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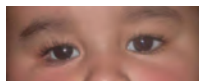


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Editorial

Technology versus skills: Push button pheco machines

Dear friends,

This past midterm a very interesting discussion was heard on technology versus skills. It seemed from the discussion that technology in pheco machines and perhaps in entire ophthalmology is advancing to such an extent that skills may be secondary in the making of a good ophthalmologist. If this was a discussion among machine engineers and company representatives falling over one another in showcasing their machines, I would have been happy as it would show that efforts are on to help minimize human errors in the science and art of cataract surgery and though the role of the human hand cannot be removed, science will advance to help reduce the learning curve associated with pheco surgery. It was amazing that the role of human skills and capability was being underplayed by the people that have tended to symbolize the very epitome of skillful cataract surgery and have set benchmarks for surgery outcomes that all of us aim to achieve but not all reach. They are the ones that have shown that cataract surgery is not only science and technology but an art. Watching the surgery of many of our experts is like listening to Mozart, seeing a Picasso, watching an old black and white movie.



Can a learner avoid accidents while driving a ferari instead of a maruti 800? Can technology replace the hand that wields it, guides it and controls it? Technology in cataract surgery has come a long way and I'm sure will continue to ease the life of the surgeon in making it safer and helping to give better and consistent outcomes. But till the time a push-button-do-it-all pheco machine is available, skills, expertise and experience will always separate the men form the boys. Hard work in acquiring skills and knowledge will always be an important ingredient for success.

The Editorial Committee looks forward for this technological marvel of a pheco machine while saluting the pioneers in surgery who continue to raise the bar of quality surgery.

Dr. Rohit Saxena

The Delhi journal of Ophthalmology is now indexed at Index Copernicus. The editorial board is involved in the task of getting the journal indexed in other sites as well as improving the quality of articles and their presentation. This is only possible with the support of each and every DOS member.

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Idiopathic Juxtafoveolar Retinal Telangiectasis

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Idiopathic juxtafoveolar retinal telangiectasis (IJRT), also known as idiopathic macular telangiectasia (IMT), refers to a heterogeneous group of well recognized clinical entities characterized by telangiectatic alterations of the juxtafoveolar capillary network of one or both eyes, but which differ in appearance, presumed pathogenesis, and management. Classically, three groups of IJRT are identified. Group 1 is unilateral, easily visible telangiectasis occurring predominantly in males, and causing visual loss as a result of macular edema. Group 2, the most common, is bilateral occurring in both middle-aged men and women, and presenting with telangiectasis that is more difficult to detect on biomicroscopy, minimal exudation, superficial retinal crystalline deposits, and right-angle venules along with characteristic and diagnostic angiographic and optical coherence tomography (OCT) features. Vision loss is due to retinal atrophy, not exudation, and subretinal neovascularisation (SRNV) is common. Group 3 is very rare characterized predominantly by progressive obliteration of the perifoveal capillary network, occurring usually in association with a medical or neurologic disease. This article presents a current review of IJRT, including the classification, clinical features, pathogenesis, complications, differential diagnosis, and treatment modalities.

Introduction

Idiopathic juxtafoveolar retinal telangiectasis (IJRT) is an uncommon cause of vision loss, which may be unilateral or bilateral. It comprises a group of retinal vascular anomalies characterized by retinal vessel dilation and tortuosity, multiple aneurysm formations, varying degrees of vascular leakage, incompetence and lipid exudate deposition. Visual loss is related to intraretinal edema, foveal atrophy and/or the development of subretinal neovascularisation (SRNV).[1,2] Historically, there has been confusion differentiating Coats' disease and IJRT. The term Coats' disease is now reserved for congenital retinal telangiectasis associated with massive exudation, retinal detachment and retinal degenerative changes. This differs from IJRT, whereby exudation or diffusion abnormalities from incompetent capillaries are confined to the juxtafoveolar region and are either of congenital or unknown origin.[3]

Classification

Gass,[2] who originally described this entity in 1968 and later coined the term IJRT in 1982, classified the disease into several types on the basis on biomicroscopic and angiographic findings.[3] In 1993, Gass and Blodi revised this classification and defined 3 distinct groups.[4] Group 1 patients have clinically visible retinal telangiectatic blood vessels and retinal exudation; group 2 patients have occult telangiectasis and minimal exudation; and group 3 patients

have clinically visible telangiectasis, parafoveolar capillary occlusion and minimal exudation. Subclassification of groups (Table 1) include those which are predominantly congenital, exudative and non-familial (groups 1A and 1B), and those that are primarily acquired, non-exudative, obstructive and occasionally familial (groups 2A, 2B, 3A and 3B). Group 2A is the most common subtype reported and its development has been summarized into five stages (Table 2).[3] Group 1A is the second most common.

In recent years, newly recognized manifestations have expanded and refined the clinical spectrum of these macular vasculopathies. Furthermore, the use of high speed angiography and optical coherence tomography (OCT) have provided a better understanding of the nature of the vascular abnormalities. In 2006, Yannuzzi et al proposed a simplified classification termed idiopathic macular telangiectasia (IMT) with 2 distinct types (type I, or aneurysmal telangiectasia, equivalent to group 1A and B and type II, or perifoveal telangiectasia, equivalent to group 2A).[5] The third type, occlusive telangiectasia, was omitted from the classification based on its rarity and presence of capillary nonperfusion rather than macular telangiectasia as the primary abnormality. Perifoveal telangiectasia was further classified in 2 stages: the nonproliferative stage when there are exudative telangiectasia and foveal atrophy, and the proliferative stage with the advent of SRNV.

Pathogenesis

Gass and Oyakawa[3] suggested that chronic venous stasis due to obstruction of the retinal veins as they cross retinal arteries on both sides of the horizontal raphe may be a cause of group 2A IJRT. Low-grade nutritional damage induced by specific retinal circulatory disturbances affects the retinal cells, particularly those at the level of the inner nuclear layer, which includes the Müller cells, leading to degeneration and atrophy of these cells and the connecting photoreceptor cells resulting in growth of vessels and the migration of retinal pigment epithelial (RPE) cells into the retina.[4] Abnormalities of glucose tolerance may be found in cases with Type 2A IJRT.[6] This supports the hypothesis that bilateral disease occurs in relation to a widespread metabolic disturbance in the retina, whereas unilateral cases represent a local and truly vascular defect.

Preliminary data using 2-wavelength autofluorescence imaging indicate that macular pigment density (MPD) is significantly reduced in the central retina. These recent findings have provided increasing evidence that group 2A IJRT is not a disease limited to the retinal vasculature but that neurons are intrinsically involved as well.[7]

Clinical picture and Diagnosis

The diagnosis of IJRT rests on a combination of stereoscopic biomicroscopy, fundus fluorescein angiography (FA) and OCT (Figures 1 – 4). On FA, the telangiectatic vessels are easily visible straddling the horizontal raphe and filling promptly in both the superficial and deep juxtafoveolar capillary plexus. Central cystic or noncystic macular edema is evident angiographically as late intraretinal staining. Diagnostic dilemma commonly exists to differentiate IJRT from occult SRNV or cystoid macular edema (CME) on FA. The merit of OCT is to provide information about the retinal structure and thickness in IJRT, as well as provide diagnostic clues in cases which are equivocal on FA. Following are the OCT features in IJRT:[8,9]

1. Foveal cyst in the innermost retinal layers – most common finding
2. Internal limiting membrane (ILM) draping across the foveola related to an underlying loss of tissue
3. Intraretinal hyperreflective lesions – second most common finding. They correspond to ophthalmoscopically visible hyperpigmented lesions.
4. Disruption of the inner segment/outer segment (IS/OS) photoreceptor (PR) junction line
5. Foveal detachment
6. Blunting of the foveal pit/foveal flattening
7. Foveolar thinning
8. SRNV
9. Lamellar or full thickness macular hole[10,11]

Whenever there is absence of macular oedema on OCT in spite

of prominent leakage of fluorescein in the fovea, IJRT must be suspected. Presence of foveal thinning despite occurrence of foveal cysts/detachment indicates that there is some degree of retinal atrophy and serves as a distinguishing feature of IJRT. Disruption of the IS/OS line can be visualised even in early cases with good vision, and does not necessarily indicate loss of the photoreceptor cells. Intraretinal RPE proliferation has been explained by the loss of PR cells, which allows the RPE cells to migrate into the overlying retina, especially along the venules. All eyes exhibiting RPE proliferation and migration demonstrate disruption of the IS/OS PR junction.[8]

Macular holes (MH) may occur as a sequel to chronic macular oedema. We would then expect macular holes to occur more frequently in association with IJRT. However, the rarity of MH in IJRT as well as the preservation of good visual acuity in patients with MH implies that the holes were the result of lateral separation of the photoreceptors within the fovea and that there could not have been profound atrophy of the photoreceptors. There is a loss of the structural aspects afforded by Muller cells, particularly the Muller cell cone, in the central macula in IJRT.[12]

Differential Diagnosis

When IJRT is suspected, it must be differentiated from venous occlusive disease, diabetic retinopathy, radiation retinopathy, Eales' disease, carotid artery occlusion and sickle cell retinopathy. Group 1 patients, in addition to that noted above, should be distinguished from those with Coats' disease which is defined by extensive peripheral retinal telangiectasis, exudative retinal detachment, relatively young age of onset and male predilection. Group 2 patients, during the early stage of the disease, may demonstrate foveolar atrophy that simulates lamellar macular hole formation, or may possess a yellow foveal lesion that may be mistaken for adult vitelliform dystrophy or Best's disease. In the late stage, patients who exhibit macular stellate pigment plaques with SRNV may be misdiagnosed as having age related macular degeneration (ARMD) or focal choroiditis. In differentiating patients with IJRT associated with SRNV from those with exudative ARMD, IJRT is rarely associated with pigment epithelial detachment and large neovascular complex formation. Group 3 patients who demonstrate atrophy of the juxtafoveolar retina with capillary occlusion and minimal exudation are most similar to those with sickle cell retinopathy.[13]

Management

Macular edema and exudation are the main cause of visual loss in group 1 IJRT; the amount of exudation, edema, and subsequent visual acuity loss is variable.[3,4] Treatment options include laser, intravitreal steroids, or anti-vascular endothelial growth factor (VEGF) agents.[15,16] Laser may not always be possible due to the close proximity

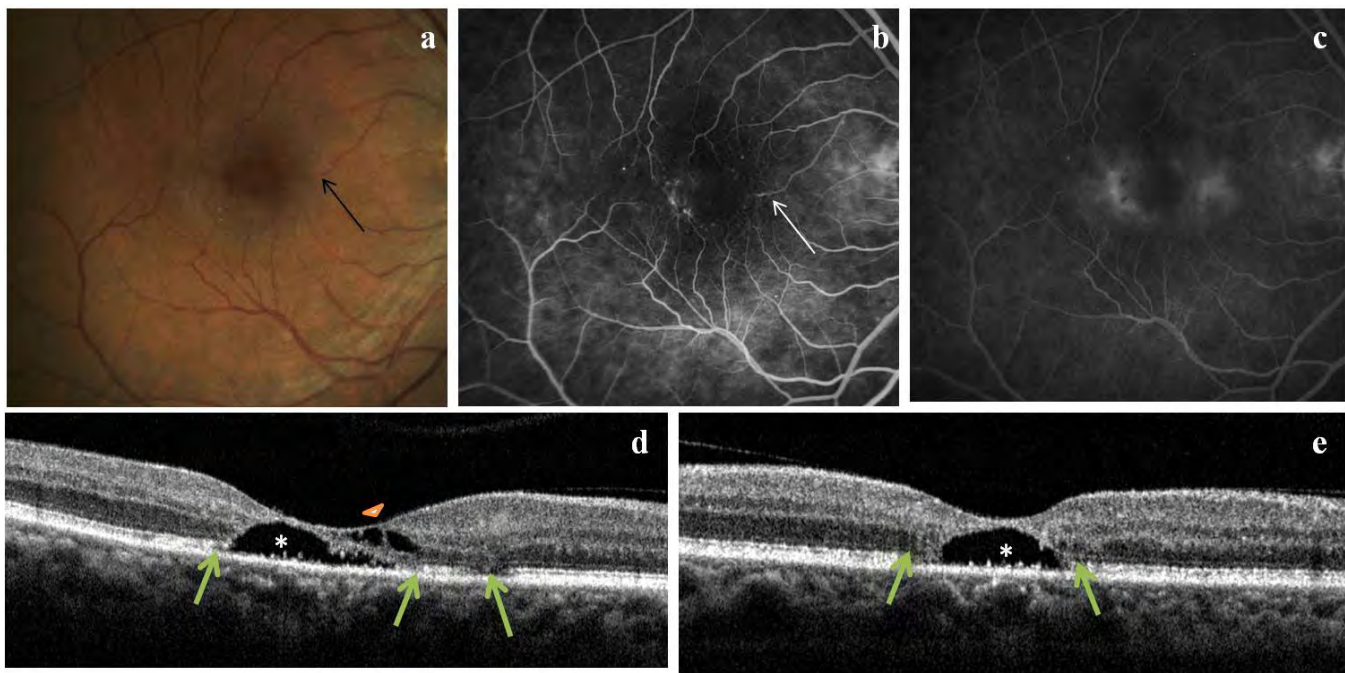


Figure 1 – (a) Colour fundus photograph of a patient with IJRT 2A showing a greyish ring around the foveal centre with numerous superficial retinal crystals and a blunted, right angled draining venule (arrow). (b) Corresponding fluorescein angiogram shows in the early phase clearly visible dilatation and telangiectasis of the perifoveal capillary network with confirmation of the right angled venule (arrow). These capillaries show late intraretinal staining (c). Horizontal (d) and vertical (e) OCT scans show foveal detachment (asterisk), subfoveal cysts (arrowhead) with partial loss of the highly reflective line considered as the boundary between photoreceptor inner segments and outer segments (arrows).

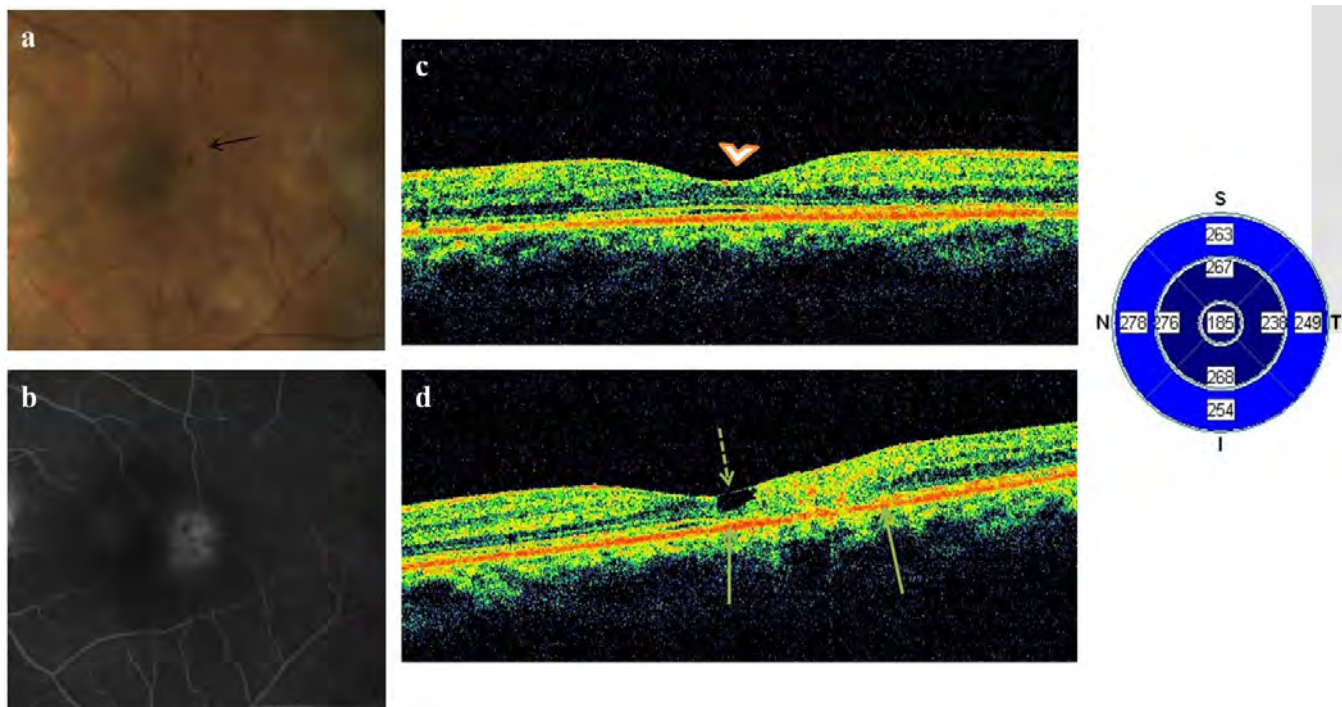


Figure 2 - (a) Colour fundus photograph of a patient with IJRT 2A showing a greyish ring around the foveal centre with early intraretinal pigment deposition in the vicinity of a right angled venule (black arrow). (b) Corresponding fluorescein angiogram shows late intraretinal staining. Vertical (c) OCT scan shows blunting of the foveal pit (arrowhead) with foveal thinning. Horizontal OCT scan shows the characteristic ILM drape (dashed arrow) with partial loss of the highly reflective line considered as the boundary between photoreceptor inner segments and outer segments (arrows).

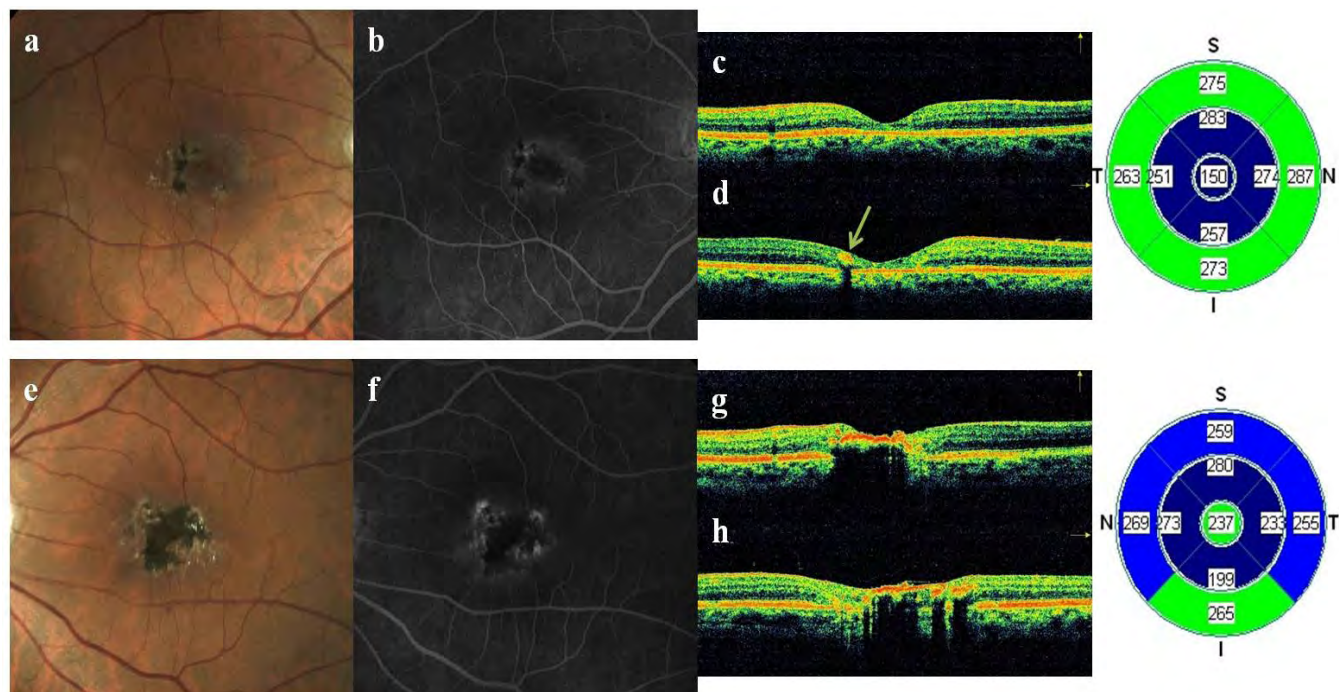


Figure 3 - (a, e) Colour fundus photograph of a patient with IJRT 2A showing stellate intraretinal pigment epithelial plaques, right angled draining venules and refractile retinal crystals (Stage 4). The disease is asymmetric, being more advanced in the left eye. (b, f) Corresponding fluorescein angiograms show blocked fluorescence due to the RPE hyperplasia with some intraretinal staining. Vertical (c) and horizontal (d) OCT scans of the right eye show blunting of the foveal pit, foveal thinning and a hyperreflective intraretinal lesion corresponding to the pigment (arrow). In the left eye, the flat pigmentary proliferation on the foveal surface masks the underlying retinal structure on OCT (g, h).

