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A Closer Look at “Informed Consent”

An informed consent is a legal document: an agreement for a proposed treatment, or non-treatment; or a proposed invasive procedure. An informed consent requires the physician to disclose the benefits, risks and alternatives to the proposed treatment or non-treatment and it is the method by which fully informed rational individuals may be involved in choices regarding their health care.

What does the informed consent stem from? It stems from (1) the ethical right an individual has to decide what is done to his or her body,(2) the physicians' ethical duty to make sure that individuals are involved in the decisions about their own health care.

There are 3 phases of procuring an informed consent and all three phases involve information exchange between the doctor and the patient and are part of patient education. In words, and in a language that the patient can understand, a physician must convey the details of the planned procedure, its serious risks and potential benefits and any other feasible alternatives. The patient should also be presented with information on the most likely outcome of the treatment. The information given to the patient should include the diagnosis, its seriousness and implications; available treatment options and feasible alternatives, associated risks or complications and benefits with each modality, whether any discomfort is expected to be associated with treatment, immediate and long term side effects of the treatment, the impact of treatment on the patients’ daily life and activities, its duration and all the implied and hidden financial implications.

In the 2nd phase of obtaining the informed consent, the physician must ascertain whether the patient has understood what has been said, and that the risks have been accepted and the patient is giving consent to the procedure with full knowledge and forethought. Finally, the individual must sign the consent form which documents the major points of consideration. It is critical that the patient should receive enough information on which to base the informed consent and the consent should be wholly and voluntarily given and not forced in any way. It is the responsibility of the physician to enter details of the conversation leading to the informed consent in the case sheet.

For treatments that carry little risk approval of the doctors’ plan is implied by simply getting a prescription filled, allowing blood to be drawn for lab tests or seeing a specialist. This is called simple consent.

However for treatments that carry some risks for the patient, an informed consent is necessary. Sometimes health care workers refer to the consent form itself as an informed consent. This is not accurate as informed consent is the process or action that takes place as the patient learns
about and considers a treatment before agreeing to it. The patients’ signature in the consent form is evidence that it took place. If the patient does not want the procedure he may be asked to sign a refusal form.

Informed consent laws specify the type of information that the patient must be given so that they can make an informed decision about having medical care, diagnostic studies or treatment. The information given to the patient can be “reasonable information” or a “full complete disclosure”.

Informed consent for a clinical trial is usually required to be more detailed and thorough than consent for a standard medical procedure or treatment. This is because of the higher chances of unknown effect with new treatment, and it is even more important that the patient is aware of these possibilities.

Some of the information that the patient wants may not be available. In such a situation, the spirit of the laws of the informed consent requires the healthcare provider to give the best answer possible which may be ‘we don’t know yet’!

The patient is judged competent to make his own medical decisions and has the right to refuse any and all the medical treatment and diagnostic procedures. In this situation it is the duty of the healthcare provider to inform the patient about the risks or likely consequences of this choice, and get the patients’ signature on the ‘informed refusal’. If the patient does not wish to sign, the doctor may ask the witnesses to sign stating that the patient was informed.

If a patient can restate the information that has been imparted, then that will help to confirm that he/she has received adequate information and understood it. The nursing personnel who gets the patients’ signature is obliged to report about the patients’ understanding regarding what has been said or any concern about his/her capacity to make decisions.

Although the law requires a formal presentation of the procedure or treatment to the patient, physicians have expressed doubt on the wisdom of this. Many believe that informing patient of the risks of treatment might scare them into refusing it, even when the risks of non treatment are greater.

The result of informed consent is greater safety and protection for patient, physician and society in general.

**Dr. Meena Chakrabarti MS DO DNB**
Editor, KJO
Toxoplasmosis is the most common cause of posterior uveitis in many parts of the world. Prevalence is more in tropical countries than cold areas.\textsuperscript{1,2} It is caused by \textit{Toxoplasma gondii}, an obligate intracellular protozoan parasite which exists in three forms: oocyst, bradyzoite and tachyzoite. Tachyzoites are the invasive forms, bradyzoites are the encysted forms and sporozoites (oocysts) exist in the cat only. Cats are the definitive hosts where as humans and other animals act as intermediate hosts. Human infection by \textit{T.gondii} can be acquired or congenital. The transmission occurs by ingestion of raw or undercooked meat infected with tissue cysts, ingestion of food and water contaminated with oocysts, ingestion of eggs and milk contaminated with tachyzoites, blood transfusion, organ transplantation and transplacental transmission.\textsuperscript{1,2} \textit{T.gondii} can also rarely be transmitted via blood and infections of laboratory personnel through contact with contaminated needles.

### Congenital toxoplasmosis

Congenital infection develops in 30\% to 50\% of infants born to mothers with acquired toxoplasmosis during pregnancy.\textsuperscript{6} The prevalence of acquired toxoplasmosis during pregnancy is 0.2\% to 1\%. The lowest incidence occurs in the first trimester (15\% to 20\%), and the highest incidence is in the third trimester (59\%) possibly because of increased vascularity of the placenta at that time. Early maternal infection in the first trimester is severe and leads to spontaneous abortion. Transmission during second trimester may result in moderate disease that presents as fever, maculopapular rash, hepatosplenomegaly, seizures, jaundice, thrombocytopenia and lymphadenopathy. The classic triad of congenital toxoplasmosis is retinochoroiditis, hydrocephalus and cranial calcification. Maternal infection in the third trimester usually results in asymptomatic infants.

### Acquired toxoplasmosis

Typically 70\%-90\% of immunocompetent patients who acquire toxoplasmosis are symptom free. Even a symptomatic disease is so mild and nonspecific that it goes unrecognized. The true incidence of ocular toxoplasmosis in the immunocompetent patients is estimated to be ranging from 2\%-20\%. The time interval between systemic infection and the appearance of ocular lesions is variable, ranging from few days to years. Acquired toxoplasmosis initially starts as an acute flu like illness with malaise, myalgia, hay fever, maculopapular skin rash and lymphadenopathy. This picture is usually self-limiting and usually resolves in 2-4 weeks. Rarely the clinical manifestations may be severe leading to pneumonitis, polymyositis, myocarditis, encephalopathy, hepatitis and splenomegaly, resulting in significant morbidity and mortality. Toxoplasmosis can also affect immunocompromised patients with AIDS, Hodgkins disease, hematological malignancies, and organ transplant recipients.\textsuperscript{7} Ocular toxoplasmosis in AIDS patients occurs in 1-2\% cases and 30 -50\% of these patients have intracranial involvement. Hence, all AIDS patients with ocular toxoplasmosis must undergo a complete neurological evaluation including computed tomography (CT) or magnetic resonance imaging (MRI) with contrast and lumbar puncture.
Ocular toxoplasmosis

Ocular toxoplasmosis occurs when the parasite invades the intraocular tissue through the blood stream. The proliferating parasites cause retinal necrosis and hypersensitivity reaction to *T. gondii* antigens causes vasculitis, uveitis and papillitis. Recurrence of infection is due to multiplication of parasites from the retinal cysts located at the borders of retinochoroidal scars. Earlier reports suggested that ocular toxoplasmic scars were residua of congenital infection, however there are no laboratory methods to discern between congenital and acquired infection. Recent serological studies have shown that acquired infections play more role in ocular toxoplasmosis.

Clinical features

**Symptoms:** Children present with reduced visual acuity, strabismus, nystagmus and leuucoria. Adults complain of decreased vision, floaters, and metamorphopsia.

**Signs:** The typical toxoplasmic lesion is a focal necrotizing granulomatous retinochoroiditis at the edge of a pigmented scar accompanied by vitreous inflammation (Fig. 1). When vitritis is severe that the retinal lesion is just seen, it is described as “headlight in the fog”. Ophthalmoscopically, a yellowish–white exudate is seen with ill-defined borders due to surrounding retinal oedema (Fig. 2). The lesion progressively decreases in size and cicatrisation occurs from the periphery towards the center, with variable pigmented hyperplasia and choroid atrophy. Vitritis is usually seen in all cases. It may present as diffuse or localized exudates, pigments, hemorrhage or posterior vitreous detachment.

Vasculitis in ocular toxoplasmosis, either adjacent or distant from the active lesion, mainly involves the veins (Fig. 3). It may present as diffuse periphlebitis, frosted branch angiitis, (Fig. 4) or segmental vasculitis. It is produced by the antigen-antibody deposition in the vessel walls. Less commonly, *kyrieleis arteritis* – presence of periarterial exudates or plaques, not associated with vascular leakage or obstruction may also be seen (Fig. 5 a, b). Complications such as retinal hemorrhages, vascular occlusions, shunts, and choroidal neovascular membrane (Fig. 6) are however uncommon.

Anterior uveitis in ocular toxoplasmosis may either be granulomatous or non granulomatous. It is probably a hypersensitivity reaction to *Toxoplasma* antigen.
### Table showing various antitoxoplasma drugs with dosage in adults and children

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<th>Adult dose</th>
<th>Pediatric dose</th>
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<tr>
<td>Pyrimethamine</td>
<td>Loading dose: 100mg</td>
<td>Infants: 1mg/kg once daily for 1 year</td>
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<tr>
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<td>Treatment dose: 25mg/day</td>
<td>Children: Loading dose 2mg/kg/day (max. 100mg/day)</td>
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<tr>
<td>Folinic acid</td>
<td>7.5 - 15 mg in alternate days</td>
<td>5mg every 3 days</td>
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<td>Sulphadiazine</td>
<td>4g daily divided every 6 hours</td>
<td>Newborns: 100mg/kg/day divided every 6 hours</td>
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<tr>
<td>Clindamycin</td>
<td>150-450 mg/dose every 6-8 hrs (max. dose: 1.8g/day)</td>
<td>Children: Loading dose: 75mg/kg Treatment dose: 120-150 mg/kg/day; divided every 4-6 hours (max. dose: 6g/day)</td>
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<tr>
<td>Trimethoprine – sulphamethoxazol</td>
<td>1 tablet twice daily for 4-6 weeks</td>
<td>8-25 mg/kg/day in 3-4 divided doses</td>
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<tr>
<td>DS tablet (160mg/800mg)</td>
<td>Commonly used 300mg 4 times a day.</td>
<td>6-12 mg TMP/kg/day in divided doses every 12 hours</td>
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<td>Azithromycin</td>
<td>Loaded dose: 1g</td>
<td>Loading dose: 10mg/kg/day (max. dose: 500mg/day)</td>
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<td></td>
<td>Treatment dose: 500 mg once daily</td>
<td>Treatment dose: 5mg/kg/day (max. dose: 250mg/day)</td>
</tr>
<tr>
<td>Atovaquone</td>
<td>750mg every 6 hours</td>
<td>40mg/kg/day divided twice daily (max. dose: 1500mg/day)</td>
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It presents as mutton fat keratic precipitates, fibrin, anterior chamber cells and flare, Koeppe and Busacca nodules and posterior synechiae. In case of delayed treatment, complications like pupillary block, rubeosis iridis, cataract and glaucoma may develop.

Atypical presentations of ocular toxoplasmosis may be:

1. Punctate outer retinal toxoplasmosis
2. Neuroretinitis
3. Papillitis
4. Multiple pseudoretinitis
5. Fuchs’ heterochromic iridocyclitis

---

**Fig. 4. Frosted Branch Angiitis with retinochoroiditis**

**Fig. 5. a - Active retinochoroiditis with overlying vitritis and Kyrieleis arterialitis. b - Healed lesion after 2 months of antitoxoplasma therapy**

**Fig. 6. Healed toxoplasmic retinochoroiditis scar with choroidal neovascular membrane**
Fuchs’ heterochromic iridocyclitis

The incidence of chorioretinal scars in Fuchs’ patients varies from 8% to 65%. It has been proposed that primary retinochoroidal inflammation results in production of antibodies which cross react with anterior chamber antigens causing anterior uveitis, iris atrophy and heterochromia.

Multifocal or diffuse retinochoroiditis is usually seen in immunocompromised or elderly patients (Fig. 8). Toxoplasmic scleritis is associated with severe retinitis producing overlying choroidal and scleral inflammation.

Complications of ocular toxoplasmosis include cataract, secondary glaucoma, band keratopathy, retinal detachment, cystoid macular edema, optic atrophy and choroidal neovascular membrane.

Punctate outer retinitis

These lesions occur as gray white multifocal, fine punctate lesions of the deep retina and retinal pigment epithelium. There is little or no vitritis. Usually significant optic nerve involvement with atrophy is seen leading to significant visual loss. These lesions are most frequent in the first or second decades of life and may be congenital or acquired. Autoimmune reaction to retinal antigen is said to be responsible for the development of such lesions.

Neuroretinitis

It initially presents as severe disc edema, hemorrhages, venous engorgement and overlying vitritis followed by juxtapapillary retinochoroiditis and macular star (Fig. 7). The treatment must be prompt and aggressive to prevent visual loss.

Multiple pseudoretinitis

It is characterized by multiple retinal lesions, apparently active. After regression, there is only one scar, derived from the real retinochoroiditis and other pseudo lesions completely disappear.

Unilateral pigmentary retinopathy

It has been reported as a sequela of chronic recurrent toxoplasmosis.

Fig. 7. Toxoplasmic retinochoroiditis with neuroretinitis

Ocular toxoplasmosis in AIDS

Lesions are multifocal, extensive, aggressive, and may be bilateral, with large areas of confluent retinal necrosis (Fig. 9). Moderate to severe vitritis may be seen, although vitritis may be minimal in some cases. Lesions do not occur adjacent to a retinochoroidal scar, instead the lesions develop in a peri-vascular distribution, which suggests newly acquired infection or dissemination of parasites from non-ocular sites in the body. Infection usually produces a full-thickness retinal necrosis, but early lesions may be confined to either the outer or inner layers.
Diagnosis

The definitive diagnosis of ocular toxoplasmosis can be made by either isolation of the organism from body fluids, or detection of \( T. gondii \) DNA using polymerase chain reaction (PCR) or detection of antibodies. Various serological tests for diagnosing toxoplasmosis include Sabin-Feldmann dye test, indirect fluorescent antibody test, immunosorbent agglutination assay and enzyme-linked immunosorbent assay (ELISA). ELISA test is the standard test used by most laboratories to detect IgG, IgM, IgA and IgE antibodies, however, false positive results can occur due to presence of rheumatoid factor (RF) and antinuclear antibodies (ANA). In cases of diagnostic dilemmas, ocular fluids (aqueous or vitreous) can be tested for polymerase chain reaction and antibodies. Antibodies titers are measured in aqueous humor and serum and Witmer-Goldman coefficient is calculated. Detection of \( T. gondii \) antibodies by ELISA and DNA by PCR test in aqueous humor helps in diagnosing ocular toxoplasmosis especially in immunocompromised patients.

Management

Ideal therapy of ocular toxoplasmosis should completely eradicate the parasite. But current treatment aims to stop the multiplication of the parasite and limit the intraocular inflammation. The standard treatment includes a course of antiparasitic drugs along with oral corticosteroids for a minimum of 4 -8 weeks. Holland et al studied various treatment regimens for ocular toxoplasmosis given by uveitis specialists all over the world. They concluded that the most popular regimen known as ‘classic therapy’ consists of pyrimethamine, sulphadiazine and prednisolone. Quadruple therapy includes clindamycin along with the triple regimen. Other systemic antibiotics which are used especially when there is intolerance to the above drugs include trimethoprim – sulphamethoxazole, azithromycin, spiramycin, atovaquone and tetracyclines.

