye coordination of their non-dominant hand through practice. This need not be only in the operating room, wet lab, or surgical simulator. They can practice, for example, brushing their teeth or writing with their non-dominant hand.

In summary, a perfect surgeon needs to learn how to operate, when to operate and when not to operate. It is not just the medical profession they need to get a grip on, but should be able to integrate attributes of other professions, too. Having good memory and excellent accuracy, extensive knowledge and a curious soul, precision like an engineer and vision like a keen observer, reckoning like a lawyer and wisdom like a philosopher; a surgeon should be a know-it-all person having the essence of several professions to do the most crucial job in the world.[7-10]

Ophthalmologists work in a highly charged environment with a wide spectrum of patients. They are required to be decisive and yet dynamic in their approach to both clinical and surgical problems. Simultaneously, they must also promote

a positive workplace for a multidisciplinary team. They must be inspirational leaders who motivate others to work beyond the call of duty to improve patient safety and quality care outcomes. The path towards greatness is actually a continuous journey of self-improvement – but it is also the surest route to a satisfying and successful career in ophthalmology.

Ophthalmic surgeons must be technically sound; make sensible, logical decisions; communicate well with their patients; and know when (and when not) to operate.

- Perfection is the process of repeated practice. Resident in training, learn about using operating microscope, practice surgical steps in the wet lab using surgical simulator (or animal eyes) and then practice continuously to become a surgeon who is perfect in your field.
- With skills, work on precision as well to master the surgical procedures. Precision allow you to use your skills wisely and appropriately with accordance to the situation.
- A perfect ophthalmic surgeon has tremendous communication and interpersonal skills which they use for building patient satisfaction and developing competent team.
- Work continuously on increasing your knowledge base to ensure that optimum care service is delivered to the patients.
- During the surgical procedure, pay attention to the patient and talk to them about what and how they are feeling. Also focus on the details of the operating room to make process easier for you.
- Have a good eye for details. Observe things and acquire information from them, and keep yourself abreast of all the recent developments.

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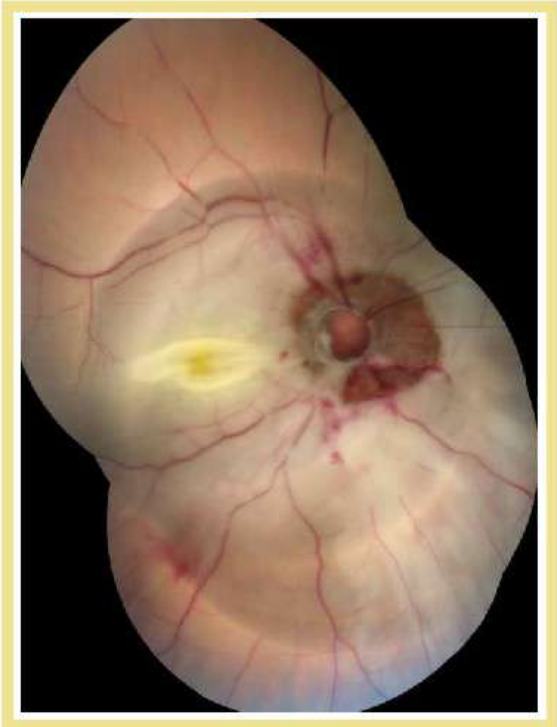
Image on the Cover Page