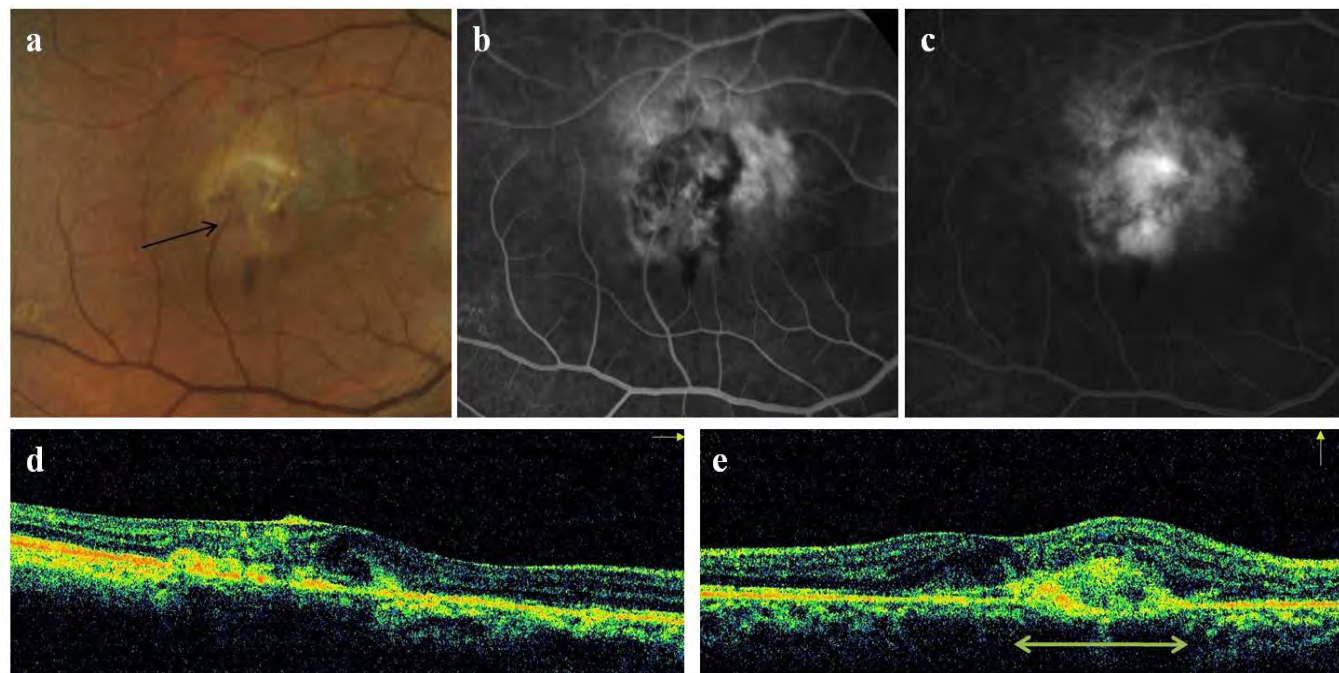


Figure 4 - (a) Colour fundus photograph of the right eye of the patient in Figure 2 showing temporal parafoveal retinal elevation with few crystalline retinal deposits, a right angled venule (arrow), nasal RPE alterations and subretinal blood characteristic of SRNV (IJRT 2A, Stage 5). (b) Corresponding fluorescein angiogram shows early hyperfluorescence with intense late leakage (c). Horizontal (d) and vertical (e) OCT scans show elevation of the juxtafoveal retina secondary to the presence of a hyperreflective fusiform complex lying at the outer retina/retinal pigment epithelium (RPE) level (extent marked by arrow) and minimal intraretinal fluid.

Table 1: Classification of IJRT

Group	Mean age of onset of symptoms	Predominance	Typical area of involvement, clinical picture	Visual loss	Systemic association
1A	40 years	Male, Unilateral	Telangiectasia and aneurysms temporal to fovea, 2 Disc Diameters	Amount of exudation and visual loss variable	None
1B	Middle age	Male, Unilateral	Focal, limited to two clock hour	Exudation and edema may or may not occur. 20/25 or better	None
2A	Middle age and older	Male=Female, Bilateral but asymmetric	Retinal thickening temporal to the fovea, right-angle venules, RPE hyperplastic plaques, superficial crystalline deposits, SRNV	Progressive	Possibly diabetes
2B	Juvenile	Bilateral	SRNV	Visual loss due to SRNV	None
3A	Middle age or older	Female, Bilateral	Minimal exudation, capillary obstruction and occlusion	Visual loss due to capillary obstruction and occlusion	Polycythemia, hypoglycemia, ulcerative colitis, multiple myeloma, chronic lymphatic leukemia
3B	Middle age or older	Male=Female	Minimal exudation, capillary obstruction and occlusion	Visual loss due to capillary obstruction and occlusion	With CNS vasculopathy

Table 2: Stages of Group 2A IJRT

Stage 1	Asymptomatic	Difficult to detect clinically Abnormal capillaries seen with fluorescein angiography (occult staining)
Stage 2	Asymptomatic	Mildly dilated perifoveolar capillaries Slight graying of the retina, mild loss of transparency Superficial refractile particles
Stage 3	Progressive decreased acuity	Dilated right-angled venules
Stage 4	Progressive decreased acuity	RPE hyperplasia clumped around the right-angled venules Pseudovitelliform lesion
Stage 5	Rapid and severe vision loss	Intraretinal and subretinal neovascularization, exudation and hemorrhage

of the abnormal vessels to the fovea. Also, due to lack of significant improvement in visual outcomes and increased risk of the development of SRNV following treatment, laser photocoagulation for macular edema associated with IJRT is currently not recommended.[14] Intravitreal triamcinolone acetonide (IVTA) is beneficial in the treatment of macular edema by its anti-inflammatory effect, downregulation of VEGF production, and stabilization of the blood retinal barrier.[15] Intravitreal injections of anti-VEGF agents, such as Bevacizumab, have shown improved visual outcome and significant and sustained decrease in leakage on FA and macular edema on OCT. It is likely that patients with group 1 IJRT with pronounced macular edema from leaky telangiectasis may benefit functionally and morphologically from anti-VEGF injections supposedly even at a lower treatment frequency than in other diseases.[16]

When considering treatment for group 2 IJRT, therapeutic attempts for nonproliferative IJRT and those for the SRNV of the proliferative stage must be distinguished. The angiographic late intraretinal staining pattern in nonproliferative IJRT 2A has prompted many ophthalmologists to interpret it as macular edema secondary to retinal vascular leakage. Several treatment modalities have been tried to treat this "macular edema." To start, laser photocoagulation is not effective in the treatment of nonproliferative IJRT 2A. In addition, treatment maybe associated with RPE changes, post-treatment retinal hemorrhages, and increased retinal vascular distortion.[17] Given that OCT shows that the fluorescein leakage seen is not associated with retinal thickening, the angiographic "leakage" is probably due to the staining of the extracellular matrix rather than extracellular leakage, and visual acuity correlates with photoreceptor layer disruption and not the degree of "leakage," IVTA is likely to have a minor or no therapeutic effect in nonproliferative IJRT 2A.[18] Recent publications on intravitreal anti-VEGF injections, namely Bevacizumab, report on possible short term benefits in some cases of IJRT 2A.[19,20] Inhibition of VEGF may be useful before atrophic changes occur. VEGF plays a pathophysiological role in IJRT 2A, because the structural capillary changes described histopathologically lead to a disturbed exchange of oxygen and substrates between the vascular lumen and neurosensory retina, which in turn may lead to a hypoxia induced increased VEGF release by retinal cells.[19] However, despite decreased leakage on FA, underlining the effect of Bevacizumab on vessel stability and permeability, small cystic changes seen on OCT and visual acuity may remain unchanged, emphasizing that visual deterioration is caused by microcystic degeneration and progressive retinal atrophy and not by intraretinal edema, and therefore cannot be halted with intravitreal anti-VEGF injections. Moreover, VEGF plays a role in photoreceptor differentiation and survival, and in maintaining retinal vascular homeostasis. Therefore,

blocking VEGF may accelerate apoptosis among ganglion cells and photoreceptors in IJRT 2A. [19] Given the current lack of convincing evidence of efficacy, the concern about the potential deleterious effects of repeated injections and cost of treatment, treatment of nonproliferative IJRT 2A with VEGF antagonists appears questionable.

Although rare, development of SRNV generally results in poor visual acuity if left untreated, with 80% (21 of 26) of eyes in one study having a final acuity of 20/200 or worse.[14] Histopathologic studies show that while neovascular membranes in ARMD originate from the choroidal vasculature, vessels in proliferative IJRT originate from the retinal vasculature and contain more vessels and less fibrous tissue. Before the advent of VEGF antagonists, therapeutic options for SRNV in IJRT 2A included laser photocoagulation, photodynamic therapy (PDT) with or without IVTA, transpupillary thermotherapy (TTT), and surgical removal of the CNV.[18] VEGF has been implicated as the major angiogenic stimulus responsible for neovascularization in IJRT 2A. Given the risk of permanent RPE damage with PDT, coupled with the huge evidence of efficacy of VEGF antagonists in the treatment of SRNV in various entities, the anti-VEGF approach is a reasonable treatment alternative for proliferative IJRT 2A. Anatomical peculiarities related to neovascular lesions in the setting of IJRT, such as the location above the RPE and the presence of anastomotic retinal vascular connections may facilitate inflow and concentration of the drug in the neovascular complex. Both intravitreal Bevacizumab (1.25 mg) [20,21] and Ranibizumab (0.5 mg) [22] have been used successfully in proliferative group 2A IJRT. Recently, primary treatment with combined intravitreal Bevacizumab or Ranibizumab and PDT have been reported anecdotally for proliferative IJRT 2A. [23,24] In both cases, the PDT was performed with a laser spot of the same size as the SRNV and followed by the intravitreal injection. Thus anti-VEGF therapy combined with or without PDT appears efficacious and should be considered as a treatment option for proliferative IJRT 2A.

Conclusion

IJRT comprises essentially three groups that differ in their appearance, presumed pathogenesis and management. In group 1, the unilateral telangiectasis is easily visible and vision loss is a result of exudation in the macula. Intravitreal steroids or Bevacizumab is generally effective in controlling the macular edema. In group 2, the most common, the bilateral capillary telangiectasis is more difficult to detect biomicroscopically, but the angiographic and OCT findings are characteristic and diagnostic. Vision loss is progressive and primarily due to retinal atrophy, not exudation or development of SRNV. Treatment options for this group are still limited, and have shown effectiveness only for the neovascular component. This is primarily because the pathogenesis of this telangiectasis

remains an enigma and is possibly secondary to a retinal neuronal dysfunction. New imaging modalities and functional tests will hopefully improve the understanding and treatment capabilities of this condition. Group 3 is a perifoveolar capillary occlusive condition and is poorly understood because of the scarcity of cases reported.

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Ocular Complications of Leprosy

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Leprosy is a chronic granulomatous disease caused by *Mycobacterium leprae*. The eyes are frequently affected in Leprosy and most eye complications occur in advanced lepromatous Cases [1] There is no systemic disease which so frequently gives rise to disorders of the eye as leprosy does. Extraocular structures and anterior segment structures of the eye are generally affected by leprosy. Posterior segment structures, particularly the choroid may show some pathology, in which lesions have been identified, though rarely. In posterior segment there may be yellowish nodules on the retina.

Direct injection of eye by *M. leprae* occurs mainly in lepromatous leprosy and is mainly blood borne. *M. leprae* may also reach the eye from the skin of eyelids, the meibomian glands or from the nose via the lacrimal drainage system.

Leprosy remains one of the world's major blinding disease and yet few ophthalmologists are aware of the spectrum of ocular [2] complications. upto 20% of leprosy patients develop sight threatening lesions and between 5-7% are blind. Visual impairment in leprosy needs special consideration by leprologists and ophthalmologists. Most of the ocular complications of leprosy are sight threatening and can be prevented if timely treatment is given. It is estimated that worldwide about three quarters of a million leprosy sufferers are blind. Blindness in persons suffering from leprosy is an irreversible double tragedy, since often such persons can neither see nor feel. Therefore involvement of the eye seriously affects the patients quality of life and causes an additional intolerable burden on him and his relatives.

Anterior Segment Complications

1. Corneal Complications

- Thickening and beading of corneal nerves (corneal anaesthesia)
- Superficial punctate keratitis
- Interstitial Keratitis
- Corneal ulcers.

2. Conjunctival Complications

- Chronic conjunctivitis
- Lepromatous nodules
- Erythema Nodosum Leprosum
- Pterygium

3. Ciliary Body and Iris related complications

- Iritis
- Iridocyclitis
- Cataract – chronic iridocyclitis may be responsible for the early formation of cataract. Steroids, used in the treatment of lepra reactions may hasten the formation of subcapsular cataract.

4. Episclera and Sclera

Common in untreated lepromatous patients

- Scleritis (commonly seen)
- Episcleritis (rare)

Posterior Segment complications

Leprosy lesions in the posterior segment are very rare. [3] There may be extension of lepromatous lesions from the ciliary body to the choroid and retina and may manifest as yellowish nodules on the retina.

Others

- Madarosis
- Lagophthalmos
- Chronic Dacryocystitis
- Entropion of upper eyelid
- Blepharochalasis
- Trichiasis

Corneal Complications

Cornea is supplied by anterior ciliary nerves which are branches of ophthalmic division of the fifth cranial nerve.[5] Due to infection of *M. leprae* nerves supplying the cornea becomes thickened causing corneal anaesthesia. Corneal complications in leprosy occurs due to corneal insensitivity and lagophthalmos caused by paralysis of the orbicularis oculi muscle due to infiltration of seventh cranial nerve more frequently the zygomatic branch.[4] Lagophthalmos may be partial or complete, unilateral or bilateral. The patient is unable to close the eyes and this results in a staring look, with the lids wide open (unblinking stare). He is prone to develop conjunctivitis, exposure keratitis and corneal ulceration resulting from failure of eyelid function eg. cleaning the cornea and keeping it moist. The patient at greatest risk of developing corneal ulceration and the other complications of

lagophthalmos as there is corneal hypoaesthesia. Patient is unaware of symptoms normally associated with dryness or injury. He neglects the eye until the condition has possibly become irreversible. Thus an ulcer may perforate causing intraocular infection, a common cause of blindness.

Continued exposure of conjunctiva leads to chronic conjunctivitis and erythema nodosum leprosum lesions may appear on the conjunctiva.

Cornea has no blood vessels[2] and *M. leprae* can invade the structure only by direct extension from surrounding tissues. Some believe that bacilli may move in along the nerves and form micronodular swellings. Since the cornea is transparent these changes are detected early as dense white grains of chalk. These are called corneal pearls. Appearing in other parts of cornea, these form diffuse superficial punctate keratitis (Fig – 1). As lepromatous leprosy advances, the corneal lesions get aggravated and cornea becomes vascularised.

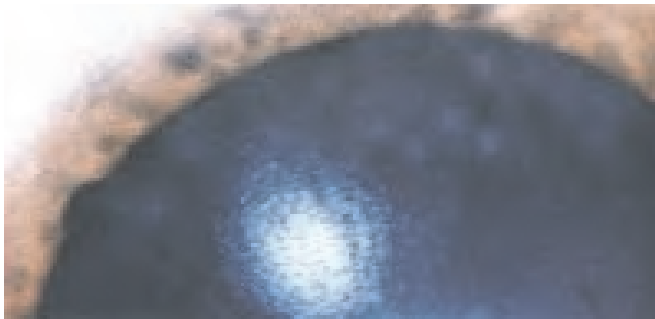


Figure 1 Superficial Punctate Keratitis

Ciliary Body and Iris Related Complications

Lepromatous iridocyclitis is one of the commonest cause of blindness in leprosy.[6] With bacillemia a common feature of leprosy is involvement of iris and ciliary body as both are highly vascular. It is likely that they are infected by the hematogenous route. The sphincter muscles of iris which are surrounded and infiltrated by lepromatous granuloma gradually undergo destruction.

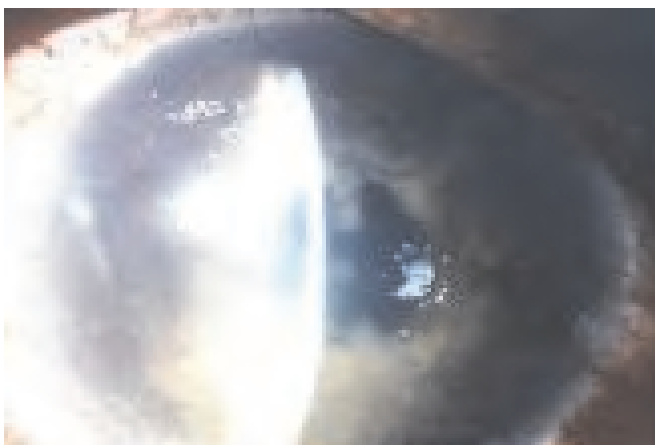


Figure 2 Lepromatous Iridocyclitis

Iritis & Iridocyclitis (Fig – 2) are all type of inflammation inside the eye and can all occur as part of a type-2 reaction.[4]

These conditions cause pain, redness, photophobia and loss of vision although the symptoms are not always severe.

The granulomatous lesion of iris with ulceration may produce an exudate composed of fibrin and polymorphs and the pupillary margins may adhere to the anterior capsule of the lens causes posterior synechiae resulting in a fixed, narrow, non reacting pupil (Fig – 3).[2,7] Eventually, destruction of the tissues of the Iris and ciliary body causes atrophy and shrinkage of the globe known as phthisis bulbi. Usually the granulomatous inflammation resolves with antileprosy treatment, but in some cases there may be continued presence of inflammatory cells resulting in persistent chronic silent iritis.

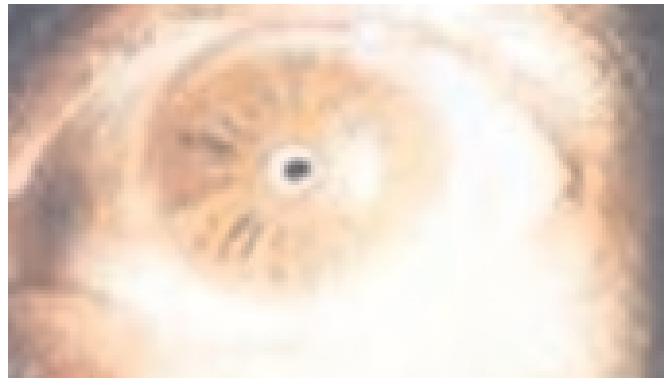


Fig-3 Fixed, Narrow, Non Reacting pupil

Chronic iridocyclitis may be responsible for the early formation of cataract. Steroids, used in the treatment of lepra reactions may hasten the formation of subcapsular cataract.[8]

Conjunctival Complications

Continued exposure of conjunctiva leads to chronic conjunctivitis,[4] and lepromatous nodules and erythema nodosum leprosum lesions may appear on the conjunctiva. [2,7] A mild conjunctival inflammation with edema and dilated blood vessels may be seen. Pterygium, with collection of macrophages containing *M. leprae* has been reported.