Indications for treatment in ocular toxoplasmosis are:

1. Lesions affecting the posterior pole close to macula or optic nerve
2. A lesion within the temporal arcade
3. Lesion threatening a large vessel
4. A lesion that has induced a large haemorrhage
5. A lesion with intense inflammatory reaction or severe vitreous haze.
6. Extensive lesion irrespective of location.
7. Congenital toxoplasma retinochoroiditis within one year of life.
8. A newborn diagnosed with congenital toxoplasmosis
9. Any lesion in an immunocompromised host

Treatment regimens during pregnancy are:

**First trimester** – Spiramycin, sulphadiazine

**Second trimester** (>14 weeks) - Spiramycin, sulphadiazine, pyrimethamine and folinic acid

**Third trimester** - Spiramycin, pyrimethamine and folinic acid

Prophylactic treatment for ocular toxoplasmosis in immunocompetent patients was studied by Silveira et al and they showed a significant reduction in the recurrence from 24% to 7% in patients given trimethoprim-sulphamethoxazole over a period of 20 months. Randomized controlled trials have shown prophylaxis with antibiotics to be effective against disseminated toxoplasmosis in immunocompromised patients. Also Bosch – Driessen et al recommended a prophylactic treatment with antitoxoplasma drugs in all patients with inactive toxoplasmic retinochoroiditis undergoing cataract surgery.
**Conclusion**

Toxoplasmosis is a recurrent and progressively destructive disease with potentially blinding and even fatal consequences. Undercooked meat and contaminated water or food contaminated by oocysts from cat faeces are the sources of infection. It has been shown in recent studies that postnatal acquired infection is more common than congenital infection. However, disease transmission can be prevented by following strict food hygiene, hand washing, and environmental measures. There is no treatment available to eradicate the encysted tissue form. Recurrence occurs in 79% of patients despite the use of antiparasitic drugs and visual prognosis is not affected by use of multiple antiparasitic medications. As there is no consensus among uveitis specialists for treatment of ocular toxoplasmosis, randomized controlled prospective studies are necessary to formulate the management.

**References**

Unusual Visual Manifestations of Pituitary Tumours

Dr. Meenakshi Y. Dhar MS, Dr. Niranjan K. Pehere MS

Abstract

A retrospective study of 57 cases with pituitary tumours was done. The visual fields [VF] were analysed and pattern of visual field was noted. 16 patients had normal visual fields. 41 patients had VF defects [VFD] in the temporal quadrant. They were variants of the classical bitemporal field defect, the characteristic finding of pituitary lesions. Unusual field defects like arcuate scotomas [n=1], binasal VFD [n=1], bilateral superonasal quadrantanopia [n=1] were found. 13 patients had unusual manifestations: Motor manifestations in 8 patients (1 patient also had Superior orbital fissure syndrome), 7 had pituitary apoplexy, papilloedema in 1 patient (due to obstruction of Foramen of Monro). An attempt was made to explain these.

Key Words: Pituitary Tumours, Apoplexy, Visual Field Defects.

Introduction

Pituitary adenoma is the most common tumour to affect pituitary \(^1\) accounting for 10 to 15 % of intracranial neoplasms. \(^2\) The clinical manifestations are varied depending on the cell type within the tumour \(^1\), hypo or hypersecretion of hormones, direction of local spread and invasion of adjacent structures \(^2\). Both nonvisual and visual manifestations are of extreme importance in the diagnosis, management and prognosis of patients with pituitary adenomas. \(^1\)

Pituitary adenomas are diagnosed earlier nowadays due to availability of radioimmunoassay techniques for the hormones and increasing use of CT scanning and MRI imaging, done at times for indications unrelated to suspicion of pituitary tumours (like after head injury, or for evaluating headache). The percentage of patients presenting with visual loss or from the effect of tumour has decreased dramatically over last 50 years and now, the most common presentation is endocrine dysfunction. However there remain patients in whom visual sensory or motor dysfunction of extraocular muscles occur either as presenting manifestation or as an associated feature of a pituitary adenoma. \(^1\)

Bitemporal field defect is the most common visual abnormality produced by pituitary adenomas.\(^1\) It can be uni or bitemporal hemianopia usually involving superior field first, and more densely than inferior field. \(^2\) Apart from these, pituitary adenomas can also have unusual ophthalmic manifestations like arcuate field defects, isolated nasal hemianopias, papilloedema, ocular motor dysfunction (of which pituitary apoplexy being the most common cause), CSF rhinorrhoea etc. \(^1\)

In this case series, we present a few of such findings in our series of patients.
Material and Methods:

57 patients were enrolled in the study.

Type of study: Retrospective analysis of the case records of patients with pituitary adenoma who visited the ophthalmology outpatient department over last 2 years.

Inclusion criteria: An ophthalmic evaluation was done including assessment of visual acuity and visual fields for all patients of pituitary tumour. Visual field defects were noted in every case. Any degree of bitemporal fields was taken as the expected type of field defect i.e. whether complete/incomplete, quadrantanopic or hemianopic, relative or absolute. All other visual abnormalities, if any, were noted including visual loss, papilloedema, relative afferent pupillary defect [RAPD]. III, IV, V, VI nerve palsies were noted. Visual field defects other than temporal were noted as atypical: Nasal field defect without temporal field defect, arcuate field defects, central or centrocaecal field defects.

Neuroimaging films were reviewed and an attempt was made to find reasons for the unusual field defects. Normal visual acuity for distance was defined as 6/6 on Snellen's chart with appropriate refractive correction. Diagnosis of visual field defects was done according to the system suggested by Ravi Thomas et al as follows: Quadrantanopia was diagnosed if either of the following criteria were fulfilled:

1. Depression of thresholds by 5 db or more, in 3 or more contiguous points adjacent to the vertical meridian in the involved quadrant as compared to their mirror image points across the vertical meridian.

2. The pattern deviation plot showed 3 or more points adjacent to the vertical meridian in the involved quadrant depressed to the 1% probability level with normal mirror image points across the vertical meridian. For the diagnosis of hemianopia, the diagnostic criteria for quadrantanopia had to be applicable to both quadrants comprising the hemifield.

3. Advanced field defects were considered hemianopic if comparison of the least involved quadrant across the vertical meridian, met the threshold depression criteria for the diagnosis of quadrantanopia. Here the pattern deviation plot criterion on its own was not considered diagnostic.

4. Atypical field defect was defined as a defect that did not fit into any characteristic diagnostic pattern considered typical of pituitary adenomas.

Observations

Out of 114 eyes of 57 patients, 62 eyes had normal vision. 11 patients had normal vision in both the eyes. (Table 1). 3 patients were bilaterally blind with only light perception. 5 others were blind in one eye with temporal hemianopic field defect in the other eye.

<table>
<thead>
<tr>
<th>Visual acuity</th>
<th>No of eyes</th>
</tr>
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<tbody>
<tr>
<td>6/6</td>
<td>62</td>
</tr>
<tr>
<td>6/9- 6/18</td>
<td>28</td>
</tr>
<tr>
<td>6/24- 6/60</td>
<td>7</td>
</tr>
<tr>
<td>&lt; 6/60</td>
<td>17</td>
</tr>
<tr>
<td>Total</td>
<td>114</td>
</tr>
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</table>

16 of the 57 patients studied, had no field defects. The types of the field defects are shown in Table 2. Only 10 patients [17%] had typical bitemporal hemianopia. 10 of them had 3 or more quadrants involved.

<table>
<thead>
<tr>
<th>Type</th>
<th>No of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>1  Bilateral superotemporal quadrantanopia</td>
<td>4</td>
</tr>
<tr>
<td>2  Hemianopia one eye, contralateral superotemporal quadrantanopia</td>
<td>4</td>
</tr>
<tr>
<td>3  Classical bitemporal hemianopia</td>
<td>10</td>
</tr>
<tr>
<td>4  Involvement of 3 or more quadrants unilateral/bilateral</td>
<td>10</td>
</tr>
<tr>
<td>5  One eye blind, other eye temporal hemianopia</td>
<td>5</td>
</tr>
<tr>
<td>6  Both eyes blind</td>
<td>3</td>
</tr>
<tr>
<td>7  Both eyes normal</td>
<td>16</td>
</tr>
<tr>
<td>8  Atypical (3 atypical of pituitary adenoma, 2 nonspecific)</td>
<td>5</td>
</tr>
<tr>
<td>Total</td>
<td>57</td>
</tr>
</tbody>
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Out of 57 patients studied, 13 patients showed atypical manifestations (Table 3)
Table 3. Atypical manifestations in patients with pituitary adenoma.

<table>
<thead>
<tr>
<th>Manifestation</th>
<th>No of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arcuate scotoma</td>
<td>1</td>
</tr>
<tr>
<td>Isolated nasal U/L or B/L field loss</td>
<td>2</td>
</tr>
<tr>
<td>Papilloedema</td>
<td>1</td>
</tr>
<tr>
<td>Motor abnormalities</td>
<td>2</td>
</tr>
<tr>
<td>Pituitary apoplexy</td>
<td>7</td>
</tr>
</tbody>
</table>

Field defects:

1. Right nasal hemifield loss (Figure 1a): In one of the patients the tumour was encasing the cavernous portion of the right carotid artery compressing the optic chiasma from right lateral side. (Figure 1b). This might have affected the optic nerve fibres coming from the ipsilateral temporal retina of right eye leading to right nasal hemianopia. Other eye was normal.

Arcuate scotoma: One patient had double arcuate scotoma in both eyes (Figure 2a). Here the tumour was encasing the intracranial portion of both optic nerves more so on right optic nerve (Figure 2b). This compression may have produced these scotomas characteristic of optic nerve involvement. This is also substantiated by the presence of optic nerve head (ONH) pallor in both eyes.

Incidently this patient also had left hyperemic optic disc. This patient had been diagnosed elsewhere as, optic neuritis in view of the field defects and optic disc hyperemia and treated with intravenous dexamethasone.

Bilateral superonasal quadrantanopia (Fig 3): This implies compression from below affecting the temporal fibers first causing nasal visual field defect

Motor abnormalities:

V1th nerve paresis: A 13 year old boy presented with vomiting, headache and double vision since 1 month. Double vision was maximum on looking to the right side. He was found to have right V1th nerve paresis. On MRI there was a sellar lesion with suprasellar extension, invading right cavernous sinus. (Figure 4) This paresis and diplopia completely disappeared after the surgical removal of the tumour.

Superior orbital fissure syndrome with partial III nerve palsy: This patient presented with headache for 2 weeks duration and on examination showed partial third nerve palsy on the right side. The vision, pupils and visual fields were normal, and on MRI, had enlarged pituitary and diagnosed as incidentaloma.
He also had a soft tissue enhancing mass in the right superior orbital fissure (Figure 5). A diagnosis of right superior orbital fissure syndrome was made and nerve palsy recovered well with oral steroids. Probably the two masses were unrelated.

1. Pituitary apoplexy with total ophthalmoplegia: One of the patients of apoplexy presented with left complete IIIrd, IVth and VIth nerve palsy. After transnasal endoscopic resection of the tumour, over a period of 7 months, these recovered completely. Another patient with apoplexy had restricted elevation in both eyes.

Pituitary apoplexy: Out of the 58 cases reviewed, we found 7 cases (10.53%) of pituitary apoplexy. Their manifestations included sudden onset of severe visual loss, altered sensorium, altered behaviour headache etc. are summarized in Table 4. The time period between onset of symptoms and diagnosis of apoplexy ranged between 2 weeks to seven months. All these patients had very good outcome after the surgical resection of tumour, except for 2 patients (one had residual tumour, and the other had gross compression of optic nerve by the tumour).
Table 4. Clinical features of patients with pituitary apoplexy

<table>
<thead>
<tr>
<th>Clinical feature</th>
<th>No of patients (%)</th>
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<tbody>
<tr>
<td>Decreased vision</td>
<td>5 (60%)</td>
</tr>
<tr>
<td></td>
<td>[total loss 5 eyes]</td>
</tr>
<tr>
<td>Motor nerve palsies</td>
<td>1 (20%)</td>
</tr>
<tr>
<td>Headache</td>
<td>7 (100%)</td>
</tr>
<tr>
<td>Vomiting</td>
<td>3 (50%)</td>
</tr>
<tr>
<td>Altered sensorium</td>
<td>3 (50%)</td>
</tr>
<tr>
<td>Altered behaviour</td>
<td>1 (20%)</td>
</tr>
</tbody>
</table>

In one patient of Stevens Johnson syndrome, vision did not improve inspite of good recovery of cornea and tear film. Best corrected visual acuity was 6/12, with Optic Nerve Head pallor in right eye. He was advised to continue tear substitutes and review after 2 weeks for visual field assessment when his systemic condition would be better. On the 3rd day after being discharged from the hospital, he came to the casualty with sudden complete loss of vision in right eye and was found to have pituitary apoplexy (Figure 6).

One patient had papilloedema with blurring of disc margins and a history of frank papilloedema as per her records.

Fig. 3. Bilateral superonasal quadrantanopia

Fig. 4. Pituitary tumour invading right cavernous sinus
in height, and width. Enlargement of the pituitary < 10 mm is called Microadenoma. An enlargement more than this is called Macroadenoma. Because of its anatomical relation with the chiasma [Fig. 7], the pituitary tumors cause VF defects by compressing the ON fibres.

The VF defects are the mirror images of the ON defects i.e. since compression is from below scotomas first appear in the temporal hemi field as the crossed fibres are most susceptible, especially the inferior ones. Thus the VF seems to progress from above – below i.e. clockwise in the RE and anticlockwise in the LE. These defects may be complete / incomplete/ partial/ total dense. Chiasma

**Discussion**

Tumours of pituitary gland are extremely important for an ophthalmologist due to their various ophthalmic manifestations.

Pituitary gland is situated in the sella tursica. The pituitary adenoma is its most common involvement [90 %]. The normal size of pituitary is $5.4 \pm 0.9$ mm
can be prefixed [10%] or postfixed [10%] [Fig. 8]. VF loss patterns are different in these cases. In prefixed chiasma, optic tract would get affected first, i.e. homonymous hemianopic pattern of VF loss and in postfixed either one of the optic nerves may be affected more, with VFD of one eye more, and an altitudinal pattern.

There is a significant change in the way pituitary tumours come to attention. Hollenhorst and Young reviewed 1000 cases of pituitary tumours between 1940 to 1962 and found that 70% of these patients had either loss of visual acuity or visual defects or both, but in a subsequent study of the patients between 1971 to 1982 at the same hospital, only 20% patients had reduced visual acuity and 32% had visual field defects. In the case series studied by Anderson et al (200 consecutive cases of pituitary tumours between 1971 to 1982) only 16% patients had decreased visual acuity and 32% had visual field defects. So there is a decline in the number of patients with visual problems in pituitary adenomas.

In our case series, 66 eyes out of 114 [57 patients] had normal VA. Also only 16 patients (28.1%) had normal visual fields in both eyes. This reflects the need of vigilance by the ophthalmologists in our country to consider the possibility of pituitary tumour in dealing with patients of unexplained visual loss.