Contributed by Dr. Vishal Agarwal, MD, FRCS,

Dr. Agarwal Hospital, Jaipur, Email: drvishalrpc@gmail.com

Post Traumatic Optic Nerve Head Avulsion

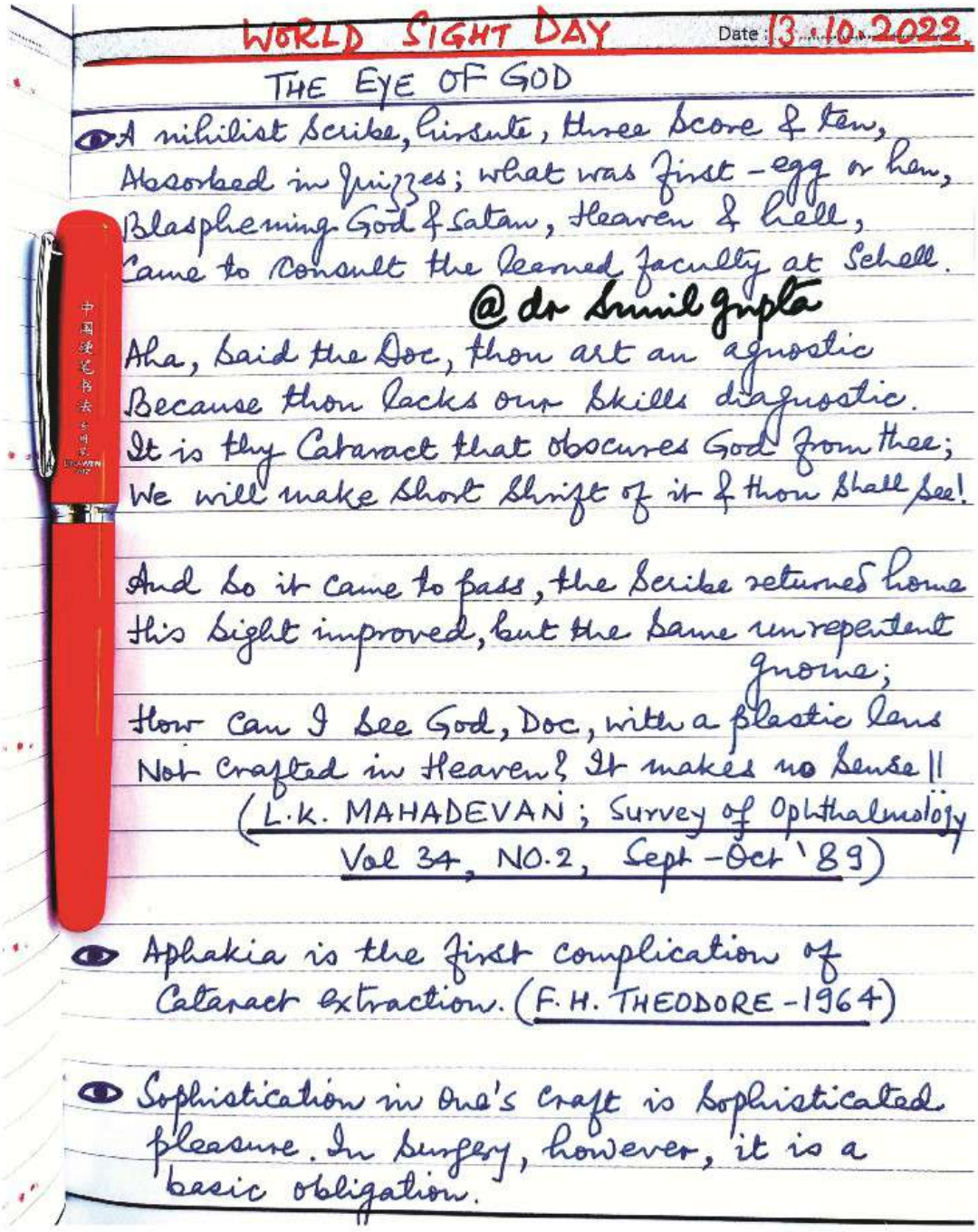
Optic nerve head avulsion is thought to generally result from injury to the head and/or orbit. Initially the injury may be difficult to diagnose. The vision may be profoundly impacted with no known medical or surgical therapy. The risk factors for optic nerve head avulsion follow the patterns for other ocular trauma. These factors would include: male gender, motor vehicle accidents, altercations, accidental finger pokes (such as with sports), and falls. Avulsion of the optic nerve head can occur from direct or indirect force resulting in a traumatic optic neuropathy.



Direct injury to the optic nerve head, from a penetrating orbital injury, is thought to be less common. Indirect injury can be from rapid torsional force to the globe resulting in shearing at the optic nerve head. Other potential mechanisms include sudden rapid increase in intraocular pressure blowing out the nerve and acute anterior displacement of the globe shearing the nerve from the globe. Prevention is focused on avoidance of potential trauma including use of appropriate eye protection for sports.

Ophthalmic "PEN"nings

Dr. Sunil Gupta, MS
Consultant Glaucoma Surgeon
Ram Avtar Eye Hospital & Glaucoma Pavilion, Jaipur



WORLD SIGHT DAY Date: 13.10.2022

THE EYE OF GOD

A nihilist Scribe, Cursute, three score & ten,
Absorbed in Quizzes; what was first - egg or hen,
Blaspheming God & Satan, Heaven & Hell,
Came to consult the learned faculty at Schell.

@dr Sunil gupta

Aha, said the Doc, thou art an agnostic
Because thou lacks our Skills diagnostic.
It is thy Cataract that obscures God from thee;
We will make short shrift of it & thou shall see!

And so it came to pass, the Scribe returned home
His sight improved, but the same unrepentent gnome;
How can I see God, Doc, with a plastic lens
Not crafted in Heaven? It makes no Sense ||

(L.K. MAHADEVAN; Survey of Ophthalmology
Vol 34, NO.2, Sept-Oct '89)

Aphakia is the first complication of Cataract extraction. (F.H. THEODORE - 1964)

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NOTES

NOTES

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Suncool™ Plus Eye drop 10ml
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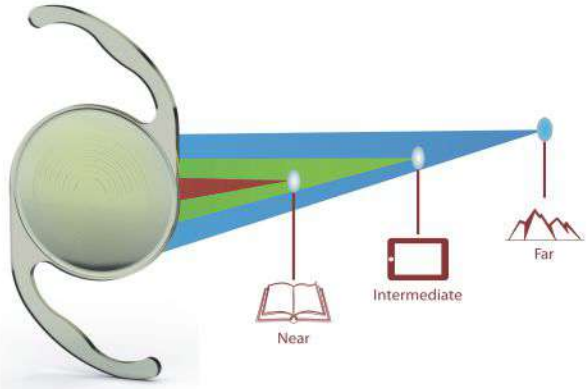

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