Episclera and Sclera

Scleritis is commonly seen in untreated lepromatous leprosy patients (Fig – 4).[7,9] Episcleritis is rarely seen. Scleritis occurs as part of a type-2 reaction. This involvement presents with nodules upto 5mm in diameter at sclerocorneal junction and may weaken the globe.

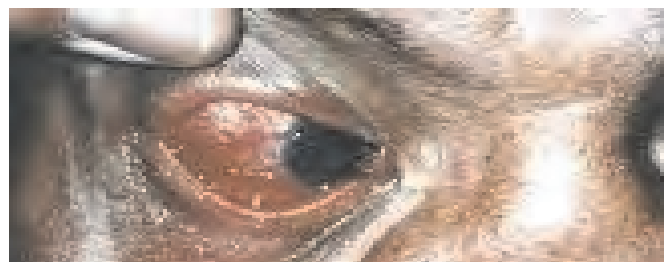


Fig-4 Scleritis

Posterior Segment Complications

The anterior segment of the eye, which is cooler than its posterior segment, most commonly affected by leprosy.[1,4] Leprosy lesions in the posterior segment are very rare and may manifest as yellowish nodules on the retina.

Other Complications

The thinning or baldness of the eyebrows is an early sign of lepromatous leprosy and is due to the deep seated infiltration.[2,4] This often leads, in advanced stage, to complete loss of eyebrows and is called madarosis. Lepromatous infiltration may cause of loss of some lashes and atrophy of the tissues supporting the remaining lashes which than hang limply against or actually turn in towards the eye causing trichiasis. Trichiasis causes irritation which may lead to corneal vascularity and opacity. Bilateral granulomatous infiltration of the lacrimal and meibomian glands in lepromatous leprosy and lacrimal gland in tuberculous leprosy is seen.[7] The eye is involved in all forms of leprosy, more in lepromatous than tuberculous leprosy.[10] Considering the seriousness of eye complications, repeated and careful examinations of the eye especially of those with lepromatous leprosy and those with nerve involvement affecting the eye can not be overemphasized, especially since M.leprae can survive in the iris and ciliary body long after skin lesions have become negative.

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Orbital space occupying lesions in children

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Space occupying lesion that contribute to orbital volume eventually cause proptosis. Malignant lesions have a faster pace of growth which if occurring in orbit manifests as rapid onset proptosis which surely raises a sense of alarm in parents as well as clinicians. In part I of this series we have detailed about approach to a child with proptosis. This article focuses primarily on malignant space occupying lesions in orbit in children.

In a child with an orbital mass which is presumably malignant complete work up in the form of history, general physical and systemic examination is mandatory. Ancillary investigations like imaging in the form of ultrasonography or preferably CT-scan or MRI and pathological studies are required for the diagnosis. Structurally distinct masses like cystic masses e.g. hemangiomas and dermoids may be diagnosed on radiology and a diagnostic biopsy procedure may be avoided. Systemic status need to be evaluated in all malignant cases so as to rule out any systemic metastasis in case of primary malignant neoplasm or to look for primary in case of metastatic orbital tumors. Investigative modalities like abdominal ultrasound, MIBG, and bone marrow aspirate or biopsy can be helpful to diagnose non-orbital primary tumors such as metastatic neuroblastoma and hematolymphoid malignancies or metastasis from a primary.

Pathological studies can be performed in the form of incision biopsies, excision specimens, and fine needle aspiration cytology (FNAC) material. Incision biopsy can be done more comfortably for anteriorly and laterally located tumors, while for more posterior tumors an exploratory orbitotomy may be required to take biopsy material. If the mass is well circumscribed it can be removed totally by excisional biopsy otherwise a debulking surgery may provide material for the pathological studies. Although tissue diagnosis is the preferred way of diagnosing the lesion by histopathology, FNAC may be helpful by providing a rapid diagnosis in case of rhabdomyosarcoma or hematological malignancy. A simple test like peripheral smear examination may serve as important investigative guide for hematological malignancies with orbital involvement. The material is subjected to routine histopathology or cytology. Special stains such as Periodic acid Schiff (PAS), Gomori methanamine silver (GMS) for fungal infections, or Ziehl Neelsens (ZN) stain to detect acid fast bacilli for tuberculosis may be used where required.

Immunohistochemistry (IHC) and molecular diagnostic studies can be done to confirm and refine the diagnosis in malignant neoplasms.

Incidence and differential diagnosis of orbital space occupying lesions in children

There have been a number of studies related to the incidence of various space occupying lesions in children.[1-7] There is marked variation in the reports of different series, depending primarily on the source and the age of the patients under study. Pediatric hospitals, ophthalmic hospitals, pathology referral centers, and different geographic areas have different frequencies of orbital lesions. Also, clinical and histopathologic series have varying profiles of diagnosis.

Bullock and Colleagues performed a study of both clinically and histopathologically diagnosed cases presenting to a clinical practice and compared their findings to nine other published series.[8] The common malignant lesions of the orbit in children were rhabdomyosarcoma, secondary malignant tumours/malignancies (including neuroblastoma, Ewing's sarcoma, and orbital involvement in retinoblastoma), lymphomas and leukemia. Incidence of lymphoma and leukemia was skewed by the African study, which found 60% of cases presenting with these diseases, reflecting the high incidence of Burkitt's lymphoma in this region.[9] This tumour is known to be the most common orbital malignancy among children uniformly in all parts of the world, but it is not a very common cause of childhood proptosis as a whole.[2-6] Depending on the patient population, surveys of orbital masses have shown that primary orbital rhabdomyosarcoma account for 1% to 3% of orbital masses that undergo biopsy in all age groups and 4% to 6% of orbital masses that undergo biopsy in children.[2-7,10-12]

In a series by Shields et al,[10] rhabdomyosarcoma represented only 3% of all orbital masses in children younger than 18 years. In a series by Bajaj et al, secondary orbital involvement of retinoblastoma was the most common cause of proptosis.[13] This finding contrasts with most reports from the west, where retinoblastoma is a relatively uncommon cause.[1-3] However, a study from Turkey found this tumour to be the most common etiology among children with proptosis, accounting for 34% of cases of orbital tumours.[6] A study from Nepal has reported proptosis with

orbital extension to be the most common mode of presentation (in 40% of these 43 patients) with retinoblastoma.[11]

Literature on the incidence and mode of presentation of pediatric space occupying lesions is thus conflicting, while most of the western literature report cystic lesions to be the most common, in the developing countries, orbital malignancies (rhabdomyosarcoma and retinoblastoma with orbital spread) have been reported as the most common cause of pediatric proptosis. Familiarity with the differential diagnosis and their common presentations will aid the clinician in making a timely accurate determination of the child's condition.

The malignant lesions in orbit in this age group can be primary or secondary. Although, the list of neoplastic lesions that can occur in orbit can be exhaustive due to almost all varieties of tissues that can be found in orbit like the eyeball and its tissues, the extraocular muscles, vessels, nerves, glandular and mesenchymal. We have described the usually encountered tumors in an ophthalmic oncology practice.

Primary orbital neoplasm

Rhabdomyosarcoma

Rhabdomyosarcoma is viewed as the most common orbital malignancy of childhood accounting for 24% of all orbital masses in children under 16 years.[13] This tumour occurs early in life, usually in the first decade. Although the most common age of presentation is 7 years, the disease has been reported as early as at birth[14] and as old as 78 years.[15] There is no apparent racial or hereditary predilection, but the tumour is thought to be slightly more common in males. The postulated origin of these tumours is from pluripotential mesenchymal elements.

Presentation and diagnosis

The classic presentation of the patient with orbital rhabdomyosarcoma is rapidly evolving unilateral proptosis with displacement of the globe (Figure 6). Eyelid erythema and conjunctival chemosis are also seen. Other less common presenting signs include ptosis, tearing, headache, nose blood and complaints of pain.[16] Nose bleeds are secondary to sinus involvement, with extension into the orbit. A palpable mass is seen at presentation in approximately 25% of patients.[16] Also less common are papilledema, choroidal folds and retinal vascular congestion secondary to large intracoronary lesions.[17] Rhabdomyosarcoma can also appear as an eyelid lesion simulating chalazia as dacryocystitis or as a subconjunctival mass and can have a more prolonged time course of presentation.

Ancillary studies, including orbital CT and MR imaging, can aid in the diagnosis of orbital rhabdomyosarcoma. A review of CT scans in 30 patients revealed the following radiological characteristics: irregular tumour shape with distortion of the globe and proptosis, moderately well-defined margins, soft

tissue attenuation similar to muscle, homogenous density and evidence of bony destruction in almost one half of cases.[18] One case had evidence of intralesional calcification. MR imaging reveals a signal similar to muscle on T1-weighted images and higher than muscle on T2-weighted images. CT may be a superior study because it allows for evaluation of bony erosion.

A complete blood cell count (CBC) should be performed in patients suspected of harboring rhabdomyosarcoma. A normal CBC with peripheral smear helps differentiate this disease process from orbital cellulitis and leukemia. As soon as possible, a biopsy should be performed to substantiate the diagnosis histopathologically. The surgical approach is by way of the pathway associated with the least morbidity. Eyelid and conjunctival lesions are biopsied directly. If the lesion is small an attempt should be made to remove it in toto. Vital structures, such as extraocular muscles, should be avoided, however, as the disease is very amenable to radiation and chemotherapy. Orbital lesions are approached by way of a conjunctival or skin incision.

Rhabdomyosarcoma can be divided into four main types: embryonal, alveolar, pleomorphic and botryoid.[19] The embryonal type is the most common, occurring in 2/3rd of cases, the alveolar is second most common and is the most malignant with a high frequency of metastasis; the pleomorphic type is the most differentiated type with the best prognosis. The botryoid variant is thought to be identical histopathologically to the embryonal type; however its growth has the classic polypoid appearance. Botryoid rhabdomyosarcoma is commonly seen in the genitourinary tract of female infants. In the orbit, it occurs in the anterior portion, where it grows as a polypoid, grape like mass beneath the conjunctival epithelium.

Management

When a patient presents with signs and symptoms consistent with orbital rhabdomyosarcoma, an orbital CT scan is obtained initially to outline the size and extent of tumour involvement. CT is preferable to MR imaging because visualization of bony erosion is useful diagnostically. A biopsy is then performed. Once the diagnosis is confirmed, a metastatic workup, including chest radiograph, liver function test, bone marrow biopsy, lumbar puncture and bone scan is performed, which aids in staging of the disease. All these investigations should be done on urgent basis for early treatment, as it is a rapidly growing tumour can lead to loss of vision secondary to exposure keratitis / optic neuropathy.

Over the last 40 years, treatment of orbital RMS has evolved from conservative surgery/biopsy and postoperative radiation therapy in the 1970s, to a multidisciplinary approach combining conservative surgery/biopsy and radiation for local treatment and systemic chemotherapy. The combined use of antimetabolite vincristine, antitumour antibiotics

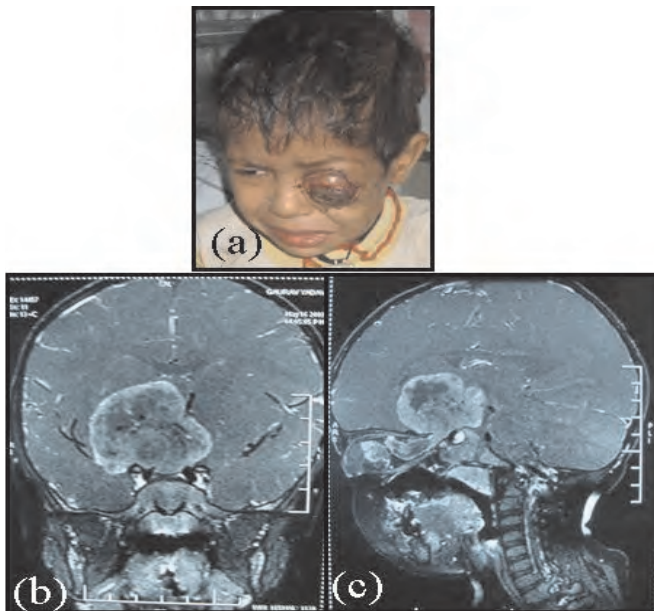


Figure 1- (a) Clinical photograph of a child with rhabdomyosarcoma presenting with severe proptosis. (b) CT scan of the same child showing heterogeneous mass occupying whole of left orbit.

actinomycin D and adriamycin, and the alkylating agent cyclophosphamide following irradiation, has permitted more complete tumour eradication in the orbit and suppression of potential metastasis. With improved outcome, late effects of treatment become more important to consider. Late effects of radiotherapy include functional and structural effects, such as facial bone hypoplasia, cataract formation and growth hormone deficiency.[20-24] The challenge of present day management protocols is to maintain excellent survival while at the same time avoiding the late effects of treatment. Treatment is constantly being improved with new technologies in radiation oncology including improved planning systems, 3-D conformal radiotherapy, intensity modulated radiotherapy (IMRT), proton therapy and tomotherapy for reduction in late effects.

Orbital spread of intraocular tumor

Retinoblastoma

Retinoblastoma is the most common intraocular malignancy diagnosed in children, accounting for 3% of all childhood malignant tumours in developed countries, the rate being much higher in developing countries[25] and late referral might account for the delayed diagnosis (Figure 7).[26] The survival of patients with retinoblastoma has gradually improved over the years,[27] in part because of the introduction of a multimodality approach. When tumour extends beyond the globe into the orbit, a combination of radiation therapy and chemotherapy is being used to prevent exenteration.[28] Multidrug treatment strategies warrant further investigation to improve outcome as well as minimize toxicity.[29] Prognosis remains relatively poor for patients whose disease

disseminates into the central nervous system (CNS) and those with distant metastatic disease.[30]

According to the recent CCG (Childrens Cancer Group) classification for extraocular retinoblastoma, it is classified into 5 groups - Class I:Microscopic involvement of the scleral emissaries; Class II:Microscopic involvement of the cut end of the optic nerve; Class III:Orbital disease in the biopsy; Class IV:CNS disease with brain mass or CSF with positive tumour cells; Class V Blood-borne metastases to bone marrow, bone, or lymphatic metastases to lymph nodes.

Various staging systems for extra-ocular retinoblastoma have been established by different groups, of which one of the most commonly used is that by Chantada et al (table-I).

Table-I: Classification of Retinoblastoma by Chantada et al

Stage 0. Patients treated conservatively

Stage I. Eye enucleated, completely resected histologically

Stage II. Eye enucleated, microscopic residual tumour

Stage III. Regional extension

a. Overt orbital disease

b. Preauricular or cervical lymph node extension

Stage IV Metastatic disease

a. Hematogenous metastasis (without CNS involvement)

1. Single lesion

2. Multiple lesions

b. CNS extension (with or without any other site of regional or metastatic disease)

1. Prechiasmatic lesion

2. CNS mass

3. Leptomeningeal and CSF disease

Stage I patients require standardized substaging of resected microscopic disease. Stage II includes patients who underwent enucleation but have microscopic residual disease. Included in this stage are patients with involvement of the optic nerve to the transection line and those with microscopic trans-scleral invasion. In these group of patients, adjuvant therapy is given in form of radiotherapy and chemotherapy with various regimens. There is uniform agreement of the need for adjuvant therapy in these latter patients, but there is some controversy regarding their need for radiation therapy. Stage III includes patients with overt regional extension (orbital or lymph node involvement) in whom a combined approach using surgery, chemotherapy and radiotherapy is the standard of care. Survival can be achieved with conventional therapy in the majority of patients. Stage IV includes patients with hematogenous metastases and those with CNS disease. Successful treatments using very high dose chemotherapy with autologous stem cell rescue have been reported for patients with hematogenous metastases. In contrast, patients with CNS disease continue to have a poor outcome. However,

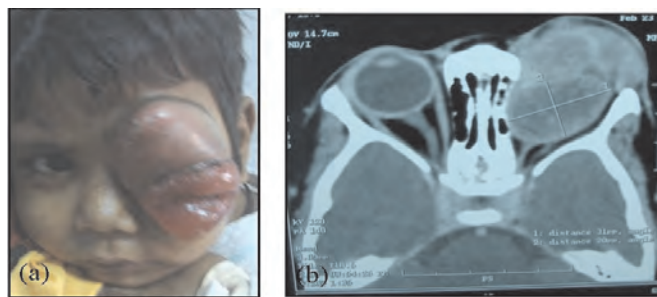


Figure 2 - Extraocular retinoblastoma. (a) Clinical photograph of a child with initial history of leukokoria presenting with proptosis OS, MRI (b) Coronal and (c) sagittal sections revealing involvement of total length of the optic nerve and intracranial involvement.

some patients with prechiasmatic CNS disease as their only metastatic site may have a more favorable outcome, so they are considered separately in this classification. Chemotherapy and external beam radiation therapy continue to be a mainstay in the treatment of patients with advanced retinoblastoma. Radiation therapy is important to achieve local control in patients with optical nerve disease and orbital involvement. Patients with CNS dissemination or metastatic disease continue to die with progressive disease and remain incurable, despite the aggressive treatment. Awareness through education and outreach to the community can help in early referral, so that vision can be preserved and survival may be improved.

Metastatic tumors

Leukaemia

Leukemias are a group of heterogeneous neoplastic disorders of white blood cells. Based on their origin, myeloid or lymphoid, they can be divided into 2 types. Leukemias traditionally have been designated as acute or chronic, based on their untreated course. Leukemia may involve almost any ocular tissue, by direct infiltration, by hemorrhage, and by ischemic changes. Orbital involvement is not a rare complication of leukemia. It occurs mainly in children with acute myelogenous leukemia (AML) and adults with chronic lymphocytic leukemia (CLL).

Three patterns of orbital involvement have been described by Valvassori besides hemorrhagic complications.[31,32]

1) Involvement of the uvea, choroid, retina, and optic nerve. On imaging, there is diffuse or localized thickening of these structures. Leukemic infiltration of the optic nerve can result in rapid loss of vision and should be promptly recognized and treated.

2) Infiltration of the orbital soft tissues. Usually this is limited to the orbital fat, but it can extend into the lacrimal gland. In CLL in adults, it may involve the extraocular muscles. On MR imaging it appears as an area of medium to low signal intensity, more intense than the vitreous and isointense to

muscles on T1, becoming only slightly more intense on T2.

3) Granulocytic sarcoma with bone erosion in the absence of peripheral blood involvement. This lesion is described by Valvassori as centered in the orbital subperiosteal space, usually involving the lateral wall of the orbit. It may extend into the temporal fossa as well. It can involve into the medial wall of the orbit and extend into the ethmoid air cells, cribriform plate, and occasionally the anterior cranial fossa. Patients with leukemia are prone to hemorrhagic complications due to thrombocytopenia or platelet dysfunction. Whenever acute proptosis occurs, CT or MR imaging is useful to differentiate retro-orbital bleeding from leukemic infiltrates. A peripheral smear is thus an invaluable aid in children presenting with proptosis to rule out leukaemia.

Neuroblastoma

Neuroblastoma is a common childhood cancer, accounting for 8% to 10% of all childhood malignancies.³³ The median age for diagnosis of neuroblastoma is 22 months, with the majority of cases occurring before 5 years. Metastasis to the orbit is seen in approximately 20% of cases, although not usually as a initial finding. In case of neuroblastoma metastatic to the orbit, the most common site of the primary tumour is the adrenal gland.

Unilateral or bilateral proptosis and lid ecchymosis are the classic presentations. Patients may also have swelling of the eyelids, ocular motility disturbances, ptosis and Horner's syndrome caused by a thoracic tumour.[33] Additional signs and symptom may include abdominal fullness and pain, venous obstruction and edema, hypertension caused by renal vasculature compromise and bone pain. Incisional biopsy, demonstrating the typical small round blue cells, confirms the diagnosis. Urine analysis for catecholamines is positive in 90% to 95% of cases.

Staging, as defined by the International Neuroblastoma Staging system (INSS), is based on the extent of the tumour at presentation. By definition, children who present with orbital disease are stage IV caused by distant dissemination of tumour. The child's prognosis is indicated by the determined disease stage, considered along with his or her age and site of primary tumour. Younger children, especially those less than one year of age have a better prognosis.