Bitemporal hemianopia has been described as the classical field defect in pituitary tumours. It was found in only 10 patients (17.5%). Depth perception difficulties may be seen in patients with bitemporal field defects like difficulty in clipping nails, threading, catching a ball because of the blind triangular area just beyond fixation in these patients [Fig. 9]. None of our patients had these complaints. Different kinds of field defects were found like one eye hemianopia and contralateral superotemporal quadrantanopia; one eye blind and contralateral temporal hemianopia; bilateral superotemporal quadrantanopia etc. This emphasizes that pituitary tumours cause not only bitemporal hemianopia but other kind of field defects also and an ophthalmologist should be aware of these. As the tumor grows the visual field defects keep extending. Nonsecreting tumors are detected later when the size is larger, as only visual symptoms may be the presenting feature.

One of our cases had an arcuate scotoma. Earlier arcuate field defects have been reported in patients with pituitary tumours. These have been ascribed to the damage to intracranial portion of the optic nerve, with postfixed chiasmas. Superior arcuate field defect can occur with a postfixed chiasma as, an optic nerve [ON] rather than chiasma, lies over the sella turcica.
compressing the ON from below and causing a superior arcuate defect.

Walsh & Hoyt\(^1\) writes, that in rare cases when tumour grows between two optic nerves shifting them laterally, it may compress optic nerves against the anterior clinoid process and internal carotid arteries, affecting the temporal nerve fibers first, giving rise to bilateral nasal field defects. We propose a similar explanation for our patient with right nasal hemianopia where tumour was found to encase right internal carotid artery, which might have compressed the temporal fibres in right optic nerve giving rise to a nasal hemifield loss.

Many explanations are given to explain the reason for bitemporal hemianopia as the first field defect in pituitary lesions. Fibers from inferior parts of retina are situated inferiorly in optic chiasma which get compressed first by the pituitary tumour. So it should produce a superior altitudinal field loss, but actually it affects temporal field preferentially. This has been explained by the susceptibility of nasal fibers to effects of compression, or it may be related to blood supply of crossing fibres at chiasma.\(^1\) In our case which showed bilateral superonasal quadrantanopia, we could not offer any explanation by the neuroimaging study. The cause could either be the fragile blood supply of the crossing fibres (as demonstrated by Lao and Gao in cases where bitemporal hemianopia was present without any chiasmal compression) \(^9\) or fine mechanics of compression selectively affecting the crossing fibres.

Papilloedema can occur as a part of Foster Kennedy Syndrome [very rarely seen with pituitary lesions] or due to hydrocephalus with compression of the foramen of Monro. Our patient had only blurred disc margins. She had come after excision of the pituitary mass to our institution for radiotherapy and carried reports that indicated papilloedema prior to surgery, due to hydrocephalus with compression of the foramen of Monro as per the CT scans available.

Pituitary tumours causing abnormalities of extraocular movements are rare. In Hollenhorst and Young's review, they found 4.6 % patients \(^4\) while Trautman et al found 1.4 % patients with abnormalities of extraocular movements \(^10\). We found 4 such patients [7.2 %] in our study with a smaller sample size. This number is slightly higher compared to the earlier studies. Isolated involvement of VIth nerve is also rare with very few cases reported so far.\(^11,12,13\) We found one such patient. It was ascribed to invasion of cavernous sinus by the tumour affecting the VIth nerve.

Other unusual presentations not seen in our series are hemifield slide phenomena, visual hallucinations, junctional syndrome, homonymous hemianopia and see saw nystagmus.

The patient who had superior orbital fissure syndrome, had only a microadenoma and probably his motor manifestations were not related to the pituitary tumour. But there was a soft tissue mass in superior orbital fissure. This emphasizes the need for a careful examination of the neuroimaging, which may reveal other coexisting abnormalities which might be responsible for some of the clinical features of the patient and not get biased by the tumour itself.

Pituitary apoplexy is a rare, major clinical event with neurological, neuro-ophthalmological, cardiovascular and hormonal consequences, resulting from an acute infarction of pituitary adenoma. There has been confusion about the exact definition of the entity. Walsh and Hoyt suggest that this term should be used for cases with acute haemorrhage into pituitary adenoma producing, not only infarction of the gland but also damage to adjacent structures particularly optic nerves, optic chiasma, ocular motor nerves and hypothalamus.

We found 7 (12.3 %) cases with apoplexy according to the above definition. About 5 % patients are said to suffer from this complication \(^2\). Headache was the commonest symptom followed by decreased vision. This finding is similar to the earlier reports \(^14-17\). But compared to these studies, the incidence of motor palsies was less in our series. We had a 13 year old boy with apoplexy, although it is rare in children \(^1\).

Except for 3 patients with apoplexy, others had a good outcome after trans-sphenoidal resection of the tumour. One of these cases with poor postoperative outcome, had residual tumour, and two had direct compression of optic nerve by the tumour. Histopathologically these 3 cases were found to have haemorrhagic infarction. According to a study by Semple PL, De Villiers JC et al, patients who presented with histological features of pituitary tumor infarction alone had less severe clinical features at the time of presentation, a longer course prior to presentation and a better outcome than those presenting with hemorrhagic infarction or frank
hemorrhage.\textsuperscript{18} Our observation of poor outcome in these patients also corroborates with this.

4 of these patients with apoplexy were treated earlier for altered consciousness, abnormal behaviour, hyponatremia etc. and later were found to have apoplexy. Agrawal D and Mahapatra AK in their study have shown that even completely blind eyes may have remarkable improvement in vision if surgical decompression of the optic apparatus is undertaken early.\textsuperscript{19}

**Conclusion**

Still significant numbers of patients with pituitary tumours either present with or have associated decrease in visual acuity or visual field defects. Although bitemporal hemianopia is the classical visual field defect caused by pituitary tumours, other variants of field defects also occur. Field defects like arcuate scotomas, isolated nasal field loss, although uncommon, can occasionally be seen and their presence does not rule out pituitary tumours per se. Looking at the need for early surgical intervention for a better outcome, it is very important both for an internist and an ophthalmologist to keep in mind the entity - pituitary apoplexy (in patients with headache, visual loss and altered sensorium), in the appropriate clinical setting.

**Acknowledgements**

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**References**

Visual Field Progression in Primary Open Angle Glaucoma in Patients With Unilateral Field Loss

Dr. Vijaya Pai H MS, Dr. R Anjana Devi MBBS

Aim: 1) To study the visual field progression in patients with unilateral visual field loss due to POAG. 2) To determine the risk factors for progression.

Materials and Methods: 31 patients with POAG with unilateral field loss by Anderson's criteria were followed up with Humphrey's Field Analyzer. Variables noted were age, duration of disease, duration of follow up, number of visual fields, initial C: D ratio, mean IOP, MD [dB], PSD for both eyes and AGIS score for first affected eyes. The criteria for progression were modified from Anderson, after taking into account the initial AGIS score.

Results: Progression of the field loss was noted in 11 (35.48%) first affected eyes over an average period of 22.72 ±10.22 months and three (9.67 %) fellow eyes over an average period of 19.33 ± 13.31 months. The duration of follow-up was found to be significantly associated with progression in first affected eyes and no variable was significant in case of fellow eyes. Risk of progression estimated by Kaplan-Meier survival analysis as 46.74% at 3 years for the first affected and 15.38% at 4 years for the fellow eyes.

Conclusion: The risk of progression of the field defect in the fellow eyes without field loss is low compared to that in the eyes with field loss. None of the variables investigated were found to be significantly associated with progression in the fellow eye.

Key words: POAG, FELLOW EYE, UNILATERAL FIELD LOSS

Introduction

Primary open angle glaucoma (POAG) is the common form of glaucoma, affecting 1 to 2% of the world population. 1 Our understanding of the pathophysiologic events that result in the optic atrophy and visual field loss is imperfect. POAG is a bilateral disease of adult onset1. In patients with visual field loss in one eye, the fellow eye is at high risk for developing visual field defects because the patients may have some systemic susceptibility factors. 2

We studied the progression of visual fields in POAG patients with unilateral field loss with automated perimetry. We also sought to identify any risk factors for progression.

Materials and Methods

Patients seen at the Out Patient Clinic of the Department
of Ophthalmology, from January 2000 to May 2005 were included in the study.

The inclusion criteria were:

1. Patients with Primary Open Angle Glaucoma.
2. The visual field of one eye abnormal by Anderson's criteria.
3. The visual field of contralateral eye not meeting Anderson's criteria.
4. Patients with a minimum follow-up of 1 year with Humphrey visual fields.

The exclusion criteria were:

1. Secondary glaucomas.
2. Visual field loss due to causes other than glaucoma.

Age and sex of the patient were noted. A detailed history and the presenting symptoms were recorded. The duration of the disease, treatment taken and history of any ocular surgeries were noted. Any history of trauma to the eye and history of medications like topical or systemic steroids were noted. Presence of systemic diseases like Diabetes Mellitus, Hypertension and Cardiovascular disease was noted. Family history of glaucoma was also noted.

Visual acuity and anterior segment findings including the presence of cataract, IOP, diurnal variation of IOP and gonioscopic findings were recorded. In fundus examination, cup: disc ratio and other glaucomatous changes like notching, peripapillary atrophy etc. were noted. Any other fundus abnormalities were also noted.

HFA 30-2 SITA was done and HFA 10-2 was performed in patients field loss. Visual field data like mean deviation [MD] and pattern standard deviation [PSD] were recorded and the AGIS score of the visual field noted. The criteria used to determine the abnormality was the Anderson's criteria. A visual field test was considered abnormal if any two of the following three criteria were met on at least two consecutive visual field tests:

1) A Glaucoma Hemifield Test outside normal limits.
2) A cluster of three or more non-edge points in a location typical for glaucoma all of which are depressed on the pattern deviation plot at a p < 5 % level and one of which is depressed at a p < 1 % level.
3) A corrected pattern standard deviation p < 5 %.

Since in the SITA program the short-term fluctuation and the CPSD are not available the pattern standard deviation was substituted as criteria in making the diagnosis. For abnormal visual fields, the criteria for progression were modified from Anderson, after taking into account the initial Advanced Glaucoma Intervention Study (AGIS) score, as done in a previous study by Chen and Park. This was done to compensate for the higher level of fluctuation seen in eyes with more advanced glaucomatous damage. For eyes with an initial AGIS score of 5 or less (mild visual loss by AGIS classification), the progression was defined as three adjacent points depressed 5 dB or more from the initial level of loss on the total deviation plot, with at least 1 point depressed 10 dB, on two consecutive fields. For eyes with an initial AGIS score of 6 or more, progression was defined as three adjacent points depressed 10 dB or more from the initial level of loss on the total deviation plot, on two consecutive fields.

Patients were followed up according to the severity of the glaucoma (every 3-6 months). During follow-up the medications used, visual acuity, IOP and cup: disc ratio were recorded. Visual fields were examined to assess progression in the affected eyes and development of field loss in the normal eyes. Time taken for visual field progression was also noted. In the normal eyes where there was no field loss, SWAP was done wherever possible.

The variables noted were age, duration of the disease in years, follow-up in months, number of visual fields, mean IOP, initial characteristics like cup: disc ratio, MD,PSD for both eyes and AGIS score for the first affected eye. The IOP's at the time of follow-ups were averaged to derive the mean IOP.

Variables were compared between the eyes with and without progression using independent samples two-tailed ‘t’ test. The correlation between the progression of the field defect in the first affected and the fellow eyes was assessed by Spearman correlation. Kaplan-Meier survival analysis was used to estimate progression in first and fellow eyes. Cox proportional hazards regression analysis with forward stepwise variable selection was also used to evaluate variables for association with progression. A statistical spreadsheet software program was used for all calculations (SPSS 10.0).
Results

62 eyes of 31 patients with unilateral field loss from POAG who attended our hospital from January 2000 to February 2005 were studied. The study group included 18 males and 13 females. Out of the 31 first-affected eyes, 18 were affected in the left eye and 13 in the right eye. Family history of glaucoma was present in one patient. Demographic data of the patients studied is given in Table 1.

Progression of field loss was noted in 11 (35.48 %) of 31 first affected eyes. The average time to progression was 22.72 ± 10.22 months.

Among the variables investigated the duration of follow-up (in months) was found to be significantly associated with progression (P = 0.015). Risk of progression estimated by Kaplan-Meier survival analysis was found to be 46.74 % at three years. The distribution of the IOP and C:D ratio in the first affected eyes is shown in chart 1 and 2. The difference between the variables of the progressing eyes and the stable eyes is given in Table 2.

Three out of 31 fellow eyes (9.67 %) were found to have progression of the visual field defect. The average time to progression was 19.33 ± 13.31 months. No significant differences were found between stable and progressing eyes among the variables investigated. Kaplan-Meier survival analysis estimated the risk of progression to be 15.38 % at four years. The distribution of the IOP and C:D ratio in the fellow eyes is shown in chart 3 and 4. The difference between the variables of the progressing eyes and the stable eyes is given in Table 3.

Three out of 31 fellow eyes (9.67 %) were found to have progression of the visual field defect. The average time to progression was 19.33 ± 13.31 months. No significant differences were found between stable and progressing eyes among the variables investigated. Kaplan-Meier survival analysis estimated the risk of progression to be 15.38 % at four years. The distribution of the IOP and C:D ratio in the fellow eyes is shown in chart 3 and 4. The difference between the variables of the progressing eyes and the stable eyes is given in Table 3.

Bilateral progression was noted in one patient (3.22 %). 18 patients (58.06 %) were stable bilaterally. The correlation between the visual field progression in the first affected and fellow eye was not significant (P=0.937).

The number of antiglaucoma drugs used in the first affected and fellow eyes is given in table 4. Nine patients did not receive treatment in the fellow eye. None of them had visual field progression in the same eye.

| Table 1. Demographic Data and Characteristics of the Patients studied, expressed as Range (Mean + SD) |
| Age (In years) | 28-77 (57.12 +/- 12.08) |
| Duration of disease (years) | 1-14 (3.86 +/- 3.09) |
| Follow-up (months) | 12- 50 (28.19 +/- 10.43) |
| Number of visual fields | 2- 12 (5.7 +/- 2.5) |
| Ocular Characteristics | First eye |
| Initial C: D ratio | 0.4-0.9 (0.75 +/- 0.12) |
| Mean IOP | 10-30 (16.37 +/- 4.2) |
| Mean Deviation (dB) | -30.28 to -0.4 (-10.72 +/- 10.03) |
| PSD (dB) | 2.53-16.68 (8.09 +/- 4.5) |
| AGIS Score | 1 - 20 (8.13 +/- 7.12) |
| Fellow eye |
| Initial C: D ratio | 0.3-0.9 (0.56 +/- 0.16) |
| Mean IOP | 10-22 (15.21 +/- 2.65) |
| Mean Deviation (dB) | -3.45 to 2.18 (-0.73 +/- 1.2) |
| PSD (dB) | 1.33 – 5.88 (2.66 +/- 1.14) |
Three patients underwent combined surgery in the first affected eye. Four patients underwent trabeculectomy in both eyes. Five patients could discontinue antiglaucoma medication after the surgery. In two patients who underwent combined surgery, antiglaucoma medication was restarted, the reason being inadequate control of IOP in one and progression of visual field defect in the other patient.

Initial C: D ratio (P = 0.00), Mean Deviation (P = 0.00) and Pattern Standard Deviation (P = 0.00) were found to be significantly higher in the first affected eye when compared to the fellow eye. Worsening of final central acuity by more than 2 lines was noted only in one of 62 eyes, which was a first affected eye.

**Discussion**

Previous studies regarding the incidence of visual field loss in the fellow eyes of POAG showed that the level of visual field loss was higher than that seen in patients with ocular hypertension.

Harbin et al \(^7\) reported that 9 of 21 (43 %) fellow eyes of patients with POAG developed glaucomatous visual field loss over 4.4 years. Kass et al \(^8\) found 9 of 31 (29 %) fellow eyes developed visual field loss over a 3 to 7 year period. Susanna et al \(^2\) calculated progression of 25 % over 5 years in fellow eyes with median follow up of 3 years. Olivius and Thorburn \(^9\) found 25 % of unilateral glaucoma becoming bilateral in five years. Chen and Park \(^6\) calculated the progression in fellow eyes to be 6.3 % over an average period of 37 ± 9 months.

In another study by Chen PP and Bhandari A \(^10\), assessed the fellow eye prognosis in patients with severe visual field loss in one eye from chronic open angle glaucoma, and found that 6 of 36 fellow eyes (17 %) had significant visual field progression. In another study by Chen PP \(^11\), he studied correlation of visual field progression between the two eyes of patients with open-angle glaucoma, and found that 24.3 % of the better eyes progressed. In our study, the fellow eye progression was found to be 9.67 % over an average time of
19.33 months, which is corresponding to Chen et al’s studies. The older studies were based on Goldmann fields. The studies by Chen PP, and our study were based on visual fields by Humphrey’s Field Analyser.

In the present study, risk of progression in the fellow eye estimated by Kaplan-Meier survival analysis was 15.38% at 4 years. This was comparable with previous studies. Chen and Park estimated 7.2% at 5 years; Chen and Bhandari 12.4% at 5 years and Chen 33% at 10 years.

In our study, the progression in the first affected eye was 35.48% over an average period of 22.72 months. Hart and Becker showed that 73% of their patients with glaucomatous visual field defects progressed during the course of their disease. In the study by Mikelberg et al, 76% patients showed progression during the follow-up period. Harbin et al found in his study of 21 patients with monocular field loss, that 16 of the 21 eyes (76.19%) already had field loss, progressed over a period of 4.4 years. In recent studies, Chen and Park found progression of the first affected eyes in 21% patients; Chen and Bhandari in 33% and Chen PP in 35.5% patients.

In our study, risk of progression of the first affected eye as estimated by Kaplan-Meier survival analysis was 46.74% at 3 years, which was high compared to that estimated by Chen and Park (25% at 5 years) and Chen (44% at 10 years).

The higher incidence of progression compared to the previous studies may be partly due to differences in the patient populations like racial composition and educational level.

The number of untreated fellow eyes in this study (8 of 31 eyes, 25.81%) was similar to that in the study by Kass et al (19%) and Chen and Park (21%). No progression was seen in untreated eyes, in all the three studies.

In our study, the severity of initial visual field loss was measured by AGIS score unlike older studies, which generally assessed severity of visual field loss by broad grading schemes. AGIS score of the initial visual field in the first affected eye was found not to be significantly associated with progression (P = 0.443) unlike Chen and Park’s study. Some studies have shown that visual field progression is more in patients with advanced visual field damage, some have found progression to be more likely in eyes with less damage and some have found progression unrelated to the initial level of damage.

Previous studies have found the risk factors for progression in the fellow eyes to be elevated IOP, larger cup/disc ratio, and optic disc hemorrhage. In our study, no variables were found to be significantly associated with progression. Chen and Park noted a similar observation.

In our study, among the variables investigated, only the duration of follow-up (in months) was found to be significantly associated with progression (P= 0.15) in the first affected eyes. The more we follow up a patient,

### Table 3

<table>
<thead>
<tr>
<th>Variables in fellow eyes with and without progression (Mean +/- SD) and their association with development of field defect (P value)</th>
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<tbody>
<tr>
<td><strong>Progressing eyes</strong></td>
</tr>
<tr>
<td>Age (years)</td>
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<tr>
<td>Duration of disease (years)</td>
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<tr>
<td>Follow-up (months)</td>
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<tr>
<td>Cup: Disc ratio</td>
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<tr>
<td>Mean IOP (mm Hg)</td>
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<tr>
<td>Mean deviation (dB)</td>
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<tr>
<td>Pattern SD (dB)</td>
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### Table 4

<table>
<thead>
<tr>
<th>Medical and surgical treatment in the first affected and fellow eyes</th>
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<tbody>
<tr>
<td><strong>No. of eyes on 1 drug</strong></td>
</tr>
<tr>
<td>First affected eyes</td>
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<tr>
<td>Fellow eyes</td>
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the more the chance for detecting progression. Some of the previous studies have found IOP \cite{8,15,17-19}, cup: disc ratio \cite{17,20-23}, systemic diseases like diabetes \cite{24}, hypertension \cite{25} to be associated with progression and some studies have found them to be not significant \cite{26-31}. The cup: disc ratio was found to be significantly high in the first affected eye than the fellow eye. But the initial cup: disc ratio was not found to be associated with the development of field loss in the fellow eye. This may mean that other patient related factors (like decreased blood velocity in retrobulbar vessels) \cite{32} may be contributing to development of field loss.

**Conclusion**

The fellow eyes of patients with initially unilateral field loss were found to be at high risk for developing visual field defects in previous studies \cite{2,7,8,33}. The risk of development of visual field loss in the fellow eyes was found to be significantly higher than that in ocular hypertension. This led to the current practice patterns of treating both the eyes in POAG, even if one eye has high IOP and field loss and the other eye has only high IOP and no field loss.

Recent studies \cite{6,10,11} have shown that the risk of development of field loss in the fellow eye is low. This improved prognosis may be due to the current practice patterns.

Our study confirms that the risk of progression in fellow eyes without field loss is low compared to that in the eyes with field loss. So the presence of field loss is a risk factor for further progression. The progression was not associated with the severity of initial field loss measured by AGIS score. There was no correlation between the progression of the field defect between the two eyes. None of the variables investigated were found to be significantly associated with progression in the fellow eye. In the first affected eye only the duration of follow up was found to be significantly associated with progression.

**References**


Incidence of Normal Tension Glaucoma in Fellow Eyes of Unilateral Central or Hemi Central Vein Occlusions

Dr. S.J. Saikumar MS, Dr. A. Giridhar MS, Dr. Mahesh G MS DNB FRCS Dr. Sandhya N MS

Purpose: To study the glaucoma profile of fellow eyes in unilateral CRVO or Hemi-CRVO, and to assess the incidence of normal tension glaucoma in such eyes.

Methods: Observational case series evaluating vision, IOP, CD ratio and Automated Perimetry.

Results: Among 75 fellow eyes, 13.3% had IOP > 21 mm Hg and 29.3% had glaucomatous field defects. The risk of field loss in the opposite eye was 5 times in ischemic CRVO when compared with non ischemic CRVO. This difference was statistically significant (chi square test, p value = 0.07).

Conclusion: We found a significant number of previously undetected glaucomas in our study, out of which 63% had normal tension glaucoma. Glaucoma screening of the normal eye is mandatory in persons with unilateral CRVO or Hemi-CRVO.

Introduction

It was Verhoeff in 1913 who postulated a role for glaucoma in the pathogenesis of Central Retinal Vein Occlusion (CRVO). Since then, many investigators have described the association between CRVO and POAG or ocular hypertension. Some authors have also described the association between CRVO and Pseudoexfoliation. The prevalence of POAG in eyes with CRVO ranges from 6 to 69%. Moore in 1922 noted that when CRVO develops, the IOP drops by an average of 18% as compared to the fellow normal eye. Since then, many investigators have noted this fact. In many cases, perimetry cannot be performed in the affected eye because of poor vision. Even in cases where perimetry can be performed, field changes due to the vascular occlusion itself may mask glaucomatous changes. So quite often, the fellow eye has to be investigated to establish the presence or absence of POAG.

In this observational case series, we studied the glaucoma profile of the fellow uninvolved eye including vision, IOP, CDR and Automated Perimetry.

Materials and methods

75 patients with unilateral CRVO or unilateral Hemi CRVO at initial presentation were included in the study. Standard criteria for diagnosis of CRVO and Hemi CRVO were employed.

Diagnostic criteria for CRVO and BRVO:

CRVO consists of 2 distinct clinical entities, namely non ischemic and ischemic. The distinction was based on visual acuity, RAPD, Indirect Ophthalmoscopy and
Fundus Fluorescein Angiography if necessary. Detailed explanations regarding classification and criteria are discussed elsewhere. \(^{12,13,14}\)

Hemi CRVO is a variant of CRVO. In these eyes there are 2 trunks for the central retinal vein instead of one, and only one of them gets thrombosed. Hemi CRVO also consists of ischemic and non ischemic types. It is important to differentiate between Hemi CRVO and BRVO.

**Diagnostic criteria for OHT, POAG and NTG:**

Ocular Hypertension: (1) IOP > 21 mm Hg (2) Open anterior chamber angle (3) No ocular or systemic features suggestive of secondary glaucoma (4) normal optic disc (5) Normal visual fields.

POAG: (1) IOP > 21 mm Hg (2) Open anterior chamber angle (3) Visual field defect consistent with glaucoma (4) Corresponding glaucomatous cupping in the optic disc (5) No ocular or systemic features suggestive of secondary glaucoma.

Normal-tension glaucoma: (1) IOP 21 or lower (2) Open anterior chamber angle (3) Visual field defect consistent with glaucoma (4) Glaucomatous cupping of optic disc (5) Nothing to suggest secondary glaucoma (6) No neurological causes which can cause field defects.

**Exclusion Criteria**

Only unilateral cases of CRVO or Hemi-CRVO were included in this study. All patients with rubeosis or angle new vessels were excluded. All cases of Branch vein occlusion and macular venous occlusions were excluded. Cases where perimetry could not be done in the unaffected eye due to other causes were also excluded from the study.

**Examination Protocol**

All patients presenting with unilateral CRVO were examined in detail using a pre-designed protocol. History of use of anti-glaucoma medications was elicited. Salient features of the examination protocol included visual acuity using Snellen’s chart, IOP of affected and normal eye using Goldman Applanation Tonometer, detailed retina examination using both Indirect Ophthalmoscopy and Slit-lamp Biomicroscopy, and FFA as and when indicated. The presence or absence of Relative Afferent Pupillary Defect (RAPD) was looked for in all cases. When there was suspicion of narrow angles or presence of rubeosis, a careful gonioscopy using the three-mirror Goniolens was performed. Since we did not have access to ERG, this was not performed in any of the cases in this study.

We felt that many patients were quite upset during their first consultation because of the sudden onset of defective vision. Hence Automated Perimetry was performed at a later date, preferably within a week. In cases where the IOP was normal and the perimetry showed glaucomatous field defects, a diurnal variation was performed to decide whether they should be classified as POAG or NTG.

**Classification of field defects**

Early defects: have any one of the following
1. The MD is better than – 6 db
2. Fewer than 18 of the 76 points in 30-2 are defective in TD plot at the 5 % level
3. Fewer than 10 points are defective at the 1 % level
4. No point in the central 5 degrees has a sensitivity < 15 db

Moderate defects: A moderate defect exceeds one or more of the criteria required to keep it in the early defect category, but does not meet the criterion to be severe.

Advanced defect: have any one of the following
1. An MD index worse than -12 dB
2. More than 37 (50 %) of the points depressed at the 5 % level in a 30-2 field
3. More than 20 points depressed at the 1 % level
4. A point in the central 5 degrees with 0 dB sensitivity
5. Points closer than 5 degrees of fixation under 15 dB sensitivity in both the upper and lower hemifields.

These criteria have been advocated by Anderson and Patella. \(^{23}\)

**Data Analysis**

Risk factor analysis was done to predict risk factors for development of glaucoma. The chi square was used for statistical analysis.

**Results**

This study was conducted between January and December of 2004. 75 patients satisfied the inclusion
criteria. 52 patients were males and 23 were females. Only 16 patients (21.3 %) were below the age of 50. 63 patients had CRVO and 12 had hemi CRVO. In 37 patients the right eye was affected and in 38 patients the left eye was affected.

20 patients had hypertension alone, 13 had diabetes alone and 18 had both diabetes and hypertension. Only 6 patients among the 75 had a previous history of glaucoma and all of them were on anti-glaucoma medications. Visual acuity in the affected eye was 6/60 or less in 29 patients and better than 6/60 in 46 patients. Out of the 29 patients with vision less than 6/60, 25 had RAPD. Those with both RAPD and visual acuity of less than 6/60 were classified as ischemic CRVO (33.3 %). The rest 50 patients (66.6 %) were classified as non-ischemic CRVO. Among the patients with visual acuity better than 6/60, none had RAPD.

Out of the 75 patients, 26 fitted into the criteria for either POAG, NTG or OHT. 6 patients had POAG (8 %), 16 had NTG (21.3 %) and 4 had OHT (5.3 %). 21 among the 63 CRVO patients (33.3 %) and 5 among the 12 Hemi CRVO patients (41 %) had some form of glaucoma. (see Table 1) There was no significant difference in the glaucoma incidence between the CRVO group and Hemi CRVO group.

<table>
<thead>
<tr>
<th>Glaucoma type</th>
<th>Total</th>
<th>CRVO</th>
<th>Hemi CRVO</th>
</tr>
</thead>
<tbody>
<tr>
<td>POAG</td>
<td>6</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>NTG</td>
<td>16</td>
<td>13</td>
<td>3</td>
</tr>
<tr>
<td>OHT</td>
<td>4</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Proportion</td>
<td>21/63</td>
<td>5/12</td>
<td>(33.3 %)</td>
</tr>
</tbody>
</table>

All patients underwent disc photography and the cup-disc ratio was subjectively analyzed. 27 patients (36 %) had a CD ratio of 0.6 or more. This includes all the 22 patients who had field defects in our study. Only 5 patients with CDR of more than 0.6 had normal visual fields.

Out of the 22 patients with glaucomatous field defects, 10 had early defects, 4 had moderate defects and 8 had advanced field defects. The defects included paracentral scotomas, upper arcuate, lower arcuate and bi arcuate scotomas. In cases where test reliability was not good, the test was repeated after a week and only the reliable test was taken for analysis. 2 patients had unreliable tests even after repetition and so were not included in the field defect group.

Out of the 25 patients who presented with ischemic CRVO in one eye, 11 had field defects in the fellow normal eye. Among the 50 patients with unilateral non-ischemic CRVO, 11 had field defects in the fellow normal eye. Risk factor analysis showed that persons with unilateral ischemic CRVO are 5 times more prone to develop field defects in the fellow normal eye when compared with unilateral non-ischemic CRVO. This difference was found to be statistically significant with a p value of 0.01 in the chi square test.

As mentioned above, there were 26 patients out of the 75 who had some form of glaucoma. But only 6 patients gave a history of glaucoma which means that we picked up as many as 20 new glaucoma patients when they presented to us with CRVO.

**Discussion**

The association between glaucoma and CRVO has been well proven and widely documented. 15,16 This study is an endeavor to study the glaucoma profile of the fellow normal eye for presence of glaucoma. We were successful in picking up 20 new cases of glaucoma from among the 75 patients with unilateral CRVO or Hemi CRVO.