Treatment of neuroblastoma includes surgery, chemotherapy, and radiation therapy. Despite intensive treatment with chemotherapy and bone marrow transplantation, children with orbital metastasis and stage IV disease have a survival rate of less than 15%.

Ewing's sarcoma

Ewing's tumour is a primary tumour of bone in childhood that only rarely involves the orbit.[34] Most such cases are metastatic from distant sites. In most cases with orbital

involvement, ophthalmic symptoms consist of proptosis, pain, and occasionally visual loss and motility restriction. The diagnosis is typically unsuspected before histologic evaluation. Electron microscopic and immunohistochemical analyses are essential in making the diagnosis. Local treatment relying on surgical extirpation and radiotherapy alone has proven inadequate, with 5-year survival rates of <10%. The addition of chemotherapy has improved survival rates significantly to about 50%.

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Ophthalmic Viscosurgical Devices

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Ophthalmic viscosurgical devices(OVDs) or viscoelastic agents are becoming a necessity in many ophthalmological procedures today.

Viscosurgery is a term used to designate the procedures and manipulations performed with viscoelastics. The aims of Viscosurgery and hence OVDs are as follows:

1. Prevention of tissue damage
2. Wider space for manipulation
3. To avoid adhesions post operatively

This article will discuss the different types of viscoelastics, their behaviors and clinical tips for using viscoelastics appropriately.

Rheological Properties of OVDs

OVDs are transparent, gel-like substances that have viscous and elastic properties. There are essentially four different properties to mention when discussing OVDs:

- **Elasticity:** the tendency of a viscoelastic to go back to its original shape after it is deformed or stretched.
- **Viscosity,** or resistance to flow, is primarily determined by the molecular weight and concentration, so that the higher the viscosity, the better the OVD at displacing tissue and staying in place.
- **Pseudoplasticity;** the ability of a viscoelastic to transform under pressure from a gel to a liquid substance. This property enables easy injection and removal of an agent at increasing flow rates.

- **Cohesiveness/dispersiveness** or whether the agent adheres to itself or to surrounding tissues

Cohesive OVDs, such as Healon, ProVisc, and Amvisc, are long chain, high molecular weight, high viscosity substances that act like “spaghetti”. These agents maintain space well at no or low shear rates, while at high shear rates they are easily displaced. The advantage is that they are easier to remove from the eye since they stick together and are aspirated as long pieces. Thus, there is less chance of retention and risk of an IOP spike. However, they have minimal coating ability and therefore afford less tissue protection during surgery.

Dispersive OVDs, like Viscoat, DisCoVisc are short chain, low molecular weight, low viscosity substances with low surface tension that act like “macaroni”. These properties produce excellent coating and protection at high shear rates; however, they are more difficult to remove from the eye since they don’t stick together and are aspirated in short fragments. Therefore, they have an increased risk of elevated IOP.

Viscoadaptives:

Desired Properties of an Ideal OVD

1. Ease of infusion
2. Retention under positive pressure in the eye.
3. Retention during phacoemulsification
4. Easy removal
5. Does not interfere with instruments or IOL placement.

TASK	FUNCTION NEEDED	AGENT TO BE USED
Capsulorhexis	maintain a deep anterior chamber	Cohesive
Emulsification	Stay in eye to cushion and coat tissues	Dispersive
Remove cortex	Endothelial coating	Dispersive
Open bag and insert IOL	Maintain deep chamber and bag	Cohesive
Remove OVD at inclusion of surgery	Remove quickly and completely	Cohesive
SPECIAL CASES		
Compromised cornea	Coating the cornea for protection	Dispersive
Very shallow anterior chambers	maintain a deep anterior chamber	Cohesive
Small pupils/floppy irises	For opening up the eyes,	Cohesive
Dense cataract	Endothelial coating	Dispersive
In eyes with small pupils	Viscodilation	Adaptive like healon 5

6. Protects the endothelium
7. Non toxic
8. Does not obstruct aqueous flow
9. Clear

Clinical Uses of OVDs

- OVDs are used during surgery in order to maintain and preserve space, displace and stabilize tissue, and coat and protect tissue.

USES

1. Cataract surgery
2. Corneal surgery and penetrating keratoplasty
3. Glaucoma surgery
4. Ocular trauma
5. Posterior segment surgery
6. Strabismus surgery

Viscoelastics In Cataract Surgery

Since their introduction in the early 1970s, ophthalmic viscosurgical devices (OVD) have revolutionized cataract surgery as well as IOL implantation.

Remember

- When first filling the eye with OVD, surgeons should move all the way across the anterior chamber and start injecting from the opposite side and backfill the anterior chamber. This will push all of the aqueous humor out of the eye.
- For infants and children, surgeons should overfill the anterior chamber to flatten the anterior surface of the lens. This will provide increased control over the capsulorhexis.
- With Healon 5, before hydrodissection is started, create a tunnel through the viscoelastic so that the aqueous injected for hydrodissection has a way to get out. Otherwise the pressure will increase greatly and the iris may prolapse.

Soft-shell technique

- The technique was given by Arshinoff (1999)(Figure1)
- Using dispersive and cohesive OVDs creates a smooth, flat layer of dispersive OVD adjacent to the corneal endothelium over a high-viscosity, cohesive OVD.
- This technique is effective in reducing corneal endothelial cell loss after phacoemulsification surgery, especially in eyes with dense nuclear opacity, when compared with results using a single cohesive or dispersive OVD only

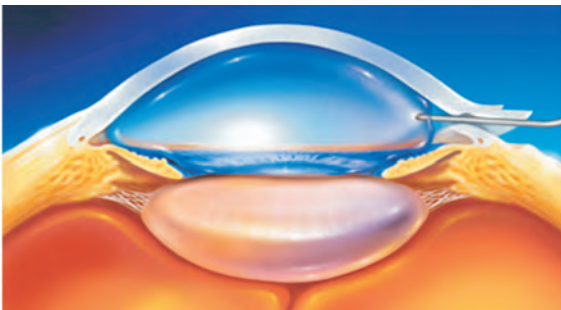


Figure1: Soft shell technique

Ultimate soft shell technique

- Arshinoff1 (1999) has developed a new technique called the ultimate soft-shell technique.
- This technique compartmentalizes the anterior chamber using the ultimate low-viscosity fluid (balanced salt solution or trypan blue) underneath viscoadaptive OVDs with which the anterior chamber is filled to the desired extent (for capsulorhexis, 60%–80%; for capsular staining, 90%; for IOL implantation, 60%).
- The technique reduces the resistance to advancing the capsulorhexis with a needle or forceps while maintaining tamponade to the lens surface well, and also reduces the amount of dye required for capsular staining of mature or white cataracts.

Viscoelastics in glaucoma surgery

1. Cohesive viscoelastic have been tried to reduce the corneal toxicity of subconjunctival 5FU injections given after trabeculectomy. Corneal epithelial toxicity of 5-FU is attributed to its leak into the tear film via the injection site that is used to enter subconjunctivally in the first place. To negate this, first cohesive viscoelastic like Healon GV is injected subconjunctivally, then 5-FU is injected through the Healon GV thus placing the “Healon wall” between the 5-FU and the injection hole(Figure2). This stops the 5-FU from leaking out through the tear film protecting the corneal epithelial problem. Also, because it delays the flow of 5-FU into the tear film, the 5-FU lasts much longer.[2]
2. Hyaluronic acid facilitates and maintains separation of dissected structures in glaucoma microsurgery, thus preventing complications during surgery and in the immediate postoperative phase. Goniotomy is more easily controlled and is safer. Hyaluronic acid can therefore be used in glaucoma microsurgery to keep prepared layers of tissue separated for longer and can enlarge newly created space and almost eliminate the adhesive forces between the layers. Using hyaluronic acid the safety of glaucoma microsurgery is enhanced and the complication rate reduced.[3]

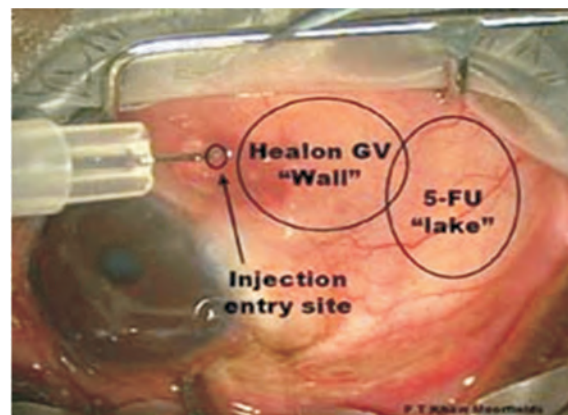


Figure2: Healon GV wall to prevent 5-FU corneal toxicity

Viscoelastics in posterior segment surgery

1. Use of viscoelastic materials in the vitreous cavity during removal of large and heavy intraocular foreign bodies adds to the safety of surgery and can protect the fovea in cases when inadvertent drops occur.[4] Dispersive viscoelastics exert resistance against the dropping foreign body because of their high viscosity and dampen the kinetic energy of the IOFB, reducing the risk of damage in case of drop on the fovea. When the dropping foreign body comes in contact with the viscoelastic material, it is slowed down and may change direction. Theoretically, cohesive materials are better than dispersive ones for this purpose; their greater surface tension provides additional resistance against the force of the drop and may even prevent the IOFB from entering the OVD bubble causing it to float over the bubble. But the major concern with their use is difficulty in removing them from the posterior segment through a 20-gauge needle.

Another concern is that by forming a bubble with convex surface, the IOFB may float and displace peripherally where removal may be more difficult and entail complications. It is prudent to completely remove all viscoelastic material from the vitreous cavity to avoid inflammation.

2. Sodium hyaluronate has also been reported to be effective in posterior segment surgery for dissection of epiretinal membranes, e.g. proliferative diabetic retinopathy and macular pucker.[5]

Viscoelastics in ocular trauma Canalicular injury

Viscoelastics have a role in canalicular repair where the uninjured canaliculus is irrigated with fluorescein dye tinted viscoelastic, that spills from the other end hence helping to locate the proximal end of the injured canaliculus.

Viscoelastics in strabismus surgery

The efficacy of hydroxypropylmethylcellulose and sodium hyaluronate as adjuncts during adjustable strabismus surgery has been evaluated experimentally in studies done on rabbits. It has been found that the force required to bring the muscle to its insertion is significantly less with the use of subconjunctival viscoelastic. Also it has been reported in studies done on rabbit eyes that subconjunctival use of viscoat with antifibrotic agents like 5FU can delay the postoperative adjustment by one week[6]

Complications of OVDs

- Elevation of IOP
Healon block glaucoma – it was first noted with use of healon, elevation of IOP is severe and prolonged, if the material is not thoroughly removed at the conclusion of surgery. This increase in IOP is dose related and transient, occurring in first 6-24 hours and resolving spontaneously in 72 hours post operatively. This ocular hypertensive effect is the result of large molecules of OVDs creating

mechanical resistance in trabecular meshwork and hence decreasing the outflow. Therefore, materials possessing lower viscosity and lower molecular weight clear eye faster and reduce IOP elevation.

- Plastic anterior uveitis
Because of viscous nature and electrostatic charge of these materials, inflammatory and red blood cells may remain suspended in anterior chamber, giving appearance of plastic anterior uveitis.
- Calcific band keratopathy
- o Post operatively, sub epithelial corneal deposits identified histochemically as calcium phosphate precipitate were associated with use of intracameral viscoat and OVDS which had phosphate as buffer.
- Capsular distention syndrome
- o It was described by Davison and Masket. In this syndrome, OVD is retained and entrapped behind the IOL. As the anterior capsulorhexis adheres to the anterior surface of IOL and entrapped OVD denatures, IOL is forced anteriorly by the pressurized capsule and the posterior capsule is distended posteriorly. Patient complains of reduced distance visual acuity and improved near acuity due to induced myopia from forward shift of IOL. IOP is normal, despite shallow anterior chamber. Treatment is done by yag laser application to anterior capsule to allow OVD to escape anteriorly or posterior capsule may be lasered with escape of OVD posteriorly.
- Formulation problems
- o HPMC is known to contain micro organisms. There have been reports which suggest, healon may increase fungal growth.

Thus viscosurgical devices have found profound applications in ophthalmology and have become indispensable tools in many surgical procedures.

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Periocular injuries following unknown animal bite

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We report an unusual case of unknown animal bite over left side of face with eyelid avulsions. A 15 years old child presented to us with a history of un-known animal bite on left side of face, left eyelid and peri- orbital region. On examination, there was medial canthal ligament avulsion both superficial and deep heads with un-displaced upper eyelid but lower eyelid was displaced with extra marginal laceration. We treated him with broad spectrum antibiotics, anti-inflammatory, active immunisation against rabies with local infiltration of immunoglobulin. After thorough irrigation and debridement of wound, it was closed in layers. After 6 months follow up, he is doing well with mild telecanthus. In case of unknown animal/dog bite to orbital and periorbital area, wound management includes irrigation, debridement and primary wound closure if cornea is not protected and delayed or secondary wound closure if cornea is protected.

Introduction

Major part of our country includes rural and tribal area. Dog bite is very common but we rarely come across animal bite (wolves, foxes, coyotes, jackals) with periocular involvement. No or very few cases have been reported in Ophthalmology to best of our knowledge. In India, the animal bites are neither notifiable nor reported in routine surveillance system. There is always dilemma in management of such patients.

Case History

A 15 years old Male child presented to us from rural-tribal belt of Mangrol, District Surat, Gujarat with a history of unknown animal bite on left side of face, neck, left eyelid and peri- orbital region on previous night while sleeping outdoor. There was history of similar cases on that same night. He could not identify the animal but he was sure that it was not a dog. He had taken primary medical care at nearby community health centre including an injection Tetanus Toxoid 0.5 cc. and a dose of injection Ampicillin.

On examination, there was medial canthus avulsion both superficial and deep heads with- undisplaced upper eyelid but lower eyelid was displaced with an extra marginal laceration. There was associated upper and lower canalicular tear. There were scratch marks on neck with surrounding edema. Fortunately anterior segment was normal with only mild epithelial abrasion. Extraocular movement and dilated fundus were normal. Visual Acuity in OD and OS were 20/20 and 20/30 respectively.

We started intravenous antibiotics, Inj. Cefotaxime (50mg/kg/day), Inj. Lincomycin (20 mg/kg/day) and Inj. Metronidazole (30mg/kg/day), anti-inflammatory tab. Ibuprofen (30 mg/kg/day) and active immunisation against rabies (five doses

of Purified Chick Embryo Vaccine). Half of the Human Rabies Immunoglobulin (HRIG) in the dosage of 20 mg/kg was infiltrated around the wound and half of it was injected intramuscularly. After thorough irrigation and debridement of wound, it was closed in layers using 6-0 polyglactin for inner layers and 6-0 silk for skin. Due to an extensive tissue loss, we could not do stenting of canalicular system.

Discussion

The incidence of animal bites is 17.4 per 1000 population in India.[1] Most animal bites (91.5%) are by dogs, seconded by cat bite (4.7 %). Other animal bites comprise 3.8 %. A person is bitten every 2 seconds and someone dies from rabies every 30 minutes. The incidence of animal bites is more in rural areas (1.8%), children (2.6%) and low income groups (75%).[2] The data on animal bites in India is scanty, unreliable and controversial due to poor surveillance/reporting system. Orbital & Peri-Orbital injuries comprises 4-8 % of total Facial Animal Bites.[3-5]





Figure 2: Immediate Post Operative Photograph



Figure 3: One Year Post Operative Photograph

Principles of management

Immediate wound management includes Wound Irrigation & Debridement.[6-9] All animal bite wounds are presumed to be contaminated and therefore, must be decontaminated prior to surgical repair to limit infection. Forceful irrigation with at least 200 ml normal saline using a 35 ml syringe and 18 gauge cannula is recommended, while the cornea is protected with a Scleral contact lens.

Immunisation

Tetanus prophylaxis should be given to the patient who has never been immunized, administer 250 IU of intra muscular human tetanus immunoglobulin. Administer intramuscular or subcutaneous Tetanus Toxoid (0.5 ml), if the patient did not have tetanus immunization in the preceding 10 years. For unclean wounds or puncture wounds, administer Tetanus Toxoid for patients who have not had it within 5 years. Rabies Prophylaxis should be given in case of single or multiple transdermal bites or scratches by an animal who can not be quarantined for 10 days or an Animal with un-known Vaccination status.[6] Rabies Immunoglobulin provides passive protection to the individuals exposed to rabies virus. 20 I.U. per kg body wt. of HRIG or 40 I.U. per kg body wt.

of purified equine rabies immunoglobulin, irrespective of age, should be administered, approximately half the dose should be infiltrated into and around the bite wound as much as possible (given anatomical constraints) and the rest is given intramuscularly at a site remote from the vaccine administration area in the gluteal or deltoid muscle. Do not administer Rabies Immunoglobulin (RIG) if patient is previously vaccinated. At the same time also start the rabies vaccination schedule. [6] Rabies Virus Vaccine (Purified chick embryo cell Rabies vaccine) consists of inactivated forms of virus that promote immunity by inducing an active immune response. It should be administered intramuscularly in deltoid region for adults and older children. For younger children, use the outer aspect of thigh. [6]

Post exposure Prophylaxis

If patient is previously not vaccinated, total five doses of Intramuscular Inj. Each 1 ml on Days 0 (day of exposure), 3,7,14 and 30 for both adults & children, a booster dose on day 90 is optional. If the Patient is previously vaccinated, give only 1 ml intramuscular inj. on day 0 and 3 only. Stop treatment if animal remains healthy throughout an observation period of 10 days or if the animal is killed and found to be negative for rabies by appropriate laboratory techniques. [6]

Prevention of Infection

The oral flora of animal is responsible for infection in case of animal bite. Pasteurella multocida (20-30% of all infections, Common with infection <24 hours after bite), Streptococcus, Staphylococcus, Escherichia coli, Anaerobes (Bacteroides, Fusobacterium, Peptostreptococcus) and Capnocytophaga canimorsus (DF-2). Antibiotic (table 2) should be given for seven days for prophylaxis but it can be extended for 10-14 days if Cellulitis is Present. [10]

Surgical Care

Wounds less than 24 hours old and especially when there is no adequate corneal protection should be closed by sutures immediately. Puncture wounds, infected wounds and wounds older than 24 hours should be closed by secondary intention if there is adequate corneal protection. If patient has a ruptured globe and lid laceration, first repair the globe rupture. If the lid repair must be delayed ensure adequate corneal lubrication, clean the wound as much as possible and keep the wound moist. An occlusive dressing can be applied over antibiotic dressing to help protect the cornea. Whether primary or secondary, principles of lid repairs should be followed.[7-9]

Conclusion

Unknown Animal Bite to orbital and periorbital area should be considered as category III (severe) as far as rabies post exposure prophylaxis is concerned. If Rabies is suspected,

Table 1: Type of contact, exposure and WHO recommended post-exposure prophylaxis³

Category	Type of Contact	Type of Exposure	Recommended Post Exposure Prophylaxis
I	<ul style="list-style-type: none"> • Touching or feeling of animals • Licks on intact skin 	None	None, if reliable case history is available
II	<ul style="list-style-type: none"> • Nibbling of uncovered skin • Minor scratches or abrasions without bleeding 	Minor	<ul style="list-style-type: none"> • Wound management • Anti-rabies vaccine
III	<ul style="list-style-type: none"> • Single or multiple transdermal bites or scratches, licks on broken skin • Contamination of mucous membrane with saliva (i.e. licks) • Bite by all wild animals 	Severe	<ul style="list-style-type: none"> • Wound management • Rabies immunoglobulin • Anti-rabies vaccine

Table 2: Choice of Antibiotics

Adults	Children
1. Amoxicillin-Clavulanate 2. Alternative Antibiotics for Penicillin Allergic 1. Doxycycline (do not use in pregnancy) 2. Erythromycin (higher resistance rate) 3. Combination protocol 1. Clindamycin and 2. Fluoroquinolone 3. Alternative Antibiotics for questionable compliance- Ceftriaxone IM qid	1. Amoxicillin-Clavulanate 2. Alternative Antibiotics for Penicillin Allergic 1. Erythromycin (higher resistance rate) 2. Combination protocol a. Clindamycin and b. Trimethoprim-Sulfamethoxazole 3. Alternative Antibiotics for questionable compliance- Ceftriaxone IM qid

health department officials should be notified. The health department can assist in quarantine of the animal as well as offer advice about current recommendations for rabies prophylaxis.