In this study, we have used the fellow normal eye to make a diagnosis of POAG, NTG or Ocular Hypertension. It has been well documented that the IOP of the affected eye drops significantly when CRVO or Hemi CRVO occurs. This IOP drop occurs more in patients with POAG/OHT than in patients who do not have glaucoma. 17 The overall prevalence of POAG plus OHT in our study was 34.6 % which compares well with similar studies. 17,18,19

As early as in 1924 Moore had stated that extensive cupping of the optic disc was a rather common association with CRVO. 10 Similar results were also published by Dobree in 1957. 20 The same findings were proven with histopathology support by Salzmann 21 and Landolt. But all these reports concerned the cupping in the affected eye and not the fellow normal eye. Bertelson 22 found in his 17 patients with CRVO that large cupping was seen in 8 contralateral eyes. We found a CD ratio of 0.6 or more in 27 patients (36 %) among
75. The drawback of using CDR is always the problem of subjectivity. This can be rectified to a large extent by using OCT for measurement of the cup. When this study was conducted, we did not have access to Optical Coherence Tomography.

We did not perform Automated Perimetry in the affected eye since we felt that the results may not be reliable. Hayreh et al 14 have done Goldman Kinetic Perimetry in the CRVO-affected eyes and have used this as one of the criteria to differentiate between non-ischemic and ischemic types of CRVO. They feel that Goldman Kinetic Perimetry is better because it gives a much larger area than the central 30 degrees. We did Humphrey Automated Perimetry of the unaffected eye to pick up even early glaucomatous changes.

Many criteria have been mentioned in the literature for differentiating between ischemic and non-ischemic CRVO. Hayreh et al have mentioned four subjective and two objective criteria. 13 The four subjective criteria are visual acuity, Perimetry, RAPD and ERG. The two objective criteria are FFA and Ophthalmoscopy. The combination of RAPD with ERG was supposed to have the maximum sensitivity and the least reliable criterion is Ophthalmoscopy. We did not have access to ERG and we have used a combination of Visual acuity, RAPD, Ophthalmoscopy and in some cases FFA to differentiate between ischemic and non-ischemic CRVO.

There was no significant difference in the incidence of glaucoma between the CRVO and Hemi CRVO groups. This emphasizes the fact that Hemi CRVO is only an anatomical variation of CRVO and not similar to BRVO. This has also been mentioned in detail in other studies. 17

The percentage of subtypes of glaucoma in this study shows that Normal tension glaucoma (NTG) is the predominant type in our study (63%). This was in drastic variation to the study by Hayreh et al in which Ocular Hypertension was the predominant type followed by POAG and then by NTG. 17 Many factors have been implicated in the pathogenesis of CRVO and they include large cup, raised IOP, sluggish blood flow etc. Among these, large cup and sluggish blood flow may be the factors responsible for CRVO in NTG.

There were a few drawbacks in this study, namely: (1) We did not use ERG to differentiate between ischemic and non-ischemic CRVO. (2) We did not use OCT to objectively assess cup size. (3) We did not use Central Corneal Thickness (CCT) before grouping cases into Ocular Hypertension (OHT).

**Conclusion**

We found a significant number of new glaucoma patients in our study, which includes a surprisingly high number of normal tension glaucomas. The risk of developing glaucomatous damage in the fellow normal eyes is 5 times more in ischemic CRVO when compared with non-ischemic CRVO. We feel that Glaucoma Screening of the fellow eye is mandatory in all cases of unilateral CRVO or Hemi CRVO. If glaucoma or OHT is detected in the fellow normal eye, anti-glaucoma medications must be started to prevent progression of field defects when present, and also to prevent the occurrence of venous occlusion in the normal eye. But it should be stressed here that CRVO is a multifactorial disease and other factors also should be kept in mind when the fellow normal eye is being treated.

**References**

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Effects of a Modified, Purposely Tented Trabeculectomy Over Conventional Trabeculectomy in Glaucoma Patients

Dr. Rani Menon MS DO FRCS

**Aim:** To compare the effects of a modified, purposely tented trabeculectomy over conventional trabeculectomy without releasable sutures in lowering intraocular pressure (IOP) in primary glaucomas.

**Materials and methods:** 53 patients of age 55 to 77 were included in a retrospective nonrandomized clinical study. 26 of them underwent conventional trabeculectomy and 27 underwent modified tented trabeculectomy with a reverse frown incision. IOP and complications were compared in the 2 groups. IOP was measured on 2nd postoperative day, at one month and the final follow up was between 6 and 24 months.

**Results:** The preoperative IOP did not significantly differ between the 2 groups. (29.34 ± 10.90 and 25.62 ± 7.88 in the conventional and tented group respectively). Post operatively on 2nd day IOP was significantly lower in the tented group than in the conventional group (p = 0.0001). IOP more than 18 mm at final follow up was found in 10 of the conventional group and 1 in the tented group ( p = 0.002).

**Conclusion:** Making a tent purposely for aqueous drainage ensures that there is continuous flow of aqueous in one direction and consequently fibrosis is prevented due to the antifibrotic effect of the aqueous itself rather than by the use of antifibrotic agents. A larger area of drainage is obtained because of the reverse frown incision employed in the modified trabeculectomy. This study statistically proves that tented trabeculectomy is a more effective way to control IOP than a conventional trabeculectomy with out releasable sutures.

**Abbreviations:** IOP-intraocular pressure, A/C- anterior chamber

**Introduction**

Tremendous progress has been made in the management of glaucoma. To date conventional filtering surgery remains the mainstay of surgical therapy in the management of glaucoma not controlled by medication. The main reasons for delaying surgery were the high risk of postoperative complications associated with the standard trabeculectomy and failure rates in certain sub groups of glaucoma. Hypotony is a serious and common complication. In 1–2 % of cases it may even persist. Hypotony can lead to flat A/C, corneal edema, cataract, maculopathy and loss of vision. Conventional trabeculectomy fails over time because of fibroblastic proliferation and sub conjunctival fibrosis that occurs during normal healing. Adjunctive use of antifibrotic agents with glaucoma surgery significantly
reduces the risk of bleb failure although their use has been associated with a number of complications. The present study is aimed at evaluating the effect of a modified trabeculectomy technique, tented trabeculectomy technique, and its advantages over a conventional trabeculectomy to achieve a lower and more predictable IOP in primary glaucoma patients.

**Materials and Methods**

**Patients selection**

Fifty three patients of age 55 to 77 were included in a retrospective clinical study at the Amala Institute of Medical Sciences, Thrissur, during the year 2003-2006 as per the guidelines and approval of Ethics committee of the Institute. Twenty six patients underwent conventional trabeculectomy and 27 underwent modified tented trabeculectomy after obtaining their informed consents. All patients were examined pre-operatively for IOP, visual acuity, gonioscopy and visual fields. Secondary glaucomas and paediatric glaucomas were excluded from the study. All the surgeries were done by the same surgeon (author) and under peribulbar anaesthesia.

**Conventional trabeculectomy**

A fornix based conjunctival flap was fashioned and a limbus based triangular partial thickness scleral flap was dissected. The area for trabeculectomy was marked before entering the anterior chamber with a microblade. Paracentesis was then made and viscoelastic injected into the anterior chamber. The area marked out for trabeculectomy was dissected away with the same microblade. Peripheral iridectomy was done and scleral flap sutured with 3 10/0 nylon sutures one at the apex and two on the sides. Conjunctiva was closed watertight with 8/0 running suture. Anterior chamber was reformed. Releasable sutures were not used.

**The tented trabeculectomy**

A fornix based conjunctival flap is taken and a 4 x 4 mm partial thickness rectangular shaped scleral flap is created. The posterior incision of the scleral flap is curved towards the surgeon (reverse frown, Fig 1 a). The scleral flap is tunnelled with a crescent shaped microblade. The sides of the scleral flap were not opened all the way to the limbus or as far as sufficient to make the trabeculectomy. Paracentesis was done and a trabeculectomy with peripheral iridectomy was done using the microblade as in the conventional method mentioned above. Suturing is done in such a way that a tenting is made purposefully. First the sides of the scleral flap were sutured where they are open and the posterior edges of the scleral flap are brought medially and sutured to the centre of the curved incision, (Fig 1 b) so that a tent is formed (Fig 1 c). The curving of the scleral flap in a reverse frown ensures a larger area of drainage. Conjunctiva is sutured water tight with a running suture.

All patients were given a combination of antibiotic (Ofloxacin) and prednisolone acetate along with atropine. No antimetabolite was used in either method. IOP was measured on first and second postoperative day, at one month and between 6 and 24 months. Hypotony was defined as IOP readings less than 6 mm of Hg obtained on two successive readings postoperatively (Sang & Murthy 2006). Iridocorneal touch was defined as an apposition of iris or lens to the cornea. Shallow anterior chamber was defined as any other situation with a reduced anterior chamber depth. Choroidal detachment was recorded as present or not present but not graded further.

(A) Reverse frown incision of scleral flap with the side incisions not reaching the limbus; (B) Posterior edges of the flap are sutured to the middle of the curved incision, and (C) Final appearance showing the tent.
**Statistical analysis**

Intraocular pressure values are expressed as mean ± SD. All statistical analysis was performed using Graphpad Instat software programme. Statistical significance between mean value of preoperative and postoperative IOPs in both conventional and tented trabeculectomy were analyzed by one way analysis of variance (ANOVA) followed by Turkys-Kramer multiple comparison tests. Significant difference between mean value of IOP of conventional and tented trabeculectomy were done by unpaired t-test Postoperative complications were analysed by Chi-Square test. Value of p<0.05 was considered significant.

**Results**

The mean age of patients in the conventional group was 66.5 ± 11.4 and in the tented group was 65.0 ± 11.0. The type of glaucoma found in the conventional and tented groups were 25/1 (open/narrow) and 26/1 (open/narrow) respectively, which was not statistically significant (p = 0.978). IOP was measured on 2nd post-operative day, 1st month, and between 6-24 months after the surgery. The pre-operative IOP in both the conventional group and the tented trabeculectomy group did not significantly differ from each other (p = 0.08) (Table 1). The pre-operative IOP in the conventional group was 29.34 ± 10.90 and in the tented trabeculectomy group 25.62 ± 7.88.

IOP was significantly reduced in both the conventional and tented group on all post-operative days with respect to the pre-operative IOP (p<0.05) (Table 1). All the post-operative IOPs in the tented group was also found to be significantly lower than that of the conventional group. However the mean IOP measured between 6 to 24 months in tented trabeculectomy (11.96) was found to be lower than that of the conventional group (15.50) (Table 1).

The immediate post-operative complications such as shallow A/C did not significantly differ between both the groups. (p = 0.574, Table 2). Only 1 patient out of 26 patients in the conventional group and 2 patients out of 27 in the tented trabeculectomy group was found to have shallow anterior chamber in the immediate postoperative period. Similarly, choroidal detachment was noted in 1 in the conventional and 3 in the tented trabeculectomy group (p = 0.317). Post-operative hypotony was noted in 1 in the conventional and 2 in the tented group (p = 0.575). The shallow A/C noted in both groups was associated with hypotony on the

| Table 1. Intraocular pressure (IOP) in patients treated with conventional and tented trabeculectomy. |
|---------------------------------------------------|---------------------------------------------------|---------------------------------------------------|
| Conventional trabeculectomy (mm Hg) n=26 | Tentened-trabeculectomy (mm Hg) n=27 | p-value (unpaired t-test) |
| Pre-operative | | |
| 2nd day | | |
| Post-operative | | |
| 1 month | | |
| 6th to 24 months | | |
| Pre-operative | 29.34 ± 10.90 | 25.62 ± 7.88 | 0.08 |
| 2nd day | 10.50 ± 3.91 * * * | 5.96 ± 2.88 * * * | 0.0001 |
| 1 month | 14.3 ± 3.06 * * * | 11.3 ± 2.9 * * * | 0.0004 |
| 6th to 24 months | 15.50 ±3.61 * * * | 11.96 ± 2.87 * * * | 0.0001 |

Values are mean ± S.D.

* * * p < 0.001 (Turky-Kramer test) significantly differ from pre-operative IOP

| Table 2. Post-operative complications in patients treated by conventional and tented trabeculectomy. |
|---------------------------------------------------|---------------------------------------------------|---------------------------------------------------|
| Complications | Conventional trabeculectomy (n=26) | Tented trabeculectomy (n=27) | p-value (Chi-Square test) |
| Shallow A/C | 1 | 2 | 0.574 |
| Iridocorneal touch | 0 | 0 | — |
| Choroidal detachment | 1 | 3 | 0.317 |
| Hypotony | 1 | 2 | 0.575 |
| IOP >18 mm Hg | | | |
| after 6/24 months | 10 | 1 | 0.002 |
1st postoperative day and was treated and resolved with conservative treatment. Similarly, choroidal detachment also resolved with conservative management. Hypotony was found in 1 patient in the conventional and 2 patients in the tented group and resolved conservatively. IOP > 18 mm after 6-24 months was found in 10 patients in the conventional group and only in 1 patient in the tented group (p=0.002). All these 11 patients needed additional medical therapy to bring down IOP, except one from the conventional group who underwent repeat tented trabeculectomy.

Discussion

The field of glaucoma surgery is undergoing a period of revolution with many new approaches to the traditional methods of surgery. However indisputable concepts for effective treatment are still rare. In advanced optic disc cupping patients may require strict IOP control below an upper limit in order to preserve their remaining visual fields. One approach to setting a target IOP range is outlined in the Canadian Consensus guidelines that have been previously published. These challenges motivated this study to seek for a more efficient and safer procedure to control IOP. Various modifications for trabeculectomy have been cited over the years. Kolker et al (1993) used releasable scleral flap sutures with initial tight closure of scleral flap with the option to increase aqueous humor outflow in the early post operative period. Vuori and Viitanen (2001) demonstrated a sleral tunnel incision trabeculectomy to avoid postoperative hypotony. They dissected the scleral flap with a crescent shaped microblade. The sides were opened only halfway to the limbus and scleral flap was sutured with one 10/0 nylon releasable suture. However releasable sutures inside the corneal tissue may lead to infection and in this new technique of tented trabeculectomy and also in the conventional trabeculectomy performed in this study, releasable sutures were not used to avoid this problem.

Another method for a low level of IOP control employed the technique of tight scleral flap with subsequent postoperative suture lysis. This technique was not used in the present study as we get a titrated flow of aqueous with this technique rather than a sudden gush with suture lysis. Tight suturing of the scleral flap needs to be combined with postoperative laser suture lysis. Suture lysis may, however, lead to conjunctival hole with aqueous leak or be unsuccessful due to a thick conjunctiva or blood over the sutures. Tight suturing of the trabeculectomy flap can lead to higher pressure and poor drainage of aqueous. In another study by Fontana et al postoperative suture lysis was performed in 30 eyes but 11% of them needed a second glaucoma procedure. The filtering procedures tend to fail over time because of fibroblastic proliferation and sub-conjunctival fibrosis, which occurs during the normal process of wound healing. It is only recently that we have been able to modify this healing response with the use of antimetabolites like 5-fluorouracil and mitomycin C. 5-FU is known to cause complications like corneal toxicity, bleb leaks and postoperative hypotony. An increased incidence of postoperative endophthalmitis may also occur. A severe dose dependent complication of long term postoperative hypotony with associated maculopathy has been observed from mitomycin C. In this series of cases antimetabolites were not used at all.

In the present study conventional trabeculectomy results were compared with that of a modified tented trabeculectomy results for a period of up to 24 months. This tented trabeculectomy was found useful in both primary open angle glaucoma and angle closure glaucoma. Releasable sutures were not used in either technique to avoid the aforementioned problems. The anterior edges of the radial scleral incision are considered to account for postoperative aqueous outflow in the conventional method, whereas in the modified technique, aqueous passes through the tent that has been purposefully made rather than through the sides.

In a conventional trabeculectomy, over time there is fibrosis of sub-conjunctival tissue and failure of filtration whereas in the modified trabeculectomy there is copious flow of aqueous in one direction through the tent that has been purposely fashioned with the partial thickness scleral flap and the sides are more or less water tight because the side flaps are not opened all the way to the limbus. Fibrosis is prevented because of the continuous and large flow of aqueous in one direction. A larger area of filtration is obtained because of the reverse frown incision. Success was defined as an IOP less than...
18 mm Hg with no treatment. Partial failure was defined as an IOP controlled at less than 18 mm Hg with topical medication. Complete failure was defined as an IOP greater than 18 mm Hg.

The mean value of IOP on the 2nd post-operative day in the tented group was significantly lower than that of conventional technique which indicates a more abundant drainage. At the final follow up IOP was better controlled with tented trabeculectomy than conventional trabeculectomy. In the conventional group IOP > 18 mm Hg was found in 10 out of the 26 cases at the final follow up and only 1 in the tented group had IOP more than 18 mm Hg. This higher incidence of IOP in the conventional trabeculectomy group may be for want of antimetabolites usage or due to absence of releasable suture and laser suture lysis. Because of the above factors complications associated with hypotony was less in the immediate post-operative period in the conventional group. Hence severe complications associated with hypotony like flat A/C and iridocorneal touch were absent in both groups.

**Conclusion**
The greatest bugbear in glaucoma filtration surgery is the occurrence of fibrosis of the sub conjunctival tissue over time and consequent failure of the drainage. Making a tent purposely for aqueous drainage ensures that there is continuous flow of aqueous in one direction and consequently fibrosis is prevented due to the antifibrotic action of aqueous itself.

In the tented trabeculectomy technique, the sides of the flap are opened only half-way to the limbus to reduce the outflow of aqueous through the sides and redirect it in a single direction through the tent. Consequently, placing sutures on the sides near the limbus becomes unnecessary and the flap is tighter than in the conventional trabeculectomy. Fibrosis will be prevented because of the continuous and copious flow of aqueous in one direction. A larger area of drainage is obtained because of the reverse frown incision. Antimetabolites are not required because the large quality of aqueous itself prevents fibrosis. All these factors favor the tented trabeculectomy to be superior to the conventional method in achieving a lower IOP without the use of antimetabolites or releasable sutures.

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Bevacizumab in Inflammatory and Vascular Diseases of the Eye

Dr. Anju S. Raju MBBS DOMS, Dr. Biju Raju MS FNB, Dr. NSD Raju MS DOMS, Dr. PR Santha MBBS DOMS

Aim: To assess the safety and efficacy of Bevacizumab in various inflammatory and vascular diseases of the eye. Study design Prospective, non-randomized interventional trial

Methods: Intravitreal bevacizumab (0.05ml) was used in 51 patients with diabetic maculopathy, vitreous hemorrhage, AMD, intraocular inflammation, cystoid macular edema, central retinal vein occlusion and in Kimmelstein Wilson syndrome. Bevacizumab was used in cases of corneal neovascularization in limbal cell deficiency and also in graft rejection.

Results: Bevacizumab reduced the macular edema and neovascularization in all the patients without any complications. The functional improvement in cases of macular ischemia was minimal.

Conclusion: Bevacizumab is a safe and effective Vascular Endothelial Growth Factor (VEGF) inhibitor for various ocular conditions

Keywords: Bevacizumab, Avastin, Age-related Macular degeneration, Macugen, PDT, diabetic retinopathy

Introduction

Vascular endothelial growth factor (VEGF) has, on a molar basis, 50,000 times more potency at making vessels permeable when compared to histamine. This factor was cloned and expressed in 1989 and was found to be a potent stimulator of endothelial cell growth in vitro and neovascularization in vivo. The term VEGF refers to a collection of related protein isoforms derived from the same gene, the most well studied being VEGF121, VEGF165 and VEGF189. The VEGF family has grown in the past decade to encompass VEGF-A, VEGF-B, VEGF-D, VEGF-E and placental growth factor. VEGF, as it is classically known, refers to VEGF-A [1]

Of all the factors made by hypoxic retinal cells, VEGF appears to be the sole endothelial cell mitogen. VEGF receptors are present and active on all inflammatory cell types. Ischemia-induced neovascularization was previously thought of as being non-inflammatory in nature. Recent data shows that this process of neovascularization also involves inflammatory and pro-inflammatory cells. [1]

In ocular diseases like proliferative diabetic retinopathy, diabetic macular edema, age-related macular degeneration, retinopathy of prematurity and corneal vascularization, VEGF has been implicated in the process of new vessel formation and increased vascular permeability. VEGF also plays a role in inflammatory diseases of the eye. Blockade of VEGF receptor is a novel way to suppress the untoward effects of VEGF in the eye. Of the VEGF receptor blockers in clinical trials and practice, Bevacizumab is now gaining in popularity. Originally used for metastatic colorectal carcinoma, this
PAN-VEGF blocking monoclonal antibody was found to have beneficial effect in wet AMD. [2] Few studies have documented its safety profile when given systemically as well intravitreally. [3] However, its role in other diseases of the eye has not been well documented. Our aim was to assess the role of bevacizumab in various neovascular and inflammatory conditions of the eye.

Materials and Methods

Informed consent was obtained from all the patients after detailed discussion regarding the nature of the study medications used. Patients with wet AMD, CME due to diabetic maculopathy and vein occlusions, neovascular glaucoma, postoperative uveitis, and corneal vascularization were enrolled in the study. Patients with uncontrolled hypertension, recent history of stroke, ischemic heart disease were excluded. All patients recruited underwent a complete and detailed preoperative evaluation, which included best corrected visual acuity, applanation tonometry, slit lamp evaluation, dilated indirect ophthalmoscopy and stereoscopic biomicroscopy, fluorescein angiography and optical coherence tomography. Patients with known hypersensitivity to fluorescein sodium, patients with neovascular glaucoma and patients with vitreous hemorrhage were not subjected to fluorescein angiography.

Intravitreal bevacizumab injection (IVA) was administered in the operating room under all sterile precautions. Under deep topical anesthesia using a pledget soaked in proparacaine hydrochloride (Sunways, Mumbai), after through cleaning and prep with betadine 5% solution, the intravitreal injection of 0.05 ml of Bevacizumab (Avastin, Roche) was given 3mm (pseudophakic eyes) or 3.5 mm (phakic eyes) posterior to the limbus using a 30 G needle. The tip of the needle was visualized in the mid vitreous and the drug exits the needle tip in the form of a gel into the vitreous cavity on injection. The needle is withdrawn and the site is compressed with a sterile tip of a cotton bud for 4 to 5 seconds. The patient is started on moxifloxacin eye drops and antiglaucoma medications which are continued for a week. Patients receiving deep posterior subtenons injection of triamcinolone (PSTA) received the injections after subconjunctival infiltration of xylocaine at the planned injection site. The injection was delivered in to the deep subtenons space using a Venflon IV cannula, which is introduced temporally after the cannula is removed. The details of this technique have been described elsewhere and its safety has been established. [4] Along with bevacizumab 0.05 ml of commercially available Hyaluronidase was used in cases of vitreous hemorrhage [5] and in few cases a couple of weeks prior to surgery with an intention to induce PVD as well as pharmacological vitreolysis and regression of active membranes.

Two patients of neovascular glaucoma underwent cryotherapy and at the end of the procedure received intravitreal or intracameral injection of bevacizumab.

All patients were followed up 1\textsuperscript{st}, 2\textsuperscript{nd} week and 1 month. At the follow up visits, a complete ophthalmic evaluation including applanation tension was recorded. The longest follow up in this series was 6 months. Patients with recurrence as defined by drop in visual acuity and worsening of clinical signs were subjected to repeat intravitreal injection of bevacizumab and intravitreal triamcinolone. Patients with recurrence of wet AMD underwent repeat fluorescein angiography and OCT prior to the repeat injection.

Results

There were 52 eyes in this pilot study. The age of the patients ranged from 29 to 80 years with a mean of 65. There were 19 females and 32 males. Five patients received bilateral injections. Three patients received repeat injections. Diabetic eye disease was the major group in this study. There were 11 eyes of cystoid macular edema of varied etiology and 9 eyes of CNVM due to wet AMD. [Table 1]

<table>
<thead>
<tr>
<th>Table 1. Various neovascular and inflammatory conditions included in the study</th>
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<tbody>
<tr>
<td>CME (BRVO-2, IOL-1, PRP-1, DME-1, CRVO-6)</td>
</tr>
<tr>
<td>CNVM (WET ARMD)</td>
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<tr>
<td>Diabetic Maculopathy</td>
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<tr>
<td>PDR</td>
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<tr>
<td>Other indications (Endophthalmitis-1, Corneal neovascularization -1, IPCV-2, Postop uveitis-1, Eales-1)</td>
</tr>
</tbody>
</table>
Choroidal Neovascularization in wet AMD
There were 9 eyes with CNVM secondary to wet AMD. All eyes had improvement in vision following the injection. Initial 2 cases had received only avastin injection. The rest received a combination of IVA and PSTA. One eye with a massive submacular hemorrhage and exudation had a remarkable improvement in vision from CFCF to CF3m following IVA and had complete regression and scarring of the CNVM. [Figure 1A and B]

Proliferative Diabetic Retinopathy
In this study there were 14 eyes with proliferative diabetic retinopathy (PDR). Of these, 3 eyes had clinically significant macular edema and therefore, IVA with PSTA was given to these eyes to prevent exacerbation of CSME after PRP. There was a reduction in the macular edema within 24 hours along with perceptible reduction in the neovascularization, especially on the disc after the injection. In eyes with vitreous hemorrhage, IVA with Hylase cleared the hemorrhage in 6 out of 13 eyes. [Figure 2] In 2 eyes the clearance was partial and therefore underwent vitrectomy. Of the 7 eyes in which hemorrhage had not cleared 5 had traction retinal detachment and 3 eyes underwent vitreoretinal surgery. In the eyes which underwent vitrectomy, induction of PVD as well as membrane dissection was easy.

Diabetic Maculopathy
Of the 12 eyes with diabetic maculopathy, 2 eyes had CSME, and 2 eyes had CSME with macular ischemia. Among the eyes with diffuse macular edema 4, 1 eye underwent cataract surgery along with avastin injection, one eye had macular ischemia and 2 eyes had DME with no macular ischemia. One patient had renal failure and extensive lipid deposits. In this patient with Kimmelstein Wilson Syndrome (KWS), Avastin improved the macular status and visual acuity improved from 6/36 to 6/12.

Cystoid Macular Edema
There were 11 eyes with cystoid macular edema which were secondary to CRVO (6) (of which one eye was clinically ischemic CRVO) [Figure 1 C and DJ], BRVO (2), and one eye each of pseudophakic CME, CME following PRP for PDR, diffuse macular edema with no macular ischemia.
Other indications

We used intravitreal avastin intraoperatively in cases of peripheral variant of Idiopathic polypoidal. Choroidal Vascularisation (IPCV) with massive suprachoroidal hemorrhage and breakthrough vitreous hemorrhage. Both the cases had a fair outcome, considering the very poor prognosis in these cases. Both cases had marginal improvement of visual acuity from HM to CF2 m with an attached retina and no recurrence of hemorrhage.

Avastin was used intraoperatively in a case of fungal endophthalmitis. The patient had recurrence after vitrectomy and amphotericin B injection. During the repeat vitrectomy, amphotericin B was injected followed by 0.05ml of Avastin. The patient had an uneventful postoperative period with minimal inflammation. The patient had a visual acuity gain from CF1m to 6/9 and N6 and has had no recurrence of inflammation. In one case of postoperative uveitis secondary to dropped lens matter, intravitreal avastin was used during vitrectomy and the patient had good control of inflammation and had a good postoperative outcome.

Intracorneal avastin was used in a case of chemical injury and corneal vascularization. There was a regression in the corneal vascularization after the injection. In another patient with corneal vascularization after penetrating keratoplasty, topical avastin was used. This patient also showed regression of the corneal vascularization. There were 2 case of neovascular glaucoma in this series. One of these patients had a persistent corneal epithelial defect which was not responding to conventional therapy. This eye had a remarkable improvement after intracameral avastin was given. The cornea cleared, the iris neovascularization regressed and the persistent epithelial defect healed within a week postoperatively. [Figure 3 C and D] The other eye with NVG also responded well following intravitreal avastin and trans scleral cyclotherapy.

One eye having Eales disease with vitreous hemorrhage, active neovascularization and vitreomacular traction received intravitreal avastin along with hylase. This patient had undergone panretinal photocoagulation (BE) few months prior to this episode of defective vision. Following the injection, there was a spontaneous posterior vitreous detachment with relief of vitreomacular traction and resolution of vitreous hemorrhage. Within 3 weeks of the injection, the visual acuity in this eye improved from 6/36 to 6/6. [Figure 3 A and B]

Overall results [Table 2]

| 1. AMD       | IVA+PSTA | Fair to Good |
| 2. CME       | IVA+PSTA | Good         |
| 3. VH        | IVA+hylase | Fair    |
| 4. NVG       | IVA+PSTA+Cryo | Good |
| 5. Inflammatory diseases | IVA+PSTA | Good         |
| 6. Adjunctive in Membrane Surgery peeling and PVD induction | IVA | Good |
| 7. Anterior segment neovascularization | IVA | Modest results |
| 8. DME without Macular ischemia | IVA | Fair |
| 9. DME with Macular ischemia | Poor | to used with caution |

Table 2. Bevacizumab – Results of the pilot study with recommendations and response
All patients had stable visual acuity and majority of them had an improvement in visual acuity. [Figure 4] One eye lost 2 lines of vision and this eye had a preexisting ischemic status of the macula. No eye had any intraoperative or postoperative complications. No eye had uveitis, or progression of cataract. Two eyes in this series had ocular hypertension which was controlled well with topical antiglaucoma therapy (Timolol maleate and dorzolamide combination therapy, Misopt, Microvision, India). These 2 eyes had received deep posterior subtenons triamcinolone acetonide injection also. All patients had completed at least 8 weeks after the procedure (8 weeks to 20 weeks).

**Discussion**

This pilot study on the efficacy of Bevacizumab in ocular diseases has its limitations. Though it is a prospective study, there are no controls and the follow up period is short. The beneficial role of bevacizumab in diseases like Eales disease and anterior segment neovascularization has been reported for the first time in this study. However the number of patients in this group of diseases is small. We have also explored the use of intravitreal avastin in combination with hyaluronidase in achieving pharmacological vitreolysis. [5] This also is the first study to document the sustained effect of antiangiogenesis, when intravitreal bevacizumab is combined with deep posterior subtenons triamcinolone acetonide.

The best results with the combination therapy of Intravitreal Avastin (IVA) and deep posterior subtenons triamcinolone acetonide (PSTA) were seen in cases having cystoid macular edema secondary to diabetic nonischemic maculopathy and in vein occlusions. The effect was transient in cases with macular ischemia and therefore should be used with caution if macular ischemia is suspected.