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External Ophthalmomyiasis Caused by Sheep Botfly (*Oestrus Ovis*) Larva

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External Ophthalmomyiasis is the infestation of external ocular tissues (Eyelids, conjunctiva, lacrimal ducts) of man by fly larvae. Ophthalmic myiasis has been reported from various world regions. In this study we present the clinical manifestations with ophthalmomyiasis caused by first stage larvae of *Oestrus ovis* in 4 patients from rural areas during the period of January to April. All patients presented with symptoms like viral or allergic conjunctivitis after history of something falling in the eyes during outdoor work. Two of the patients were from Ahir caste (shepherds), in close contact with sheep and goat. The larvae were observed in the bulbar conjunctiva and following their removal, the symptoms of eye inflammation improved in a few hours/days with topical antibiotics and steroids eye drops. As the larvae are small, translucent and quickly avoiding the slit lamp beam, they can be easily overlooked on routine examination. We should suspect larval conjunctivitis in rural areas, especially during spring and summer, in patients presenting with viral or allergic conjunctivitis like picture following history of something falling into the eye.

Introduction

External ophthalmomyiasis is deposition of fly larvae confined to conjunctiva, eyelid, and lacrimal ducts. Ophthalmomyiasis not uncommon but underestimated in rural areas. Few cases have been published in the past but it is still frequently misdiagnosed as viral or allergic Conjunctivitis.

We reported clinical manifestations of ophthalmic myiasis in four patients. The clinical picture is that of a viral or allergic conjunctivitis with tearing, foreign body sensation and itching of the eye. The aim to report this case series is to create awareness about it amongst the ophthalmologist working in rural areas because larvae can be easily overlooked on routine examination as they are small, translucent and quickly avoid the slit lamp beam.

Case History

We observed four cases of external ophthalmomyiasis from Jan. 09 to April 09. Each patient was examined thoroughly and was asked, in detail, about the history and, in particular, symptoms related to the development of myiasis. Diagnosis of external ophthalmomyiasis was made by direct visualization of the larvae, using slit lamp. The major presenting symptoms were irritation, foreign body sensation, lacrimation, and redness. In each case, the symptoms began while the affected individual was working in a farm or travelling outdoors, during the days. None of the patients had history of allergic

reactions in the past. In all of case, the eye involvement was unilateral and extra ocular with motile larvae present in the bulbar conjunctiva. All patients were having injection of conjunctiva, lid edema but one patient also had chemosis. There was no evidence of corneal or intraocular involvement. Visual acuity was also normal in all patients.

After instillation of Proparacaine eye drop to paralyze the larvae and after photographic documentation, we removed the maggots with cotton tip applicator and the eyes washed out with normal saline. The number of larvae varied from three to six and measured 1 – 2 mm in length. They were mounted on a microscope slide, examined carefully, and photographed under a microscope and compared with *Oestrus Ovis* larvae in literature. The larvae were identified as first instars of *Oestrus ovis* (Diptera: Oestridae) which is a larviparous dipteran on the basis of their spindle shape, translucent, segmented body and two large dark oral books connected to a white cephalopharyngeal skeleton.

An antibiotic and steroid eye drops were started. Following removal of all larvae, the symptoms completely resolved within a few hours or days.

Discussion

Myiasis is the infestation of tissues and organs of animals or man by dipterous larvae. The most common site of infestation is the skin wound. Less common sites are eyes, nose, paranasal sinuses, throat, and urogenital tract.

Ophthalmic myiasis is due to deposition of fly larvae in the human eye. Various species of flies are able to provoke ophthalmomyiasis, including *Oestrus ovis* (sheep nasal botfly), latrine fly (*Fannia*), house fly (*Musca domestica*), and cattle botfly (*Hypoderma*). *Oestrus ovis* is by far the most common (80-90 %) cause of ophthalmic myiasis in human.[3] Ophthalmic myiasis due to *Oestrus ovis* was described for the first time in 1947 by James.[4] More scattered cases have been reported since then from Mediterranean area, like Italy, and also from Russia, Serbia (previous Yugoslavia), India, [9-10]Africa [6-7], America, Oman and Iran [1-5]. Ophthalmomyiasis is more common than what have been indicated by previously published reports. Ophthalmomyiasis mostly occurs in rural areas, where man lives in close contact with cattle. An interesting feature of *O. ovis* is that it can deposit larvae while still in flight. The fly darts close to the eyes or nostrils and ejects a stream of the first-instar larvae, which have previously hatched from the eggs in the fly vagina, into the target area. The sheep and goat are the main hosts for myiasis by *Oestrus ovis* and the men are infested accidentally. This eye involvement by *Oestrus ovis* is in the form of external ophthalmomyiasis, which is confined to conjunctiva, eyelid, and lacrimal ducts, as first instar larvae have no bite organs and are unable to secrete proteolytic enzymes. [6-7] In humans, *O. ovis* larvae generally do not develop past the first instars stage, although other species may grow much larger. They are capable of living in eye fluid, freely crawling on the eye balls with the help of anterior hooks and cause irritation by their curved mandibular barbs and body spines.

Grammer et al. summarized numerous cases of external ophthalmomyiasis due to *Oestrus ovis*. [3]

Masoodi et al described few cases of external ophthalmomyiasis caused by oestrous ovis in farmers in close contact with sheep and goat. [1] Misra et al described external ophthalmomyiasis due to *Oestrus ovis* as endemic disease in rural central India. [9] Narayanan et al documented few cases of ophthalmomyiasis due to *Oestrus ovis* in southern part of India in 1991. [10]

Symptoms, such as severe eye irritation, redness, foreign body sensation, pain, lacrimation, and swelling of the lids, season of occurrence, and also predominance of young male patients presented in our case series are similar to those described in other reports. [1-3],[9-10] All of the 4 cases lived in rural areas, farmers and two of them belong to Ahir caste live in close contact with sheep and goats. Complications such as corneal ulcer, invasion into eye globe, and decreased vision are not usual and none of these complications were encountered in our patients. But the larvae from some other species such as *Hypoderma* [1] or *Chrysomya bezziana* [8] can penetrate the eye globe and cause endophthalmitis and iridocyclitis, and may even lead to blindness.

Our experience showed that larvae quickly die and dry out once removed from the eye. Removed larvae should be preserved

in 70% alcohol and sent to specialists for examination. We could not immediately find specialists to help us preserving the species. We therefore tempted to identify the larva as *Oestrus ovis* based on microscopic appearance (translucent body, segmentation, large dark oral hooks connected to a white cephalopharyngeal skeleton) and extended study of the literature [1-3],[7],[9-10]

The treatment consists of anesthetizing the larvae and conjunctival sac and mechanical removal of the larvae. Topical steroids relieve symptoms. Topical antibiotics are useful in preventing secondary bacterial infection. Some people advocate hypertonic saline ointment in order to kill possibly remaining maggots by dehydration. Follow up examination by an ophthalmologist is recommended to avoid the rare possible complication of internal ophthalmomyiasis by other species.

External Ophthalmomyiasis should be considered as an occupational disease among farmers and shepherds. As the larvae are small, translucent and quickly avoiding the slit lamp beam, they can be easily overlooked on routine examination. Awareness of the larval conjunctivitis in rural areas, especially during spring and summer, leads to the more prompt diagnosis, and institution of specific therapy for the disease. Identification of the species is important to estimate the risk of penetration of the globe.



Figure 1 Larva of *Oestrus ovis* on lid margin



Figure 2 Larva of *Oestrus ovis*.



Figure 3 Larva of *Oestrus ovis*, Conjunctival Sac



Figure 4 Larva of *Oestrus ovis*, 100X- Under Laboratory Microscope

Table 1 Clinical findings in patients of Ophthalmomyiasis

Sr. No.	Age	Sex	Caste	Date of Presentation	Place of Occurrences	No. of Larvae recovered
1.	30	Male	Vasava	10.01.09	Working in the field	6
2.	35	Male	Vasava	17.03.09	Traveling on a bike	4
3.	25	Female	Ahir	24.03.09	Working in the field	4
4.	60	Male	Ahir	28.04.09	While Grazing Goats/Sheeps in the field	3

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Cases Reports

Bietti's Crystalline Dystrophy

Amar Dev, Snigdha Sen, Shalini Wadhwa

Introduction

BCD is a rare tapetoretinal degeneration combined with a marginal corneal dystrophy was first described by Bietti in 1937. The fundus characterised by numerous polygonal yellowish white glistening crystal deposits in all layers of retina. It is associated with varying degrees of choriocapillaries and RPE Loss. In some cases it is associated with superficial limbal corneal deposits.

Case History

A twenty year old young female presented with mild diminution of vision. Her history was unremarkable. There was no family history of any ocular disorder. Dietary habits were normal and she was not taking any regular medication. There was no history suggestive of night blindness. Her visual acuity was 6/6 in right eye and 6/9 P in left eye. Her BCVA was 6/6 right eye and 6/6 left eye with -0.75 cylinder at 170 degree.

The anterior segment was normal in both eyes. IOP was 17.3 mmHg in both eyes.

Fundus examination in both eyes showed numerous polygonal yellowish white glistening crystal deposits in all layers of retina especially at posterior poles, some lying in front of blood vessels. Peripheral fundus showed some pigmentary disturbances. Her visual field showed concentric contraction however colour vision was normal (Ishihara).

Fluorescein angiography showed atrophy of RPE and choriocapillaries at posterior poles however retinal vasculature was normal. The crystalline deposits did not fluoresce.

Both ERG and EOG were significantly reduced. A search of underline metabolic disorder was unrewarding, as the result of investigations viz: plasma urea, electrolytes, creatinine, plasma & urine amino acids; plasma triglycerides and cholesterol were normal. The twenty four hours urine oxalate excretion was low. A number of lysosomal enzymes involved in glycolipid and oligosaccharide metabolism were assayed in leucocyte and skin fibroblast and no abnormality was found.

Discussion

The clinical picture of marginal corneal dystrophy and crystalline retinopathy provides little latitude with regards to differential diagnosis as few other conditions show refractile bodies in both cornea and retina. The crystals of cystinosis are found throughout cornea and appear in choroid and RPE but not in neuro-retina. Corneal abnormality has not been noted in cases of crystalline retinopathy of known case but they may have been overlooked.

White retinal flakes may have been seen in patients of

primary and secondary oxalosis (prolonged methoxyfluorane anaesthesia). Refractile bodies may be found in cases of gyrate atrophy, Sjogren-Larsson syndrome and in patients treated with tamoxifen but underlying conditions are evident from clinical history and examination.

There are considerable differences in the degree of retinal dysfunction in reported cases of Bietti's disease, partly due to the progressive nature of disease but as more cases are reported the parameters of clinical severity may become more evident. Long term follow up of only two cases were reported. Both had suffered a marked decline in central vision and contraction of visual field. The chorioretinal degeneration had advanced, though the number of crystals had decreased as they are encompassed by the areas of choriocapillaries atrophy.

There insufficient cases of Bietti's disease to establish a pattern of inheritance, if any, but the presence of two siblings and the child of a consanguineous marriage among reported cases suggest an AR disorder. Five cases of crystalline retinopathy similar to Bietti's disease but without corneal dystrophy have been reported. The presence of sibships and consanguinity within this group suggest AR disease, but at present we do not know whether these cases share a common aetiology with Bietti's disease or merely show a clinical similarity.

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Father of Macular Diseases: Professor J Donald M Gass, MD

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Professor John Donald MacIntyre Gass, MD

John Donald MacIntyre Gass MD Professor of Ophthalmology and Visual Sciences, Emeritus, one of the world's most respected clinician in the field of retinal, macular and uveal diseases was one of the most significant figures to emerge in Ophthalmology in the last one hundred years. There could be few ophthalmologists who cannot attribute part of their increase in understanding of retinal disease to the influence of Professor Don Gass. His insights opened up opportunities for many new effective therapies. He has influenced ophthalmic thought world wide, if not by his presence as a visitor, then through his scientific publications, his outstanding books, and the international fellows he trained. Like many distinguished physicians, Professor Don Gass clinical acumen was well grounded in his understanding of ocular pathology. This experience was gained under the mentorship of Lorenz E Zimmerman, MD, who trained a number of distinguished ophthalmologists, who subsequently became professors. This article reflects further on Don Gass' life and contributions of in the field of ophthalmology and visual sciences, especially in the area of retinal and macular diseases.

Education:

Professor Gass was born in Prince Edward Island, Canada and raised in Nashville, Tennessee, USA. If the story of Don's entry in to medicine be true, his passion for medicine was acquired not inherited. The story was told that as a young student he was standing in a queue to enroll for engineering. He became fatigued with the length of waiting in the long queue and impulsively moved in to a shorter queue of students who sought to join the faculty of medicine. The rest is history.

Professor Gass received both his undergraduate (1950) and medical (1957) degrees from Vanderbilt University where in medical school he was awarded the Founder's Medal for the highest

academic achievement.[1] Upon completion of his undergraduate studies, he married Margy Ann Loser. He served on active duty as a line officer in the United States Navy from 1950 to 1953 during the Korean War. After medical school, he served an internship at the University of Iowa, before his residency in Ophthalmology at the Wilmer Institute at Johns Hopkins Hospital. In 1962, he took a fellowship at the Armed Forces Institute of Pathology, Washington, DC, under the mentorship of Lorenz E. Zimmerman, MD. This experience in Ocular Pathology was reflected in all of his subsequent publications. He never ceased trying to confirm the pathological basis adding to pathological examples of the clinical cases, he so carefully chronicled.

In 1963, Professor Don Gass joined the faculty of the newly formed Bascom Palmer Eye Institute of the University Of Miami School Of Medicine. His understanding and contributions to fluorescein angiography were important in his demonstration of clinico pathological correlation with disorders of the macular and retinal vasculature. His distinguished career at Bascom Palmer Eye Institute spanned more than 3 decades (1963 to 1995). He combined patient care with clinical research and teaching and made his major contributions to ophthalmology. His scholarship and intellect and clinical insight, combined with deep friendship and loyalty to Edward WD Norton, MD, and his colleagues is part of a highpoint in the history of Bascom Palmer Eye Institute. He saw chairmanship as a distraction and never sought this distinction. In recognition of his expertise, Professor Don Gass was appointed an Associate Examiner of the American Board of Ophthalmology in 1968 and in 1976 he was elected to the Board of Directors. In 1983 he was chosen to chair the Board. His service to the American Board of Ophthalmology in all spanned 15 years.

There is a sense of symmetry in the personal and professional

life of Professor Don Gass in that in 1995, after spending so many productive years in the Bascom Palmer Eye Institute, he finally retired from Bascom Palmer Eye Institute and returned to Nashville, joining the academic staff of Ophthalmology Department of the Vanderbilt University School of Medicine, chaired by Denis M. O'Day, MD adding a further Australian dimension to our association with Don Gass. His return to the Vanderbilt University School of Medicine in Nashville (1995-2005) was soon followed by national and international students, fellows, and visiting ophthalmologists finding their way to his new address.

Honors, Awards and International Recognition:

Professor Don Gass never sought the limelight though his contributions to ophthalmology have benefited millions of people throughout the world. A prolific writer, he authored several major reference books on macular diseases and intraocular tumors. His classic books, *Stereoscopic Atlas of Macular Diseases a Fundoscopic and Angiographic Presentation*, and *Differential Diagnosis of Intraocular Tumors: a Stereoscopic Presentation*, are authoritative texts in the field. In addition, Professor Don Gass authored 12 book chapters and over 270 articles in peer-reviewed journals, many of which are considered landmark contributions.[2-25]

Professor Gass was recognized as the premier medical retina specialist in the world. In 1999, at age of 70, he was identified by his peers as one of the top ten most influential ophthalmologists of the 20th century. The designation came through a poll of nearly 33,000 ophthalmologists around the world, conducted by the American Society of Cataract and Refractive Surgery (ASCRS) in Seattle, Washington in 1999. One of us (SKP) was fortunate to meet Professor Gass during this event in Seattle (USA). He was also the recipient of the Mildred Weisenfeld Award for Excellence in Ophthalmology from the Association for Research in Vision and Ophthalmology (ARVO) in Fort Lauderdale, Florida. Don Gass was the recipient of the Distinguished Faculty Scholar Award at the University of Miami in 1989. Bascom Palmer also honored Don Gass with an endowed chair.

Recognized as the Father of Macular Diseases, Professor Don Gass was singled out and honored for his outstanding achievement by almost every major professional organization in ophthalmology and/or academic institutions throughout United States and abroad. He delivered a number of prestigious lectures including the XXXIII Edward Jackson Memorial Lecture. His awards included Helen Keller Prize for Vision Research in 2001 and the Laureate Recognition Award of the American Academy of Ophthalmology at its 108th Annual Meeting in the year 2004 at New Orleans, Louisiana. The Macula Society lectureship and medal for his outstanding contribution in macular disease were established in his honor. Most recently, Vanderbilt University

honored Professor Don Gass endowing a chair and honor lecture in his name. A portrait of Professor Don Gass was also unveiled at Vanderbilt University School of Medicine in his honor. In 2004 his Alma Mater, in yet another honor, recognized him as a Distinguished Alumnus.

Professor Don Gass remained a modest, humble individual who was uncomfortable with public recognition and indeed seemed genuinely unaware of his stature in the profession. At the time of his retirement from the Bascom Palmer Eye Institute he contacted the Chair of the Department of Ophthalmology and Visual Sciences at Vanderbilt University to let him know of his plans to return to Nashville. Professor Gass explained that he would like to continue working part time and had thought of joining a group of ophthalmologists in private practice but wondered if perhaps a niche could be found for him at Vanderbilt. Needless to say that niche was quickly identified!

Over the next eight years of this partial retirement, he continued his research while maintaining a clinical practice and teaching. It was during this period of his work he completed a revision for the fourth edition of his famous Atlas²⁰ and published seminal papers on AZOOR Complex^[3,11,12,19] and the pathophysiology of macular holes.^[2,14,23] In those years alone, his research resulted in more than fifty scientific publications.

In addition to the patients he saw at the Bascom Palmer Eye Institute and Vanderbilt University, Professor Don Gass had a worldwide consulting practice. The arrival of packages of fundus photographs and angiograms always initiated a period of intense activity. People would gather in his office as he began to examine the photographs. This image of a man sitting, surrounded by colleagues, residents, students and fellows is one that is very familiar to those who knew him well. While Professor Don Gass gave innumerable lectures in his lifetime, many of them named and prestigious, he seemed more comfortable teaching in this informal setting.

Professor Don Gass was known for his contributions to an understanding of retinal disease, but his intellectual reach was far broader. An example was his active interest in ocular tumors. At Grand Rounds he could contribute on most topics, sometimes to the discomfort of experts in the field. Always though, his innate gentleness and humanity shone through.

Ophthalmology was his driving passion, but Professor Gass had other talents. An accomplished craftsman, his workshop in his home held pride of place. It was there that he would spend many hours engaged in projects that ranged from building model trains and scale models of sailing ships to full size furniture. What linked these two loves of his was a consummate attention to detail. In the last months of his life, despite his advancing illness, Professor Gass

made a special effort to attend retinal conferences at Vanderbilt. He could also sometimes be spotted at a local hardware store or timber merchant.

Family Life: Professor Gass' original contribution was prodigious but it had the underlying support and strength that comes from his family and being happily married to his wife Margy Ann, for over 55 years. The marriage was blessed with four children, and five grandchildren.

Conclusion:

Professor Gass' contributions to ophthalmology are his legacy. He remains part of the great tradition of observant clinician with outstanding clinical skills matched by deep understanding of the associated pathology. He stands well in the company of a long line of distinguished international ophthalmologists with this background, who shared with him the importance of grasping this connection.

Professor John Donald MacIntyre Gass, MD passed away on February 26, 2005 at the age of 76 years from pancreatic carcinoma. With the demise of Professor Don Gass, the world of ophthalmology has lost an extraordinary physician of great talent, commonsense and humility. On the other hand it has gained a generation of young ophthalmologists inspired by his example.

Professor Don Gass' passing should not be seen as the end of an era but a promise for a generation of young ophthalmologists to become ophthalmic physicians inspired by his example to seeking to understand their patients and to unravel the causes of blindness, for which as yet there are no answers; a generation of young ophthalmologists who see in Professor Don Gass' example how much can be contributed to the community with the understanding of ophthalmology in the quiet and contemplative clinical setting of the caring, wise and perceptive physician.

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Arthur L Rosenbaum, MD (1940-2010)

Ramesh Kekunnaya

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We all lost a shining star when Arthur L. Rosenbaum, passed away on June 22, 2010, after a difficult illness. Dr Rosenbaum was the Professor Pediatric Ophthalmology, Vice Chair of the Department of Ophthalmology, and Chief of the Division of Pediatric Ophthalmology and Strabismus at the University of California, Los Angeles (UCLA). Personally I had the great privilege to work with him as a fellow.

Dr Rosenbaum was the recipient of numerous awards and honors from both the American Academy of Ophthalmology and the American Association for Pediatric Ophthalmology and Strabismus. He has many publications to his credit and had one of the best academic positions at United States. He has written a fantastic textbook on Clinical Strabismus Management, which is well known all over the world.

His clinical practice at UCLA included the most complex and challenging cases and he was managing these patients very elegantly, providing them with the best treatment possible. His way of discussions in the clinics, operating rooms as well as in the conferences were, amazing.

He had all the qualities of a great teacher, thinker, and above all was a true leader. Besides being an excellent surgeon, he had a 'never say die' attitude, which was seen even at the times of his illness. This was a great inspiration to everyone who had the good fortune of crossing paths with him, me included.

We will profoundly miss a legendary Pediatric Ophthalmologist who has contributed a lot to our field. May his soul rest in peace.

Synoptophore

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Synoptophore is comprehensive orthoptic unit which helps in complete evaluation of a case of strabismus. It can be used diagnostically to measure the amount of deviation in all three axes and to assess binocular status of the patient. Therapeutically it can be used to provide fusional exercises, treat eccentric fixation and abnormal retinal correspondence.

Synoptophore is an instrument commonly used in orthoptics and strabismus and to assess the level of binocular interaction. It is based on the haploscopic principle which uses angled mirror to dissociate the two eyes so that right eye sees the right temporal field while left eye sees the left temporal field.

The instrument consists of two optical tubes with a mirrored right angled bend, each of which projects an image into either eye. On the outer side of each tube there is a low intensity light source for the illumination of the slides placed in the slide carrier. At the other end of the tubes which project the image into the eyes, there is a +6.5 D lens to present a parallel beam of light so that the image appears to project from infinity. This simulates distance fixation and ensures relaxation of accommodation. Although this does ensure relaxed accommodation, and prevent difference of measurement from the Prism Bar tests, some differences occur due to proximal vergences which can affect the measurements. The distance between the two tubes is adjusted to line the center of the eyepieces accurately to the patients IPD. The tubes can be rotated horizontally as well as vertically to neutralize deviation in both the axis, while torsional deviation can be measured by tilting the slide carrier. The exact amount of rotation of the tubes performed is measured in degrees and prism diopters on a scale (Figure 1a-c)

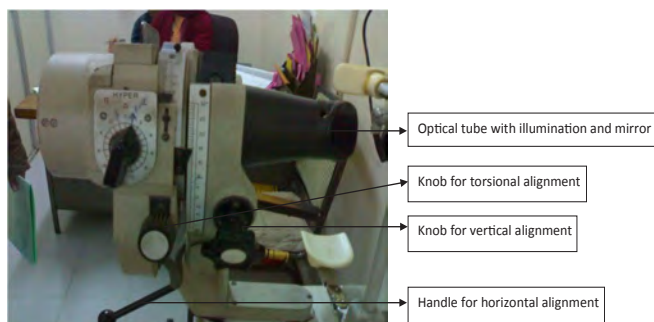


Figure 1c: Synoptophore

There are different series of slides available that are used for variety of purposes.

- Simultaneous macular perception (SMP) slides (Figure 2): These slides consist of two dissimilar objects, subtending an

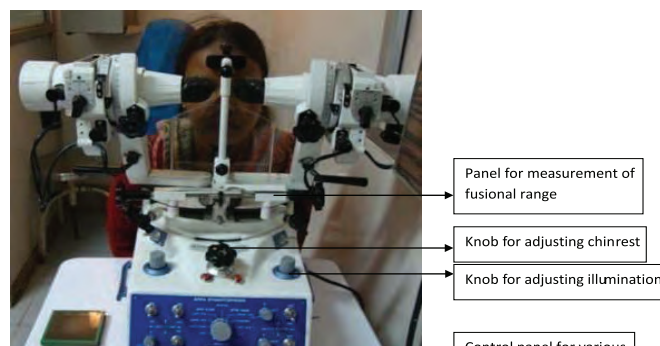


Figure 1a: Synoptophore

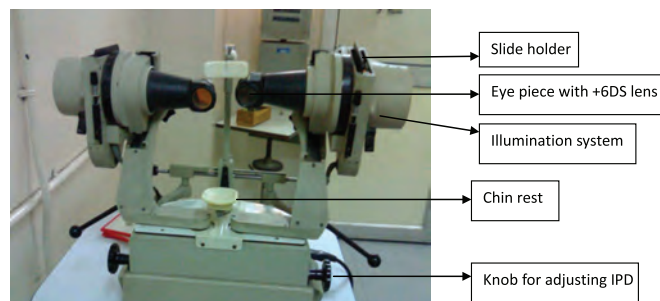


Figure 1b: Synoptophore

angle of 5 degrees at the macula. One solid and one hollow object, like lion and cage is projected in either eye. If both the images superimpose and patient perceives lion in cage indicates simultaneous perception. Absence of either target suggests suppression of that eye. Sometimes a small central scotoma can be missed in the eye with the cage in front of that eye so it is advisable to show the slides alternately in both the eyes.

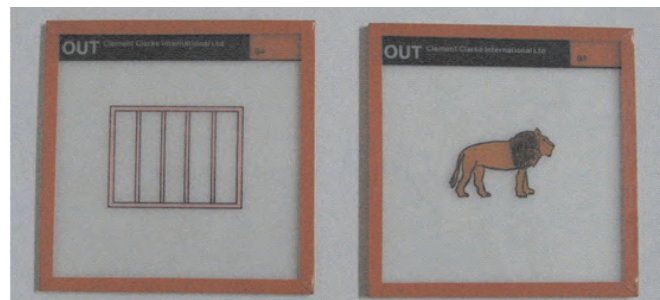


Figure 2: SMP slides with red binding

- Fusion: the targets used are same in all respect except for some part either missing in one slide or addition in the other one (Figure 3). The patient should be able to fuse the two targets sees both the target as one complete figure. Like in the figure the cat has a tail in one slide and a butterfly in the other. If fusion is present the patient will report one cat with a tail and a butterfly. Suppression in any eye can be easily recognized by the examiner as the patient will not see either the tail or the butterfly depending on the eye suppressed.

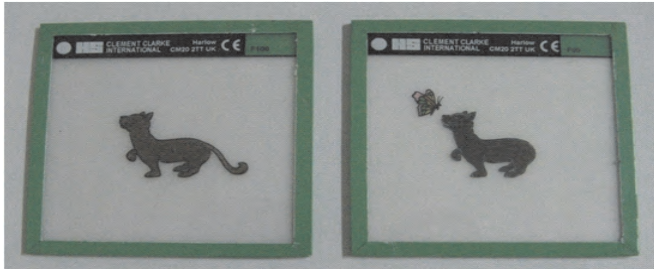


Figure 3: Fusion slides with green binding

- Stereopsis: these slides consist of two similar slides with part of the slide having horizontal disparity which gives depth perception when seen as one. (Figure 4) In the figure the gate overlaps in both the slides but the train, bird and the signal are horizontally displaced relative to each other and so give rise to a feeling of relative depth.

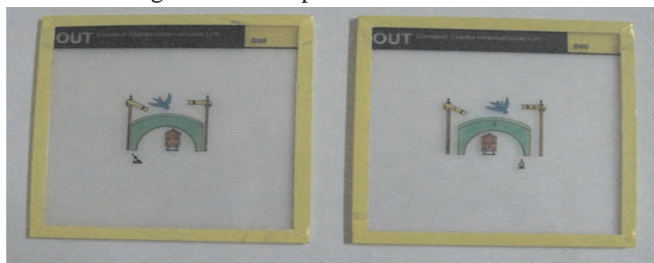


Figure 4: Stereopsis slides with yellow binding

- After image slides (Figure 5) this is the most dissociating test in which battery- powered camera flash is used to produce a vertical after image in right eye and a horizontal after image in the left eye. In the treatment of abnormal retinal correspondence it is necessary to maintain the after images for a period of time by an alternating light and dark background. This is provided by an alternating flashing device incorporated in the synoptophore that alternately illuminates each of the optical tube.



Figure 5: After Image slides with blue binding

Uses

- To measurement of the objective and subjective angle of deviation.
- Measurement of angle kappa.
- To test for the presence and type of suppression.
- To test for the presence of fusion and measurement of fusional amplitudes
- Measurement of interpupillary distance (IPD)
- Measurement of the deviation in all 9 gazes, particularly useful for incomitant squint in which the primary and secondary deviation can be separately measured
- Evaluation of binocular status, Abnormal Retinal correspondence (ARC), suppression and to certain extent presence of gross stereopsis
- Measurement of range of fusion and is used for providing fusional vergence exercise
- Treatment of ARC

Technique of examination:

- Patient is asked to seat with his chin resting on the chin rest. The chin rest is adjusted to bring his at the level of the eye piece.
- Before proceeding for the further examination is important to adjust the IPD of the patient. The IPD is measured by aligning the fixing eye with the mark present on the eye piece. The measurement is done for each eye independently.

Measurement of deviation:

For measurement of angle of deviation SMP slides are used. For determination of subjective angle of deviation the patient is asked to superimpose the two images by moving the optical tube. For objective assessment the examiner uses flash. Either eye is allowed to take fixation by illuminating one slide at a time and the movement of fixing eye is noticed. The optical tubes are adjusted horizontally and vertically depending upon the type of deviation till the re-fixation movement is neutralized.

Measurement of fusional range:

The fusion slides are placed in the slide holder after the neutralization of deviation is done. For determining convergence fusion the two slides are moved out till the patient complains of diplopia or deviation of the eye is observed and for divergence fusion the slides are moved in.

Each eye is stimulated separately and patient is asked draw the relative position of the lines.

Determination of ARC

SMP slides are used and any deviation if present should be corrected so that both the images fall on fovea. If patient can perceive both the images in presence of manifest squint it is suggestive of ARC.

- o If Subjective Angle = Objective Angle • NRC
- o If Subjective Angle < Objective Angle • ARC
- o If Angle of Anomaly (objective angle – subjective angle) = Objective Angle • Harmonious ARC (full sensory adaptation)
- o If Angle of Anomaly < Objective Angle • Unharmonious ARC

Hering Bielschowsky After Image test:

It is a highly dissociating test in which fovea of the two eyes is stimulated separately with a bright light. A horizontal image is projected in front of right eye while a vertical image is projected in front of the left eye, and the patient is asked to appreciate the after images.

- o A cross response at center indicates normal retinal correspondence.
- o An asymmetrical cross response is suggestive of ARC.
- o Absence of vertical line and horizontal line indicates suppression of right and left eye respectively.

Haidinger brush

Haidinger's brush is an entoptic phenomenon appreciated by macula, first described by Austrian physicist Wilhelm Karl von Haidinger in 1844. This property of macula can be used for treatment of eccentric fixation. A rotating polarized plate backlit with a bright white light is projected in front of the eccentric eye while the normal eye is occluded. The patient appreciates the Haidinger's brush with his macula and then looks at the test object in such a way that the Haidinger's brush overlaps the test object.

The synoptophore is a very useful instrument for the strabismologist and is an integral part of his clinic. Even for the general ophthalmologist cases of muscular asthenopia form a significant number and optimal use of the synoptophore can provide significant relief to these cases.

Orbital cellulitis in children

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Purpose: To review cases and make an effort to explore changes in diagnostic and management trends in children with orbital cellulitis. **Methods:** Noncomparative, retrospective case series. Medical records of children <18 years old with radiological and clinical confirmation of orbital cellulitis with postseptal inflammation were assessed. **Results:** Nine pediatric patients were recognized for the study. The mean age was 5.5 yrs. 7 patients were males and 6 had right orbital involvement. All the patients had chemosis, congestion, lid swelling, proptosis, restriction of movement. Most common predisposing event was sinusitis in 4 patients. 4 had orbital abscess. CT scan was the most comprehensive imaging technique in patients with suspected orbital infection. 4 patients had surgical intervention for drainage of pus and 3 underwent endoscopic sinus surgery for drainage of sinuses. Visual acuity improved in 6 cases. The average hospital stay was 18 days. The mean durations of total and parenteral antibiotic therapy were 21.5 days and 10.2 days, respectively. **Conclusion:** Delay in detection and intervention in most cases of orbital cellulitis can lead to serious complications in this uncommon but not rare disease.

Introduction:

Orbital cellulitis is a potentially lethal disease that can result in significant complications, including blindness, cavernous sinus thrombosis, meningitis, subdural empyema, and brain abscess. Prior to the discovery of antibiotics the mortality rate was 20–40%. These complications though have become rare in the antibiotic era, but the potential for significant morbidity, sight threatening complications and mortality makes prompt diagnosis and early treatment very important.

In light of our increased knowledge of orbital cellulitis and sinusitis, improved diagnostic tools, and new pharmacologic agents we make an effort to explore changes in diagnostic and management trends in children with orbital cellulitis. We report our experience with 9 children treated within a 2-year period at our oculoplastic clinic of a tertiary referral centre.

Material and Methods

Medical records of children <18 years old with orbital cellulitis who attended our oculoplastic clinic between a period April 2008 to March 2010 were identified for review. Cases were included if they had radiological and clinical confirmation of orbital cellulitis with postseptal inflammation. Cases with only preseptal (periorbital) cellulitis were excluded. Children with orbital cellulitis secondary to malignancy, fungal infections, or other immunosuppressed states were not included in the study. In our retrospective study data regarding clinical features, and details of treatment, microbiologic and radiologic characteristics, complications, and follow-up were obtained. Duration of hospital stay and antibiotic therapy were noted. CTscan findings were obtained so as to examine in detail the

nature of orbital, sinus and brain involvement.

The patients were treated with a wide range of antibiotics parenterally in combination dosage. Surgical drainage was done in cases of orbital abscess after confirmatory CTscan report. All the data was followed up till complete resolution of signs and symptoms.

Nine pediatric patients with orbital cellulitis were recognized for the study. Two were females and rest were males. Six had right sided involvement and three had left sided involvement. The ages ranged from 9 months to 16 years, with a mean age of 5.5 yrs.

Signs and symptoms at the time of presentation were present in less than 7 days in six patients and were present from one to four weeks in three patients. All the patients had chemosis, congestion, lid swelling, proptosis. Displacement of eyeball was present in five patients. Restriction of eye movements were present in nine patients which was marked in all directions in three patients. Four patients had decrease in visual acuity one line or more in the involved eye. Severe lid edema precluded visual assessment in two patients. In two infant patients vision assessment was not possible. One patient had a pthisical eye.

Only three patients showed continuous fever when first presented which responded to antipyretics and antibiotics. One patient developed fever on 7 day of admission which was diagnosed to be malarial in origin by paediatrician and was managed accordingly. The total leucocyte count was more than 10,000/cubic mm in three patients. One patient had 25–30 pus cells in urine.

Amongst radiological investigations CT scan was done in

Case	Age /Sex	Diagnosis and settings	Radiological Investigation	Management	Culture
1	16 yrs /M	®orbital cellulitis with abscess	Self ruptured on skin with antibiotics and FESS	Orbital cellulitis with lateral rectus abscess with sinusitis	
2	2 yrs/M	®orbital cellulitis with subperiosteal abscess	Drainage with antibiotics	Well defined approx. 27x17mm mixed density lesion in inferolateral orbit extending in retrobulbar area	Gram +ve cocci
3	9 mth/F	(L) panophthalmitis leading to orbital cellulitis	Intravitral repeated doses with antibiotics	USG exudates in vitreous, choroidal thickening	
4	9 yrs/M	® post traumatic orbital cellulitis	Antibiotics ,orbital drainage	Orbital cellulitis with air foci and sinusitis	
5	7 yrs/ M	®panophthalmitis leading to orbital cellulitis	Intravitral repeated doses with antibiotics	USG exudates in vitreous, sclero-choroidal thickening	Gram +ve cocci
6	6 yrs/M	(L)orbital cellulitis with subperiosteal abscess	Drainage with antibiotics	Well defined mixed density lesion in inferomedial orbit extending in retrobulbar area	Gram +ve cocci
7	4 yrs/M	®orbital cellulitis with orbital abscess	Drainage with antibiotics	Temporal wall abscess with cellulitis with clear sinuses	Gram +ve cocci
8	3yrs/M	®orbital cellulitis	Antibiotics and FESS	Orbital cellulitis with sinusitis	
9	10 yr/M	(L)orbital cellulitis	Antibiotics and FESS	Orbital cellulitis with sinusitis	

seven patients, all had evidence of orbital cellulitis. Four patients had sinusitis of maxillary, ethmoid and frontal sinuses, alone or in combination. Four patients had an abscess wherein the pus was drained surgically. BScan showed evidence of vitreous exudates with sclerochoroidal thickening in two patients.

Specimen were obtained from conjunctiva and aspirated material and were sent to the ocular pathology section. Gram positive cocci were the most common organism seen. In four patients culture reports showed the predominant organism to be staphylococcus aureus, streptococcus pneumoniae.

Predisposing factors include sinusitis in five patients, trauma to lid in one case, dental infection in one case, panophthalmitis in two cases.

Intravenous antibiotics with broad spectrum coverage were given to all patients. Four patients were already on topical and or systemic antibiotics at time of presentation. Anaerobic coverage was also given to patient with dental infection. Despite self rupture of an abscess on skin the lesion healed over a course of time with regular povidine iodine dressing and no sinus tract was formed. Four patients had surgical intervention for drainage of pus and three patients underwent functional endoscopic sinus surgery for drainage of sinuses after the acute stage subsided. The average hospital stay was 18 days. The mean durations of total and parenteral antibiotic therapy were 21.5 days and 10.2 days, respectively.

Amongst long term complications two patients developed restriction of movement. One patient had residual RAPD and three had marked visual deficit. One patient had mild motility disorder. Six patients had excellent visual recovery, no residual proptosis or neurological deficits. There was no mortality in this case series.

Discussion

Infections within the orbit itself is a serious problem which should be treated swiftly and aggressively.[1] When orbital cellulitis is adequately treated, it resolves without complications, almost always the outcome in children. In adults orbital cellulitis is uncommon, though complications are frequently seen. [2]

The striking male preponderance that we observed in this case series, has also been seen in most other case series that provided gender-specific data.[3,-6] Overall, the male:female ratio across these case series suggest that orbital cellulitis in childhood is at least twice as common among males as females. This is consistent with gender-related trends in other serious infections in childhood.

In our series of patients fever and systemic signs were present in only three patients. Laboratory data like total leucocyte counts were often normal. It is important that clinician should be aware of the gravity of situation and should not delay in starting the treatment due to the absence of systemic signs and laboratory evidence of cellulitis. Proptosis and restriction of ocular movements were seen in almost all patients but visual recovery was excellent in six patients. The patients with marked visual deficit had panophthalmitis.