In AMD, we found a sustained effect lasting for more than 6 weeks in most of the cases. In one case of a juxtapapillary CNVM, where the visual acuity improved from counting fingers \( \frac{1}{2} \) m to 6/18 the effect of the single injection was sustained for 5 months. All cases of CNVM showed stabilization. Visual acuity improved in 4 eyes. The mean number of injections in an eye was 2.

Cases of vitreous hemorrhage secondary to PDR, vascular occlusions and Eales disease received Intravitreal bevacizumab and commercially available hyaluronidase injection, both given as 0.05 ml injection at the same time. We noticed the occurrence of PVD in all cases. In one case there was complete resolution of the vitreous hemorrhage and regression of NVD secondary to ischemic CRVO. One eye with vitreous hemorrhage and vitreomacular traction and an active neovascular frond secondary to Eales disease had spontaneous relief of VMT and regression of the NVE following intravitreal injection of avastin and hylase. In 3 cases, though the visual acuity improved and the hemorrhage cleared to the extent that disc and vessels were visible, due to the professional demands of the patients, had to be undertaken for vitreous surgery. During surgery PVD was noted and regression of the new vessels was seen. Previous studies have documented good results with highly purified hyaluronidase intravitreal injection (Vitrase). [5] Though iritis was a common complication noted in that study none of our patients had any signs of intraocular inflammation. These results prove that intravitreal injection of commercially available hyaluronidase is well tolerated. The other reason for the resolution of the vitreous hemorrhage is probably due to regression of the neovascular focus by avastin which helps in preventing fresh vitreous hemorrhage.

Intravitreal avastin and hyaluronidase was used as an adjunct therapy in cases of PDR with active fibrovascular membranes and traction retinal detachments. In these cases, the vessels had almost completely regressed and induction of PVD as well as membrane peeling during vitrectomy was easy.
Neovascular glaucoma, a disease where cyclodestructive procedures were the only therapeutic options available, showed remarkable response to intravitreal avastin. We combined partial cyclocyrotherapy (180 degrees only) with intravitreal avastin in cases of NVG. The regression of new vessels on the iris as well as symptomatic improvement was seen as early as the 1st week postoperatively. None of the patients with NVG required a second injection.

Modest results were noted in anterior segment neovascularization. Intracorneal avastin was given in an eye which had sustained chemical injury and had extensive deep corneal vascularisation. Avastin along with living related limbal stem graft was done and there was a modest regression in the neovascularization. Topical bevacizumab was prepared by adding 0.1 ml of avastin into 5 ml of lubricant drops (Refresh Liquigel, Allergan). This was used QID in patients with corneal neovascularization after PK.

Cystoid macular edema secondary to intraocular inflammation also responded well to combination therapy of IVA + PSTA. None of the patients in this category required a second injection. One patient with pseudophakic CME and after cataract had good visual outcome after YAG capsulotomy was done once the CME was controlled with IVA+PSTA.

Our experience with bevacizumab has been encouraging. Although this drug was not intended for intraocular use, it was well tolerated in our series of patients. There were no cases of uveitis, endophthalmitis or thromboembolic episodes after the injection. Except one patient, none in the series had ocular hypertension.

One of the most debated topics in medical retina is the best treatment options from AMD and Diabetic maculopathy. A therapeutic approach should be cost-effective and affordable to all, especially in a developing country like India, with its large population of senior citizens and diabetics. Current options for treatment for AMD are Photodynamic therapy (PDT) and intravitreal Pegaptanib Sodium (Macugen). Macugen has been found to be effective in short term control of diabetic maculopathy also. The drug needs to be injected into the vitreous every 6 weeks and most patients require 4 to 5 injections per year [6]. PDT when combined with IVTA [7] has a re-treatment rate of 1.24 sessions to stabilize or regress the CNVM. Ranibizumab is another anti-VEGF agent introduced recently into clinical practice and the initial reports are encouraging.

Of all the currently available therapeutic options for wet AMD, Bevacizumab seems to be the most cost-effective one.

**Conclusion**

Bevacizumab is a safe and effective VEGF inhibitor. When used alone or in combination with posterior subtenons injection of triamcinolone acetonide, it is effective in treating AMD, CME secondary to DME, inflammatory diseases. In eyes with extensive neovascularization and fibrovascular proliferation, intravitreal bevacizumab with hyaluronidase prior to surgery, seems useful in PVD induction and removal of the membranes. Combining with hyaluronidase intravitreal injection, pharmacological vitreolysis can be achieved in some cases. Patients with neovascular glaucoma, after intravitreal injection of bevacizumab and partial cryotherapy, responded well with regression of neovascularization and symptomatic improvement. Thus, Bevacizumab is a useful VEGF inhibitor in several neovascular and inflammatory diseases of the eye.

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Cyclosporine
Dr. Rajiv Sukumaran DO MS FRCS, Dr. Jayasree Rajiv DO

Cyclosporin, cyclosporin or cyclosporine, is an immunosuppressant drug widely used in post-allogeneic organ transplant, to reduce the activity of the patient's immune system and hence the risk of organ rejection. It has been studied in transplants of skin, heart, kidney, lung, pancreas, bone marrow and small intestine. Cyclosporine is a cyclic nonribosomal peptide of 11 amino acids (an undecapeptide) produced by the fungus Tolypocladium Inflatum Gams, initially isolated from a Norwegian soil sample.

**Indications**
The immuno-suppressive effect of Cyclosporin was discovered on January 31, 1972, by employees of Sandoz (now Novartis) in Basel, Switzerland, in a screening test on immune-suppression designed and implemented by Hartmann F. Stähelin. Cyclosporin was subsequently approved for use in 1983.

Apart from the use as a transplant medicine, Cyclosporin is also used in psoriasis and infrequently in rheumatoid arthritis and related diseases, although it is only used in severe cases. It has been investigated for use in many other autoimmune disorders. Cyclosporin has also been used to help treat patients with ulcerative colitis who do not respond to treatment with steroids. [2] This drug is also used as a treatment of posterior or intermediate uveitis with non-infective etiology.

Cyclosporine A has been investigated as a possible neuroprotective agent in conditions such as traumatic brain injury, and has been shown in animal experiments to reduce brain damage associated with injury. Cyclosporine A blocks the formation of the mitochondrial permeability transition pore, which has been found to cause much of the damage associated with head injury and neurodegenerative diseases.

**Mode of action**
Cyclosporine (cyclosporin A, CsA) has potent immunosuppressive properties, reflecting its ability to block the transcription of cytokine genes in activated T cells. It is well established that CsA through formation of a complex with cyclophilin inhibits the phosphatase activity of calcineurin, which regulates nuclear translocation and subsequent activation of NFAT transcription factors. In addition to the calcineurin/NFAT pathway, recent studies indicate that CsA also blocks the activation of JNK and p38 signaling pathways triggered by antigen recognition, making CsA a highly specific inhibitor of T cell activation. Here we discuss the action of CsA on JNK and p38 activation pathways. We also argue the potential of CsA and its natural counterparts as pharmacological probes. It has also an effect on mitochondria. Cyclosporine A prevents the mitochondrial PT pore from opening, thus inhibiting cytochrome c release, a potent apoptotic stimulation factor. However, this is not the primary mode of action for clinical use but rather an important effect for research on apoptosis.

**Adverse effects and interactions**
Treatment may be associated with a number of potentially serious adverse drug reactions (ADRs) and adverse drug interactions. Cyclosporine interacts with a wide variety of other drugs and other substances including grapefruit juice, although there have been
studies into the use of grapefruit juice to increase the blood level of cyclosporine.

Adverse drug reactions can include gum hyperplasia, convulsions, peptic ulcers, pancreatitis, fever, vomiting, diarrhea, confusion, breathing difficulties, numbness and tingling, pruritus, high blood pressure, potassium retention and possibly hyperkalemia, kidney and liver dysfunction (nephrotoxicity and hepatotoxicity), and obviously an increased vulnerability to opportunistic fungal and viral infections.

Formulations

Cyclosporine is available in 25 mg and 100 mg tablets. Atopica® formulation is available in 10, 25, 50 and 100 mg sizes. It is also available in an injectable form. There is an ophthalmic preparation that is available for specific treatment of the eye. The drug is marketed by Novartis under the brand names Sandimmune, the original formulation, and Neoral for the newer microemulsion formulation. Generic Cyclosporin preparations have been marketed under various trade names including Cicloral (Sandoz/Hexal) and Gengraf (Abbott). Since 2002 a topical emulsion of Cyclosporin for treating keratoconjunctivitis sicca has been marketed under the trade name Restasis. Annual sales of Cyclosporin are around $1 billion.

Cyclosporin and Eye

Dry-eye syndrome (keratoconjunctivitis sicca) is an extremely common and painful condition, affecting more than 1 million people in the U.S., especially older adults, post-menopausal women, and those with Sjogren's Syndrome, rheumatoid arthritis, lupus, Parkinson's and other chronic diseases. Characterized by insufficient tear production, the syndrome eventually may lead to vision loss.

Cyclosporin applied topically as 0.05% drops showed increased tear production and less need for artificial tears. Unlike artificial tears, which temporarily replenish eye moisture, it helps treat the cause of dry-eye syndrome: reduced tear production due to inflammation. Treatment of dry eye syndrome with topical cyclosporine significantly reduced the numbers of activated lymphocytes within the conjunctiva.

Significant reductions were observed with respect to the percentages of CD4+ and CD23+ cells in the conjunctival impression cytology specimens and clinical and symptom scores following treatment with topical Cyclosporine A, while no change occurred in the
percentages of CD8+ and CD45RA+ cells. Topical cyclosporine A treatment is a very effective alternative in severe VKC cases. Clinical efficacy of topical cyclosporine A treatment in severe, resistant VKC cases can be (at least partly) related to reduction of the CD23+ and CD4+ cell populations on the conjunctival surface.

Exposure to cyclosporine A directly modified fibroblast behavior. Cyclosporin A reduced PIP and interleukin 1 (IL-1) production in a dose-dependent manner. Interleukin 6 and IL-8 were increased by 10 µg/mL of CsA, whereas transforming growth factor, PIIIP, and total protein were unaffected. Cyclosporin A exposure induced apoptosis is time and dose-dependent.

**Other uses in the eye**

Intermediate and posterior noninfectious uveitis, stromal keratitis induced by herpes simplex virus (HSV) are treated with cyclosporine A eyedrops and acyclovir ointment.

**Serpiginous choroiditis**

Bilateral inflammatory serpiginous choroiditis have been treated with Cyclosporine-A. Its usefulness seems to be greater when the serpiginous choroiditis is in its acute stage; chronic stages, however, also seem to improve with treatment to a lesser degree. Its main indication is when there is involvement of the macular region of the second eye, when the first eye is already damaged. We consider Cyclosporine-A, in these situations, to be a first choice treatment.

Cyclosporin Implant to treat uveitis is under trial. Other uses are in Corneal grafts, Mooren's Ulcer, non-healing ulcers etc.

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OCT in Glaucoma

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Glaucoma is the clinical manifestation of irreversible damage to the retinal ganglion cells resulting in retinal nerve fibre layer loss. This becomes functionally evident as visual field loss which is detected on perimetry. It is now known that structural damage detected by RNFL defects precedes functional loss. 40 % axonal loss has to occur before any detectable change occurs in visual function. 1 Another study 2 showed that RNFL loss was detected nearly six years before visual field defects occurred in ocular hypertensives. The recognition of RNFL loss in patients with normal visual fields has led to the concept of “Pre-perimetric” glaucoma, signifying early glaucomatous damage not evident on standard automated perimetry. RNFL loss also precedes optic disc changes which occur before visual field changes. It has also been shown that early treatment is important in lowering the patient’s risk of glaucoma progression. 3 However, clinical evaluation and photography of the RNFL is a difficult technique, is subjective, qualitative and variably reproducible. With the advent of Optical Coherence Tomography (OCT) or scanning laser polarimetry (GDx), highly reproducible RNFL assessment and quantification is possible, thus enabling improved management of glaucoma.

Principle of OCT:

OCT is a non-invasive imaging technology that uses light to create high resolution (<10 µ), cross-sectional images in an acquisition time of around 1.2 seconds.

Validity of OCT for glaucoma:

1. OCT measurement were found to be highly reproducible 4 especially in a circle diameter of 3.4 mm and with internal fixation.
2. Ability to distinguish normal from glaucomatous eyes is greater as established by several studies 5,6,7.
3. Effect of age : OCT normative values have been developed to adjust for the RNFL thinning with age.

OCT SCANS FOR DETECTING GLAUCOMA

The OCT enables scanning three regions for glaucoma detection-
1. The Peripapillary retinal nerve fiber layer (RNFL)
2. The macular region.
3. The optic nerve head.

1. RNFL Measurement

RNFL measurements with OCT have sensitivity and specificity in differentiating glaucomatous from normal subjects. 8,9 Of these, RNFL thickness in the inferior region and mean RNFL thickness are the best to detect early to moderate glaucoma.

2. Macular Thickness

The mean macular thickness of glaucomatous eyes has been shown to be significantly lower than that of normal control eyes. 10 A significant correlation between OCT macular thickness and visual field mean defect in glaucomatous eyes has been demonstrated. 11 However this is inferior to RNFL measurements.
3. Optic Nerve Head (ONH) Analysis

The utility of this needs to be further evaluated as variation may occur, for example disc margin evaluation may be influenced by changes in the RPE- choriocapillaris layer.

However, a study has shown the rim area and horizontal integrated rim width as the best ONH parameters, while another has shown the cup disc area ratio as the best ONH discriminator.

IMAGE ACQUISITION

There are 2 basic OCT scan patterns- lines and circles.

1. Scan placement for RNFL protocols: These are circle scans centred in the middle of the optic disc.

2. Optic Nerve head Protocols: These are line scans arranged like spokes of a wheel the center of which should be in the middle of the optic disc.

GLAUCOMA SCAN PROTOCOLS

The OCT scan protocols (Fig. 1) described for glaucoma detection and management include the following:

I. RNFL Protocols

a. RNFL thickness (3.4): Consists of 3 circle scans of 3.4 mm diameter around the optic disc which are averaged.

b. RNFL thickness (2.27 x disc): a single circular scan around the optic disc that is 2.27 times the radius of the aiming circle.

c. Fast RNFL thickness (3.4): acquires three 3.4 mm diameter scans in 1.92 seconds and compresses them into one scan.

d. RNFL Map: consists of a set of six concentric circle scans at increasing distances from the disc margin.

II. ONH Protocols

(a) Optic Disc (b) Fast optic disc: Compresses six optic disc scans into one scan in 1.92 seconds.

III. Macular Thickness Protocols

a. Macular Thickness Map

b. Fast Macular Thickness Map: Compresses six macular thickness scans into one scan in 1.92 seconds.
ANALYSIS PROTOCOLS FOR GLAUCOMA
(Fig. 2 & 3)

1. RNFL Thickness (Single Eye)

The normal RNFL graphs appear as a “double hump”: e.g. due to increased RNFL thickness at the superior and inferior poles of the disc. The output chart includes circle characteristics like quadrant and clock hour RNFL thickness averages. The RNFL is depicted in hot colours i.e. a red band superficial to the green retina (Fig. 4).