The most common organism isolated were gram positive cocci in conjunctival swabs. In four patients culture reports showed the predominant organism to be staphylococcus aureus, streptococcus pneumoniae. This is similar to another study by Kineley et al where staphylococcus aureus, streptococcus pneumoniae, hemophilus influenza were the most common isolates in paediatric age group. [7, 8]

Radiographs and CT scan of patients show presence of sinusitis in five patients making it as the most common predisposing

factor for orbital cellulitis. Ethmoid and maxillary sinus were most commonly involved. Orbital infection has long been the most common complication of sinusitis.[9] Schramm et al. noted 74% clinical and radiological evidence of sinusitis.[10] Bergin D et al noted radiological evidence of sinus disease in 64% patients.[11] The other predisposing factors in our series were panophthalmitis, trauma, dental infection. CT scan is useful in differentiating between orbital cellulitis and preseptal cellulitis. It also remains the most practical means to investigate an orbital abscess.[12] The CT scan findings tend to lag behind the clinical exam. Thus a repeat CT scan within a few days of starting antibiotics is not as relevant as clinical examination.[13]

The most common antibiotic used empirically was combination of amoxicillin clavunate (50-100mg/kg/day, 8hrly) with amikamycin (15mg/kg/day, 12hrly) or cefotaxim (100-150mg/kg/day, 12hrly) with ampicillin (150mg/kg/day, 6hrly) in appropriate dosage. Metrogyl was added in the patient which had dental infection.[14]

Almost all organisms in this case series were sensitive to one of the drugs used. This regimen may require reevaluation because of significant prevalence of community-acquired methicillin-resistant *S. aureus* infections. After the resolution of acute phase endoscopic sinus surgery was done in three patients by otolaryngologist. Endonasal endoscopic surgery also now offers a safe approach to the drainage of the subperiosteal abscess and quick rehabilitation with no external scar.[15-17] Once the diagnosis of orbital abscess is established it should be drained immediately and adequately.[18,19] Canthotomy and cantholysis should be performed on an emergency basis if an orbital compartment syndrome is diagnosed at any point in the course of the disease.

After reviewing our records we would like to stress on the fact that in cases of orbital abscess a very carefully done surgical drainage procedure, the site of drainage of pus either by skin or transconjunctival or endoscopic approach along with antibiotic coverage markedly speeds up the recovery. Often a combined ophthalmological and otolaryngological approach is required to establish drainage of both the sinus and orbit.[11] Our local study is limited by its retrospective design and relatively small number of cases. In the literature review, a number of studies had to be excluded because of confusion in terminology or the lack of clear differentiation between orbital and periorbital cellulitis cases.

Orbital cellulitis can be sight threatening and on occasion a life threatening event.[20-22] Management of orbital cellulitis is now becoming a multidisciplinary approach. CT scan with contrast is now the most comprehensive imaging technique in patients with suspected orbital infection.[23] An aggressive and timely interventional management of these paediatric patients in this antibiotic era is not just life saving but also motility and vision saving.



Figure 1. RE orbital cellulitis in 2 yr male.

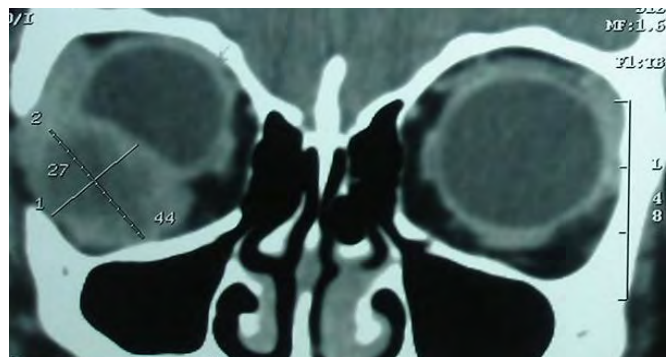


Figure 2. Coronal cut of CT scan showing well defined mass inferotemporally suggestive of abscess formation



Figure 3. Post surgical drainage and antibiotic coverage for 11 days.



Figure 4. 10 yr old patient with orbital cellulitis



Figure 5. Sinusitis and nasal discharge of the involved side

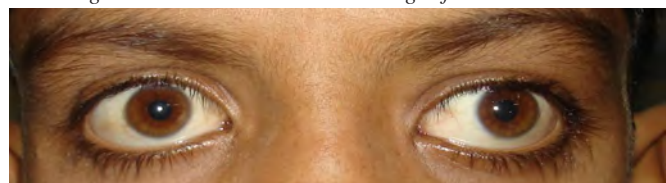


Figure 6. Post treatment -with antibiotics and endoscopic sinus drainage

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Clinical presentation and outcomes following posterior segment IOFB

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Purpose: To review the clinical features, causes of injury, types of foreign bodies, results of vitrectomy and prognostic factors in patients with posterior segment intraocular foreign bodies (IOFB). **Methods:** Study design: Retrospective review of clinical records. Study participants: 108 consecutive patients with posterior segment IOFB presenting at the study hospital between 1994 and 2007; and treated with IOFB removal by intraocular forceps or electromagnet or combination of the two following vitrectomy. Outcome measures: Clinical presentations, investigations, surgical techniques, final anatomical and visual status and duration of follow up. Statistical analysis: Chi-squared test. **Results:** The commonest presenting feature was uniocular visual loss. Work place injuries while chiseling and hammering (67.5%, n=73) without protective eyewear were the commonest. Metallic IOFBs were significantly common at 77.16% (n=98). IOFBs were removed using intraocular magnet (74%, n=80), intraocular forceps (18.5%, n=20) or a combination of the two (7.4%, n=8). Final visual acuity of $\geq 20/120$ was achieved in 32.4% (n=35) cases. Presence of Relative Afferent Pupillary Defect (RAPD) ($p<0.001$), posterior segment haemorrhage ($p<0.001$) and Retinal Detachment (RD) ($p=0.045$) at presentation implied bad prognosis. **Conclusions:** IOFBs were mostly occupational and preventable. Cases presenting with RAPD, posterior segment haemorrhage and RD had bad visual prognosis.

Introduction:

Penetrating ocular trauma with posterior segment intraocular foreign body (IOFB) is an important cause of visual loss in young adults with poor anatomical and functional outcomes of treatment. [1-5] The clinical profile of such injuries in northeast India has not been presented before. The purpose of the present study was to determine the clinical features, causes of injury, types of IOFBs, outcome of vitrectomy and IOFB removal and prognostic factors in patients with posterior segment IOFBs.

Methods:

After obtaining ethical approval from the institutional ethical committee, a retrospective review of clinical records was carried out. The hospital database from 1994 to 2007 was scrutinized and cases with posterior segment IOFBs treated with IOFB removal by intraocular forceps or electromagnet or a combination of the two following vitrectomy were included. Cases treated with modalities other than vitrectomy, with less than six months follow up and incomplete documentation were excluded from the study.

Data collected included patient's age at the time of injury and gender; circumstances of the injury, presenting visual

acuity (VA), clinical signs at presentation and location of the entry wound; method of identification, location, number and nature of the IOFB; details of surgical technique, surgical complications, and repeat surgery(ies) if any; final anatomical and visual status, and duration of follow up. The data was tabulated and analyzed. VA recorded at the last follow up visit was taken as the final VA. Final VA of 20/120 or better was taken as good visual outcome and statistical analysis using the Chi-squared test was carried out to determine which presenting clinical signs pre-empted this outcome.

Results:

208 cases of posterior segment IOFB presented at the hospital during the study period, of which 108 fulfilled the study criteria and were included in the study. Of the 100 cases that were excluded, 53 cases had undergone vitrectomy and IOFB removal but had inadequate postoperative follow up or documentation. Vitrectomy was not advised in 17 (phthisis bulbi in 12, siderosis bulbi in 2 and panophthalmitis in 3 cases) and 30 cases did not accept the treatment offered or chose to receive treatment elsewhere. Thus, considering the cases that did undergo vitrectomy but were not included, the present study had a recruitment rate of 67.1%.

The mean age of the patients at the time of injury was 30 years (5-78 years) and males far outnumbered females at 107:1.

The most common activity which the patients were engaged in at the time of injury was occupational chiseling and hammering (67.5%, n=73). The other circumstances of injury were bomb blast (7.4%, n=8), pipeline and Liquefied Petroleum Gas (LPG) blast (6.5%, n=7), Road Traffic Accidents (RTA) and windshield injuries (4.6%, n=5), fire cracker injury (3.7%, n=4), country made firearm injury (3.7%, n=4) and injury following blast in mines (0.9%, n=1). The circumstances of injury was not documented in 5.5% (n=6) [Fig.1].

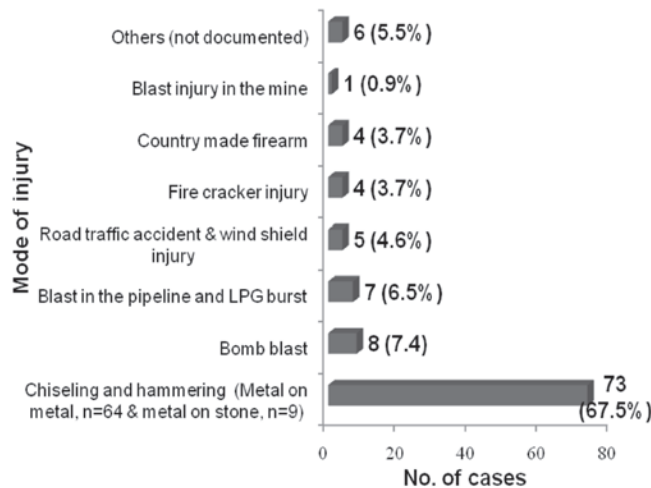


Figure 1 Activity during injury.

Presenting VA was 20/120 or better in 11.1% (n=12) of patients [Fig.2]. The common clinical findings at presentation were lenticular changes (78.7%, n=85) and vitreous haemorrhage (43.5%, n=47) [Table 1]. On an average, every patient had at least 3 different presenting clinical features. The commonest site of entry of the IOFB was cornea (78.7%, n=85) followed by sclera (12.9%, n=14) and limbus (8.3%, n=9) [Figs.3].

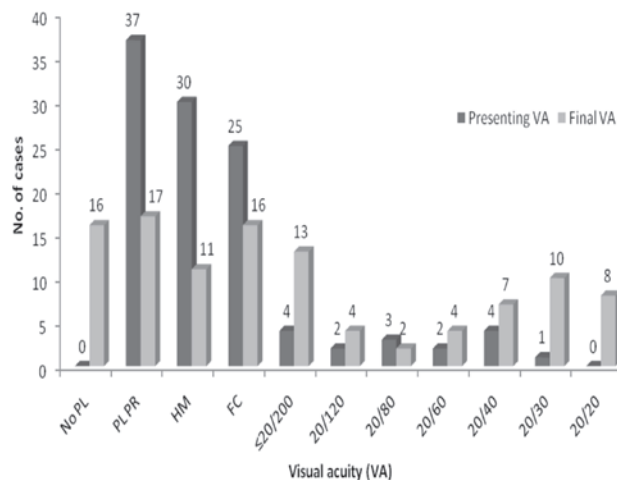


Figure 2 Presenting & final visual acuity.

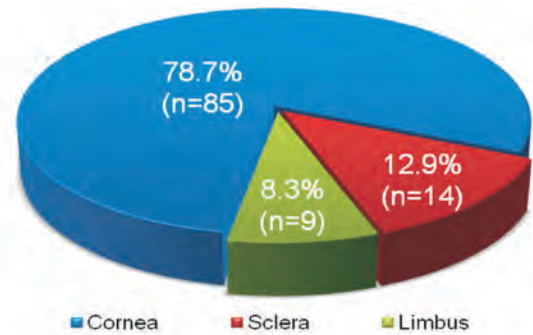


Figure 3 Site of entry wound.

The posterior segment IOFB was identified and localized clinically in 49% (n=53) while the remaining (51%, n=55) required ancillary diagnostic investigations which included B-scan ultrasonography (n=43), CT scan (n=9) and plain picture radiography (n=3).

The entry wound closed spontaneously in 38.6% (n=49). Vitrectomy and IOFB removal was performed on them at a median interval of 16 days (9-23 days). Primary wound closure was carried out in all the patients (35.4%, n=45) who reported first to our institution. IOFBs in this group of patients were removed at the same sitting (primary vitrectomy, n=16 eyes) or in a secondary surgical session (n=29 eyes) at a median interval of 14 days (12-16 days). Primary wound closure was performed elsewhere in 11% (n=14) of the patients before being referred to our institution. IOFB removal in these patients was performed at a median interval of 9.75 days (1.5-18 days) [Table 2].

Interrupted 10-0 nylon or 6-0 vicryl sutures were used in 16 cases for primary closure of the entry wound. In all the cases, surgical technique adopted for IOFB removal was standard 3 port 20G pars plana vitrectomy (STTO/ACCURUS, Alcon, USA). Enlargement of one sclerotomy was required in 11 cases to facilitate negotiation of the foreign body. During the surgery, 26 patients required a secondary, strengthening suture to secure a leaking wound. IOFBs were released of all attachments with the induction of posterior vitreous detachment (PVD) in 27.8% (n=30), while the same was not required in 72.2% (n=78) where PVD pre-existed. IOFB removal was done using intraocular electromagnet (D.O.R.C Intraocular Magnet System) in 74% (n=80), intraocular forceps (Greishaber) in 18.5% (n=20) and the two in combination in 7.4% (n=8).

Retinal detachment (RD) was associated in 39 cases. Intraocular tamponade was used in all of these cases. In these cases, scleral buckling surgery was performed along with vitrectomy and IOFB removal at the same sitting in 46.15% (n=18), with the use of silicone oil (8 eyes) and SF6/C3F8 (10 eyes). In the remaining 21 cases, vitrectomy with SF6/C3F8 tamponade was performed without buckling. Laser retinopexy surrounding the site of impact of the IOFB and in the fundus periphery was performed in 80.5% (n=87). Lens extraction

with (38.8%, n=42) or without (34.2%, n=37) Intraocular Lens (IOL) implantation was done at the same sitting, while 26.8% (n=29) of the cases underwent lens sparing vitrectomy. Penetrating Keratoplasty was required in 1 eye [Table 3]. Majority of the IOFBs (90.7%, n=98) were magnetic metals, though the precise chemical composition of the metal could not be determined. Rest of the IOFBs were stone (3.7%, n=4), glass (2.7%, n=3), wood (0.92%, n=1) and others (1.80%, n=2) [Fig.4]. More than one IOFB were present in 8 cases.

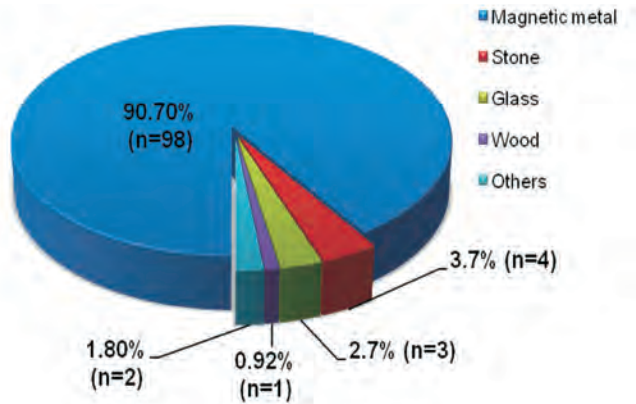


Figure 4 Nature of foreign bodies.

The most common site of lodgment of the IOFB was the retinal surface (68.5%, n=74), while 28.7% (n=31) cases had IOFBs located in the vitreous cavity [Figs.5 and 6]. The sites of impaction of the IOFB on the retinal surface were outside the vascular arcade (67.5%, n=50), posterior pole (10.8%, n=8) and pars plana (1.35%, n=1). However it was not documented in 20.27% cases (n=15). Sizes of the IOFB removed ranged from 0.5mm to 4.85mm in central sagittal section (CSS) [Fig.7].

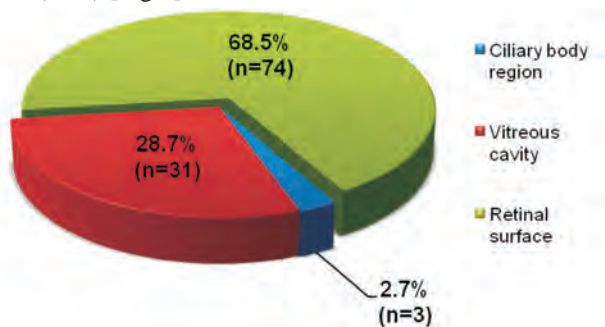


Figure 5 Location of foreign body.

Spontaneously absorbing vitreous haemorrhage was the most common postoperative complication (n=27). Amongst other postoperative complications, recurrent RD occurred in 10 cases, one of which had a giant retinal tear (GRT). Recurrent RD was treated with re-vitrectomy and tamponade (silicone oil in 6 and gas in 4 cases). Severe vitreous exudations developed in 18 cases, and were controlled with oral prednisolone. Cataract developed in 3 cases and was treated by phacoemulsification and IOL implantation. Macular fibrosis developed in 2 cases [Fig.8]. Aphakia developed in 3 patients, and these were

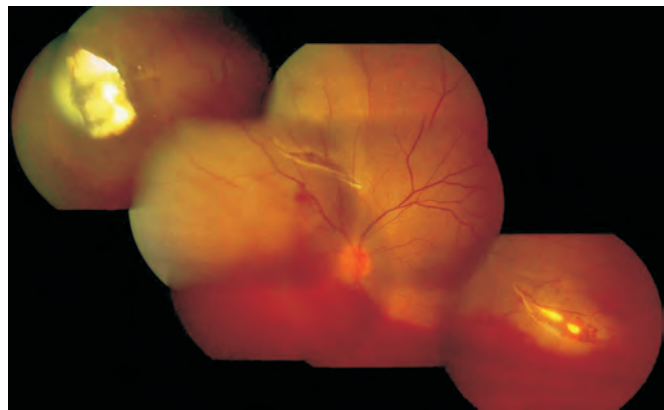


Figure 6 Showing site of retinal penetration (red arrow), site of impact on the retina resulting to cut injury to the retina (yellow triangle) and final intravitreal location of the foreign body (white arrow). Note the associated retinal detachment (white triangle) and vitreous haemorrhage (red triangle).



Figure 7 Showing some of the removed IOFBs (CSS ranging from 0.5 to 4.85mm).

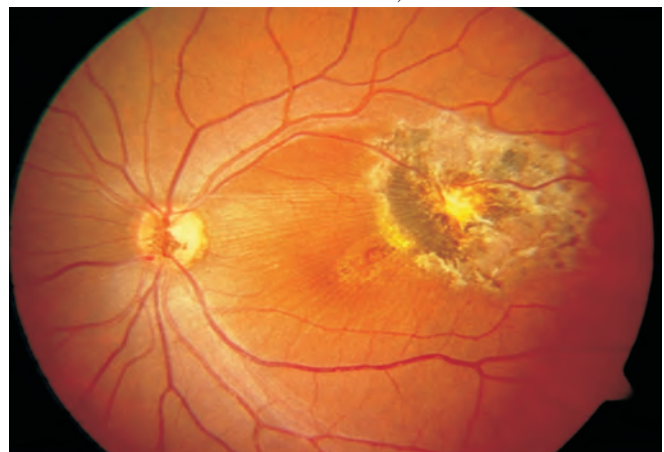


Figure 8 Macular fibrosis (white arrow) following posterior segment injury by IOFB resulting poor vision. Note the changes in the macula.

treated with scleral suture fixated IOL implantation.

The duration of follow up ranged from 6 to 62 months, with modal frequency of follow up being between 6 and 12 months (n=30).

Final visual acuity of 20/120 or better was achieved in 32.4% (n=35) patients [Fig.1]. There was no light perception in 16 patients and 11 cases had cosmetically deformed eyes due to corneal scar or phthisis bulbi. Lens status ranged from pseudophakia in 48 eyes, aphakia in 34, and 26 eyes were phakic [Table 4]. The causes of poor visual acuity after treatment were chorioretinal changes following IOFB removal and retinal reattachment surgery (8 eyes), macular scar (4 eyes), persistent macular detachment (3 eyes), secondary glaucoma (3 eyes), optic atrophy (2 eyes), subretinal neovascular membrane (1 eye), and corneal opacities of various grades (14 eyes), non improvement of vision in spite of IOFB removal and retinal reattachment surgery (9 eyes). However, 13 cases of the corneal opacity group had co-existing posterior segment pathology as mentioned above. Aphakia itself was found to be a major challenge in restoration of binocular vision.

Amongst all the presenting clinical features [Table 1], relative afferent pupillary defect (RAPD) ($p < 0.001$), posterior segment haemorrhage ($p < 0.001$) and RD ($p = 0.046$) were associated with statistically significant poor visual outcome (Final VA $< 20/120$). Corneal and limbal site of entry were apparently associated with better visual outcome. Mode of IOFB removal, i.e. by intraocular forceps or electromagnet, did not have any impact on the final visual outcome. However, intraocular forceps were used in less number of cases.

Discussion:

Ocular trauma with posterior segment IOFB is a major cause of unocular blindness in young adults. [1-3] Studies into such injuries are important to identify the best means to prevent and treat the same. Data on clinical presentation of posterior segment IOFB, treatment outcome and prognostic factors is not available from North East India.

In this case series, the mean age of the patients was 30 years. The previous studies have shown similar age distribution globally. [1-4] All except one of the cases in the present series were male (n=107). The United States Eye Injury Registry (USEIR) documented that 93% of such injury occurred in males, with an average age of 31 years. This gender disparity was attributed to more aggressive behaviour of the males as well as their participation in high risk professions.

Chiselling and hammering (metal on metal or metal on stone) without using safety goggles at the work place was the commonest activity (n=73) causing the injury. Similar finding was observed by Wickham et al and others. [4,12,19,21] This finding indicates that penetrating ocular injury by IOFB commonly occurs at the work places in our region. In the United States, the commonest cause of IOFB is hammering.

As per the USEIR, the incidence of such injuries showed a decreasing trend in the work place and was higher in the home setting. Injuries due to bomb blasts, bursting of pipelines and road traffic accidents had equal frequency in our series. In other cases, the injuries occurred during fire cracker bursting or while operating country made firearms. During these activities, splinters or particles broken off or thrown off at high speed injured the eyes. In the present series, the majority of the IOFBs were metallic, with sizes varying from 0.5mm to 4.85mm CSS. This indicates that appropriate protective eyewear made of 3mm thick polycarbonate could have prevented all of these injuries. Similar observation was also made by Trevor-Roper. [20]

Presenting VA in our series was 20/120 or better in 11.11% (n=12) of the patients. Similar or better presenting VA has been reported in a higher number of cases, ranging from 51% to 63.6%, in the literature. [3-6,12] The reasons for poor presenting vision in the majority of cases in this series are late presentation and media opacities, mainly due to cataract and vitreous haemorrhage.

The common presenting clinical features were loss of vision, corneal opacities, lenticular changes and vitreous haemorrhage. On average, each patient had 3 different clinical presenting features. The observed significance of association between presenting clinical features and poor final visual outcome found in this study did not differ from the published literature. [1,3,4] Amongst all the presenting clinical features (Table 1), RAPD, posterior segment haemorrhage and RD were associated with statistically significant poor final visual outcome ($\leq 20/120$). In the literature, poor visual outcome had been reported when IOFB was associated with RAPD [1,4,5,11], RD [4,5,11] and posterior segment haemorrhage. [1,4,5,11]

In the present series, the site of entry was the cornea in 78.7% (n=85), followed by the sclera in 12.9% (n=14) and limbus in 8.3% (n=9). This distribution is in accordance with that reported in the literature. [4] The site of entry has been reported to have an association with endophthalmitis. [3,5,6] The reported rate of endophthalmitis following penetrating ocular trauma varies from 2 to 13.5%. [3,5-11] The present series does not include any case with endophthalmitis. However, this is more likely a reflection of a selection bias in the study.

The present study highlights that appropriate clinical examination and indirect ophthalmoscopy can diagnose accurately an IOFB in almost half of the cases (49%, n=53). However, the remaining 51% cases required ancillary investigations for conclusive diagnosis. B-scan ultrasonography [23] and CT scan have been reported to have variable sensitivity and specificity in localization of IOFB. [4,13,14,22] In the present series, B-scan ultrasonography was found to be the most sensitive and cost effective investigation for IOFB localization in opaque media.

Internal and external approaches for removal of IOFB are in

practice. Both the methods have their own limitations. There is no universal consensus for the timing and the choice of surgical techniques. Early vitrectomy and internal approach have been advocated. It has been reported to decrease the risk of tractional or rhegmatogenous retinal detachment, collateral damage during manipulation and risk of endophthalmitis.[11,26] Removal of residual lens matter and vitreous debris (haemorrhage) reduces the risk of Proliferative Vitreoretinopathy (PVR) formation. Vitrectomy clears the media and allows detailed retinal examination, particularly for prophylactic treatment.

Removal of IOFB using intraocular magnet is simple and fast, but is applicable only for magnetic foreign bodies. Moreover, if PVD is not induced adequately, there is possibility of vitreoretinal traction and subsequent retinal detachment. However, forceps removal is a complex procedure with an associated increased risk of collateral damage compared to IOFB removal using intraocular magnet. On the positive side, forceps allow removal of the scaffold and substrates that cause fibrous proliferation, and also allow controlled removal of foreign bodies which are, particularly, either encapsulated, impacted or located under the retina. [5,9,12,15] Intraocular electromagnet (74%, n=80) and forceps (18.5%, n=20) were used in the present series at random depending on surgeon's choice, and magnetic property of and ability to grasp the IOFB.

Incidence of RD in this study was 36.11%. This is within the range of 6-40% reported in the literature. [4,5,8] Mechanical affect of the IOFB causing retinal tears and vitreous traction as well as exudation were responsible for the RD. Internal tamponade (gas or silicone oil) with or without scleral buckling was required in 39 of our cases. Retinal detachment recurred in 9 cases, while 1 patient developed a Giant Retinal Tear (GRT) with RD within 6 weeks of post-operative period. In the patient developing the GRT, forceps removal of IOFB was done as the primary procedure. All these cases underwent subsequent retinal reattachment surgery. At the time of final follow up, 4 cases had persistent RD. The possible beneficial effect of prophylactic retinal buckling in such cases has been highlighted in the literature. [24,25] However, in the present series, 16.7% of the cases had scleral buckling procedure without any apparent prophylactic benefit.

It has been suggested that retinopexy at the site of retinal impact is not required during IOFB removal owing to the formation of spontaneous chorioretinal adhesion. However, in our series, 80.5% of cases received laser retinopexy at the time of surgery, surrounding the site of impact as well as in the fundus periphery. Wickham et al [4] in their series performed laser retinopexy in 63% cases and found a decrease in the risk of subsequent RD. Similar observation was made in our study. However, one patient developed a foveal scar following laser photocoagulation, resulting in poor post-operative visual

recovery. Like in other studies [4,5,17,18], an association between RD and poor final visual outcome was observed in this study as well. With developments in vitreoretinal surgical techniques, the surgical outcome has improved remarkably. [4] However, in spite of it, IOFB still remains a very complicated and challenging situation.

In the present study, some degree of improvement in vision following vitrectomy and IOFB removal was achieved in 71 cases overall. While it remained unchanged in 21 patients, vision deteriorated following treatment in 17 cases, including 16 who had no light perception. Good visual recovery ($\geq 20/120$) was achieved in 35 cases. 57 cases had VA less than 20/120. Posterior segment pathology (43 cases) and corneal opacity were the major factors contributing to poor final VA. A number of vitreoretinal complications and other problems have been reported as contributing to poor visual and functional outcome in cases with IOFBs. [1-6,8,11,21] However, the complex nature of the injury and the treatment, as well as the influence of a number of factors, bring forward conflicting results in the literature.[13,25]

The present study is a retrospective case series based in a tertiary eye care setting. As such, it has its limitations and shortcomings with regard to generalisation of the results. The injuries were diverse in nature, and primary management protocol and referral strategies were different. As a result, patients presented at the institution at diverse time intervals with variable clinical status, causing difficulty in interpretation of various associations between presenting features, management strategies and final visual outcome. Of all the cases reporting at the hospital during the period of the study, only 51.9% (n=108) had complete documentation and minimum 6 months' post-operative follow up, and thus could be included as per the study criteria. We appreciate that a prospective study involving consecutive cases is better suited for commenting on the nature and circumstances of the injury, while a randomised controlled trial (RCT) is a better design for evaluating treatment efficacies.

The present series documents that penetrating ocular injury caused by IOFB is a serious sight threatening condition. Vitreoretinal pathology is an important determinant of final visual outcome. While the injury itself is a completely preventable occupational hazard in the vast majority, good visual recovery following the treatment offered in this series was achieved only in about a third of the

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Cause and Clinical Profile of 379 cases of Ocular Trauma

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Christian Medical College, Vellore

Background: Ocular trauma is a major cause of monocular blindness and visual impairment throughout the world. Population based epidemiological studies in south India have found prevalence rates of 2- 10%. The aim of our study was to find the demographic and clinical profile of patients with ocular trauma. **Materials and methods:** All patients who came to our emergency services between July 2004 and January 2005, with history of ocular trauma or foreign body in either eye, within 2 weeks of the date of presentation were included in this prospective, cross sectional survey and underwent a complete eye examination including best corrected visual acuity, slit lamp examination and fundoscopy. **Results:** Age distribution of ocular trauma showed gradual decline with age with maximum number of patients in the 0-10 year age group. Temporal spectrum showed a bimodal peak in January and November. Male predomination accounted for 71 % of patients. Work related trauma accounted only for 22.4% of patients. The “stick” predominated as the premier object of insult (22.2%), Vegetative matter 10.4%. Road Traffic Accidents 9.4% and Metal 8.2%. Chemical injuries accounted for 7.7% and Open globe injuries 19.3% of patients. **Conclusions:** The demographic factors associated with an increased incidence of ocular trauma include young age, male sex and festive seasons of the calendar year including November and January. The “stick” was the most common object of insult. Clinical profile showed a high incidence of open globe injuries(19.3%). This study highlights some differences in the profile of ocular trauma as seen in a developing country. The predominant occupation in rural India is agriculture and the premier form of fuel used in most households for cooking is firewood. Thus the results of a high percentage of ocular trauma related to sticks and vegetative matter. Chemical injuries which accounted for 7.7% of patients with ocular trauma and were predominantly related to the local practices of “pan” chewing. The high percentage of open globe injuries is indicative of the severity of ocular trauma seen in rural India.

Introduction:

Ocular trauma is a major cause of monocular blindness and visual impairment throughout the world. Population based epidemiological studies in south India have found prevalence rates of 2- 10% [1-4]. Blindness resulting from trauma has prevalence rates of about 0.6 – 0.8% [1-3]. The use of eye protective devices in India is very low [3]. Estimating the cause and clinical profile of ocular trauma could be a major step in planning preventive strategies and making it a public health priority in developing nations.

Materials and Methods:

All patients who came to our emergency services between July 2004 & January 2005, with history of ocular trauma to one or both eyes or foreign body in either eye, within 2 weeks of the date of presentation were eligible to be considered for this prospective, cross sectional survey and underwent

a complete eye examination including best corrected visual acuity, slit lamp examination, fundoscopy and intraocular pressure measurement (when indicated).

Results:

Age- The age distribution of ocular trauma showed a gradual decline with increasing age. The maximum number of patients (82) were seen in 0-10 years age group.

Temporal spectrum- Monthly distribution of ocular trauma shows 2 spikes in the months of January & November. A day-to-day analysis over these 2 months showed a higher incidence of trauma closer to the dates of festivals Sex- Males(71%) predominated over females and the trend continued to persist in all age groups. **Place of injury-** Work related trauma accounted only for 22.4% of patients **Object causing injury** The ‘kutchi’ or stick predominated as the premier object of insult (22.2%). Other Vegetative matter 10.4%, Road Traffic

Accidents 9.4% Metal 8.2%. Chemical injuries accounted for 7.7% of patients with ocular trauma. Open Globe injuries accounted for 19.3% of patients with ocular trauma.

Discussion:

Age

The number of patients presenting with trauma showed a linear decline with age. The bimodal pattern seen in various western studies did not show up here. This was probably because of the low number of cases more than 70 years corresponding to India's life expectancy. The life expectancy quoted in India at the moment is 70 years.

Age was correlated statistically with sex & the males predominated in every decade. The maximum difference was seen in the most productive years of life 20 to 50 years.

The age group between 11-20 showed the highest percentage of open globe injuries (28.8%). This could reflect the risk taking behavior, the lack of experience & knowledge of dangers & first aid in work & other activities seen in the young man. However the p value correlate between age & open globe injuries was not statistically significant. (p=0.258).

Temporal spectrum of ocular trauma

Monthly distribution of ocular trauma shows 2 spikes in the months of January & November. Incidentally they correspond to the 2 most celebrated festive seasons of the calendar year-Diwali & Pongal. A further analysis on a day-to-day basis over these 2 months showed a higher incidence of trauma closer to the dates of these festivals.

Sex distribution of ocular trauma

The male predominance of ocular trauma continued to prevail in the present study. This is more relevant in the Indian setting, where the majority of the population is rural & a considerable number of women are simple housewives with limited travel, work & sports. The higher incidence in males may be related to the injuries related to assault, Road traffic accidents, work & sports.

Place of injury

Most other epidemiological studies on ocular trauma have shown work related trauma to predominate. In our study work related injuries accounted for 22.4 %.

Object of injury

The 'kutchi' or stick predominated as the premier object of insult (22.2%). This could well be expected in an agricultural society where the predominant occupation is a daily wages labourer in the agricultural field. Also in our qualitative analysis we found that in many homes, firewood was the form of fuel used for cooking & injuries occurred when breaking sticks & cutting wood for fire.

Vegetative matter was the chief object implicated in corneal ulcer formation.

Chemical injuries constituted 7.7%. The predominant aetiology in the 0 to 10 age group were from "squeeze tubes" containing lime. Lime is Calcium hydroxide powder. Chewing of beetle nut is a very common practice in India. Lime is chewed with beetle nut to neutralize the acidic astringent juice of the nut. It is stored in disposable tubes from which it is squeezed out for use. Small children sustain lime injuries when they play with tube packets containing lime and accidentally squeeze the product onto their face. Adaptation of other methods of lime storage that are safer & tighter are warranted.

Conclusions:

The demographic factors associated with an increased incidence of ocular trauma include-Young age, male sex and festive seasons of the calendar year including November and January.

The most common object causing injury was the stick (22.2%). The most common object implicated in open globe injuries was metal (17.8%). Mechanical injuries predominated over chemical injuries which accounted for 7.7% of patients with ocular trauma. Open globe injuries constituted 19.3% of patients. Work related injuries occurred in 22.4% of patients.

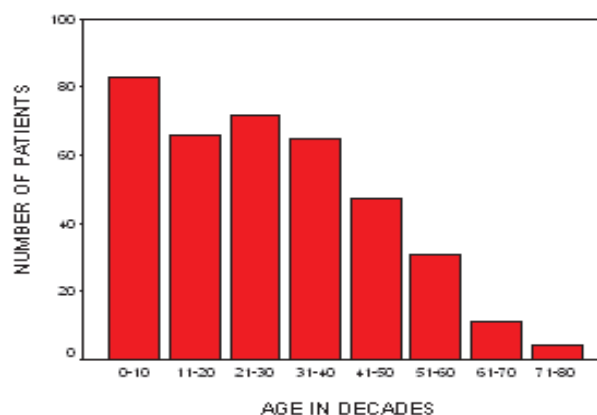


Figure 1 Age distribution of ocular trauma with increasing age

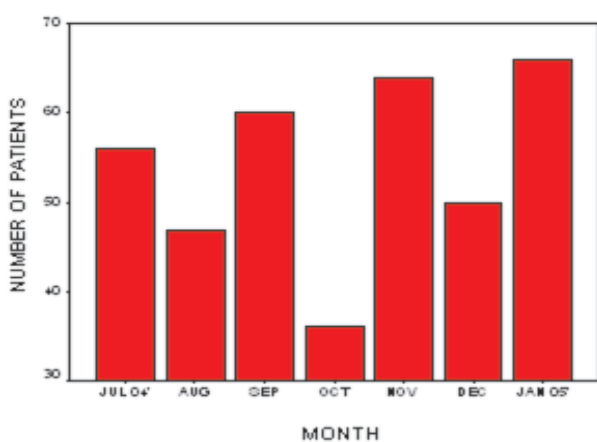


Figure 2 Temporal spectrum of ocular trauma over 7 months

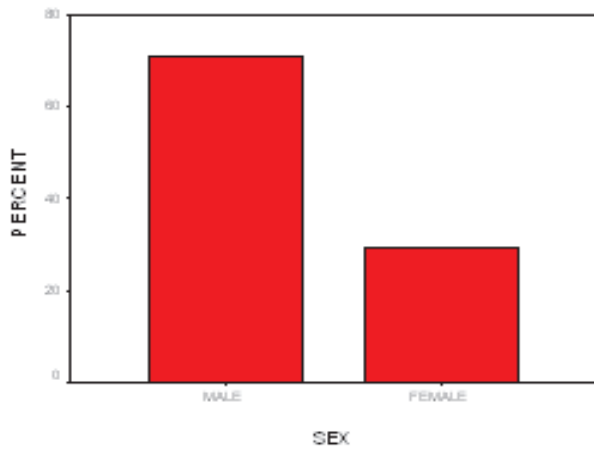


Figure 3 Ratio of males: females with ocular trauma

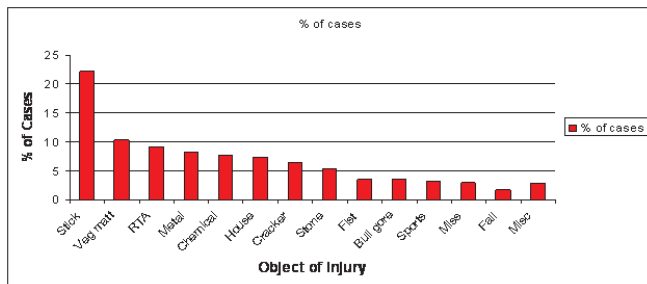


Figure 4 Profile of objects causing ocular trauma

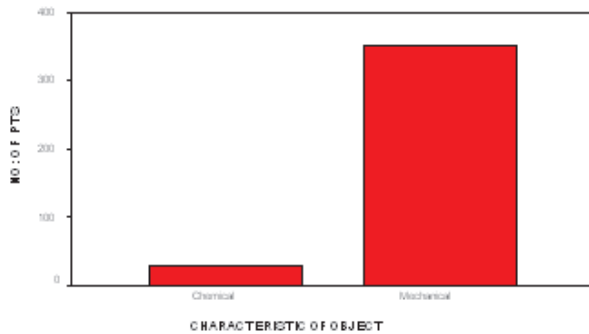


Figure 5 Bar graph showing difference in percentage between chemical and mechanical injuries.

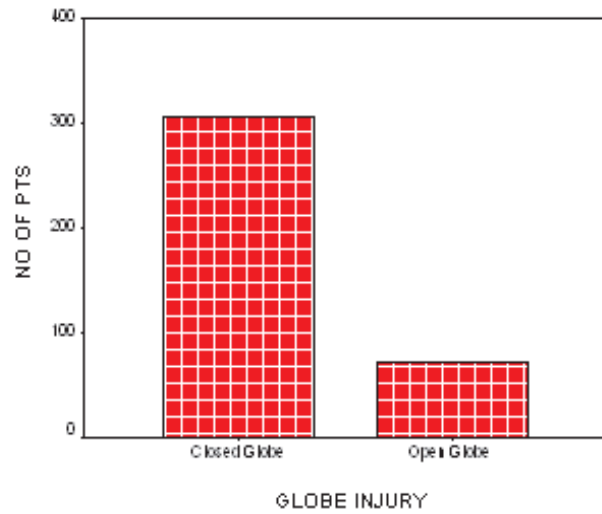


Figure 6 Bar graph showing the proportion of open globe injuries to closed globe injuries.

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