Fig. 4. RNFL thickness report

2. RNFL Thickness Average (OU)

Two maps, one showing RNFL thickness using a colour code (A) and the other showing average RNFL thickness in microns (B) are created (Fig 5).

The black graph represents the RNFL thickness of the eye being tested in the nasal, superior, temporal and inferior quadrants. The OCT has incorporated normative age-matched RNFL thickness data. The software displays graphs which are colour coded according to the probability of the RNFL thickness measured in the particular patient being normal when compared to age-matched controls.

Fig. 5. RNFL thickness average showing normal thickness of RNFL in both eyes

Of the normal population (Fig 6)

: 5 % fall within the white band
: 5 to 95% fall within the green band
: 1 to 5 % fall within the yellow band.
: 1 % fall within the red band (Outside normal limits)

Fig. 6. Graph showing probability of distribution of RNFL thickness in the normal population

The output chart also provides RNFL thickness values in clock hours and quadrants using the same colour code. (B). Summary parameters which include average thickness in each quadrant, maximum RNFL thickness in superior and inferior quadrants and ratios of RNFL thickness in various quadrants (I max/S max, S max/I max; S max/T avg; I max/T avg) are also provided (C). Usually the
inferior RNFL is the thickest and the I max/ S max ratio is greater than 1.0. In glaucoma, the ratio may be less than 1.0 due to inferior RNFL loss.

3. RNFL Thickness Serial Analysis (OU)

This allows comparison of RNFL thickness over time for up to 4 visits which are superimposed on the same chart (A) and each visit is colour coded (B) (Fig. 7). RNFL thickness change analysis shows the difference in RNFL thickness in two visits (Fig. 8).

4. Optic Nerve Head (Single Eye)

The algorithm detects and measures all features of the disc anatomy based on the anatomical markers (disc reference points) on each side of the disc where the RPE ends. It locates and measures the disc diameter by tracing a straight line between the two disc reference points (Blue line A) and measures cup diameter on a line parallel to the disc line and offset anteriorly by 150 µ (Red line, B). The output chart measures the optic disc, optic cup, neuroretinal rim and cup/disc ratio using these measurements. (Fig. 9).

5. RNFL Thickness Map (OU)

Two maps, one representing RNFL thickness using a colour code and the other the average RNFL thickness in the inner and outer areas of eight map sectors are generated.

6. Macular Thickness Map

Two maps, the upper one showing retinal thickness using a colour code and the lower one showing average retinal thickness in microns in each area are seen.
Normal OCT

The normal optic disc is seen on OCT as a cross section through the optic nerve head. The cup is shallow with sloping edges. The neuroretinal rim is seen as a wide red zone straddling the disc margin (Fig. 10).

![Fig. 10. Normal Optic nerve head](image1)

The RNFL thickness analysis shows the characteristic double hump pattern signifying increased thickness at the inferior and superior pole of the optic nerve head. The graph falls well within the green colour region and all quadrants are flagged in green indicating normal RNFL thickness (Fig. 11).

OCT in Glaucoma

In the section of the optic disc, the cup walls appear vertical and the depth of the cup is increased. The neuroretinal rim is very thin (Fig 12).

![Fig. 12. Optic nerve head analysis showing glaucomatous cupping](image2)

RNFL thickness will be reduced; the superior and inferior hump may be flattened and the graph is seen to be encroaching in the red and yellow areas. The average RNFL thickness in the quadrants in which it is reduced will be flagged in red or yellow (Fig. 13).

OCT in the Clinical Management of Glaucoma

1. To Confirm the Integrity of RNFL in Disc Suspects: (Fig. 14)

Disc suspects are defined as those eyes with a cup-disc (C/D) ratio > 0.7 or a neuroretinal rim width of less
than 0.1 of the CD ratio between 11 to 1 o’clock or 5 to 7 o’clock, but no definite field defects.

Disc suspects require followup since they could possibly be preperimetric glaucoma, not detected on conventional automated perimetry. OCT helps to quantify RNFL changes for followup and to determine any progressive loss which if present points to a diagnosis of early glaucoma.

2. Monitor Followup of Patients with Ocular Hypertension

OHT is a condition characterized by elevated IOP with out definite disc or field changes. The OHTS (Ocular Hypertension Treatment Study) investigated the predictive factors for development of glaucoma and found that increased IOP, increased CD ratio and thinner corneas were major risk factors. Serial RNFL analysis can help monitor RNFL loss in these group of patients. (Fig. 7)

3. Establish a Suspicion of Preperimetric Glaucoma: (Fig. 15)

The spectrum of glaucoma ranges from normal healthy eye through undetectable disease to detectable, asymptomatic disease to functional impairment and eventual blindness. During the asymptomatic stage, demonstration of RNFL loss in OCT analysis points to a diagnosis of pre perimetric glaucoma before field changes are demonstrable in automated perimetry. Early diagnosis is imperative for early treatment and prevention of visual loss across this continuum.

4. Monitor Asymmetric POAG

In patients with unilateral established glaucoma and normal fellow eye with increased IOP, OCT helps in followup. Such cases may need to be treated as there is 40% chance of developing visual field loss over a 5 year period. Hence early detection of RNFL defects is of importance.

5. Monitor Patients Unable to Perform Visual Field Tests Reliably

In such situations OCT may provide extra structural evidence to substantiate the clinical impression.
6. Help Monitor Patients when Visual Field Interpretation may be Fallacious due to Non Glaucomatous Causes.

7. Follow up of Established Glaucoma Patients to Detect Progressive RNFL Loss.

Conclusion

Thus OCT helps in the early diagnosis and monitoring of glaucoma even before functional visual loss in the form of visual field loss occurs. This is useful in preventing progression of glaucoma.

However, as with any other imaging modality, OCT should never be interpreted in isolation. Correlation with the clinical findings, field changes and IOP helps in the management of glaucoma in a more scientific manner.

References


Fig. 15 (A & B) showing normal C 30-2 (15 B) but mild thinning inferiorly of RNFL (15 A) on OCT in the left eye.
OPHTHALMIC HISTORY

Roger Bacon and the Spectacle Lens

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Spectacles have been around for a long time but it is uncertain when and where they were first used. They probably just evolved over a period of time.

Though the Roman Emperor Nero used an emerald while watching gladiatorial contests, it is not known whether this was as a symbol of power or to see better.

The first recorded reference to using a lens to improve vision was in 1262 by the Franciscan Friar, Roger Bacon. He may not have been the first person to observe the magnifying properties of sphere segments but he certainly recognized their use in old people and those with poor eyesight. He thus recommended the use of loupes and magnifying glasses which were the forerunners of today’s spectacles.

Bacon was a truly remarkable man who possessed one of the most commanding intellects of his own or perhaps of any age. In spite of the disadvantages and discouragements to which he was subjected, he made many discoveries and came near many more. He was an enthusiastic proponent and practitioner of the experimental method of acquiring knowledge.

The exact date and place of Bacon’s birth are unknown. It was probably in 1214 at Ilchester in Somerset, England. His father was a wealthy landowner and Bacon had the advantage of early training in Geometry, Arithmetic, Music, and Astronomy. He studied at Oxford and Paris and later taught at both places. He was an unconventional scholar being interested in Philosophy, Alchemy and Magic, even while championing the cause of Experimental Science.

Bacon’s wide range of interests included Optics and the refraction of light through lenses. He described telescopes and microscopes, flying machines, motorized ships and cars, pulleys and the making of gun powder. He also sought to reform the Julian calendar which was then in use, and was interested in Astronomy. He first recognized the visible spectrum in a glass of water, centuries before Sir Isaac Newton. He stressed the importance of Mathematics in the understanding of all Science and his greatest contribution was to insist that a study of the natural world by observation and exact measurement was the surest foundation for truth.

(Contd. on pg. 196)
Congenital Nasolacrimal Duct Obstruction (CNLDO)

Dr. Ramesh Murthy MD FRCS

Introduction

CNLDO is a common problem that ophthalmologists routinely face in their practice. This condition affects nearly 20% of all newborns. Most of the cases are self-resolving. It is important to understand the lacrimal drainage anatomy, embryological development and the natural course of this condition for appropriate diagnosis and management.

PATHOPHYSIOLOGY

Embryology

The development of the lacrimal drainage system begins at approximately 6 weeks of gestation. This develops along the line of the cleft between the maxillary and lateral nasal processes. Here an ectodermal fold extends into the underlying mesenchyme and forms a solid rod of cells between the medial canthus and the nasal cavity. Canalization of the ectodermal cord begins in the third month at the medial canthus. Canalization progresses towards the eyelid margin and towards the inferior meatus (Figure 1a and 1b).

Communication between the lacrimal drainage system and the nose occurs at the end of the sixth month. The lacrimal puncta opens into the lid margin during the seventh month, before the lids separate. The tear duct opening into the nose beneath the inferior turbinate does not become patent until birth or shortly after birth. Blockage of the nasolacrimal system typically occurs in

![Figure 1a and 1b. Development of the lacrimal drainage system: An ectodermal fold develops in the mesenchyme between the lateral nasal and maxillary processes. This undergoes canalization towards the eyelid margin and the nose finally forming the NLD.](a)
this distal portion, where the tear duct opens into the nose. Tears are normally produced a few weeks after birth; hence nasolacrimal duct (NLD) obstruction may not be recognised until several weeks after birth.

**Causes**

Most commonly, this is due to the presence of a membrane at the level of the valve of Hasner, which is present at the nasal opening of the nasolacrimal duct. Less frequent causes include congenital atresia of the NLD, congenital lacrimal sac mucocele, congenital absence of valves, absence or atresia of canaliculi and puncta, and facial cleft anomalies.

**FREQUENCY**

While up to 20% of newborns have CNLDO, only 1-6% of infants have symptomatic obstruction. The majority of cases (up to 96%), usually resolve by the age of 1 year.²

**APPROACH**

**HISTORY**

The parents will usually give a history of the child exhibiting unilateral or bilateral tearing. Other symptoms include crusting, mucoid or mucopurulent discharge and redness. The lashes may stick together in the morning or after the child takes a nap. Tearing may be aggravated by upper respiratory tract infections or with exposure to wind or cold.

Important questions include: What is the frequency of the symptoms? Are the symptoms constant or intermittent? Is the tearing only present when the child has upper respiratory tract infection? Or is it aggravated in the cold or wind? At what age did the symptoms actually appear? If the child developed symptoms later on in life, then the problem is unlikely to be a non patent valve of Hasner.

Was the child full term and if not, what is his age adjusted for gestational dates? Are there any associated congenital anomalies, especially craniofacial anomalies such as Goldenhar’s, Crouzon’s syndrome or conditions with a hypoplastic maxilla such as Treacher-Collins syndrome? Is there a history of nasal or sinus surgery or radiation treatment to the nasal area? Is there any history of photophobia? Photophobia is indicative of possible congenital glaucoma or ocular surface disease.

**EXAMINATION**

Any obvious crusting, redness or swelling of the lids is noted. The tear meniscus may be higher in the eye with CNLDO. Lid malpositions like ectropion, entropion or epiblepharon should be observed. Any facial abnormalities should be noted. Puncta should be inspected to rule out stenosis. Corneal clarity should be evaluated and the corneal diameter measured.

Pressure over the lacrimal sac may discharge mucopurulent material into the lacrimal lake and is confirmatory. A dye disappearance test can be performed. After instilling a topical anesthetic, a drop of 2% fluorescein dye is instilled into the conjunctival cul-de-sac. Excess fluorescein is wiped away. After 5 minutes, the eyes are inspected for residual fluorescein with the cobalt blue light filter on the slit lamp opened wide. Failure to clear away the excess fluorescein is indicative of CNLDO.

**DIFFERENTIAL DIAGNOSIS**

Other causes of tearing include congenital glaucoma, lid abnormalities like entropion and epiblepharon, lash abnormalities like trichiasis and distichiasis, corneal surface abnormalities and conjunctivitis or keratitis.

**MANAGEMENT**

1) **Observation and Massage**

Crigler was the first to describe lacrimal sac massage.³ This is the first line of management before probing. After cutting the nails and washing the hands, the index or little finger is placed over the common canaliculus to prevent regurgitation and the finger is stroked downward firmly, to increase hydrostatic pressure within the lacrimal sac and the nasolacrimal duct (Figure 2). About 10 strokes should be performed 2-4 times daily. The aim is to increase the hydrostatic pressure to rupture any membranous obstruction. In addition, it also empties the sac of stagnant tears, which can be a source of infection. An antibiotic drop like Vanmycetin or Ciprofloxacin is prescribed if there is mucopurulent regurgitation. 80-95% of children get cured by 1 year of age by this treatment.⁴ It is a good
idea to take a swab and send for microbiological examination. The common organisms that are isolated include Hemophilus influenzae, Staphylococcus aureus, Pneumococcus and Beta haemolytic streptococcal species.

2] Probing

Considerable controversy exists about the timing of probing. Conservative management by massage can be done safely up to 1 year of age; the reason being most of the cases (96%) will resolve within the first year of life. The success of probing falls after 1 year of age. Hence in a child 1 year of age or more, it is best to recommend probing to the parents. Success ranges between 92% – 97% if done before 1 year of age but beyond 1 year the success falls to 55% – 80%.^5,6

a) Early probing

This may be necessary if the child is very symptomatic. In addition, this is necessary in cases where any intraocular surgical intervention is necessary or there have been repeated episodes of acute dacryocystitis. Early probing prevents occurrence of fibrosis, avoids complications like infection and orbital cellulitis and suffering due to epiphora and discharge.

b) Technique

Probing is best done under sedation or general anesthesia. A cuffed endotracheal tube should be used. A decongestant nasal spray is instilled into the nasal cavity. The upper punctum is dilated using a punctum dilator. The upper canaliculus is preferred as the turn from vertical to horizontal is less acute and less traumatic. The probe is introduced vertically and the lid is pulled laterally. The probe is advanced horizontally till a hard stop is reached (Figure 3a). The lateral traction is now released. The probe is now turned 90 degrees and directed toward the NLD downward, posteriorly and laterally (Figure 3b).

Fig. 2. Technique of lacrimal sac massage: With the little or ring finger, the common canaliculus is blocked first and then pressure is applied downwards firmly over the sac to increase the hydrostatic pressure.

Fig. 3. a and b. Technique of probing: The probe is introduced through the upper canaliculus, vertically and the lid is pulled laterally. The probe is advanced horizontally till a hard stop is reached (Figure 3a). The lateral traction is now released. The probe is now turned 90 degrees and directed toward the NLD downward, posteriorly and laterally (Figure 3b).
decongestants and topical antibiotics, advised to continue massage and reviewed after 4-6 weeks.

c) Difficult probing

This is encountered if there has been a false passage or there is a tight bony obstruction. This can be circumvented by “graduated” or “stepwise” probing (where probes of progressively increasing diameters are used) or by “reaming” (where the probe is forced in a screwing fashion to enlarge the NLD). Sometimes infracture of the inferior turbinate is required if the block is beyond the NLD.

d) Repeat probing

About 5 to 10% of probings are unsuccessful and a repeat probing can be performed anytime, preferably after 6 weeks, if symptoms persist. It is successful especially where the probe passed into the nose in the first instance or irrigation fluid was recovered in the nose. Prognosis for probing decreases exponentially with the increasing number of probings and the age of the patient. Rarely, is it successful after the third time or after 3 years.

3] Infracture of the inferior turbinate

If the inferior turbinate is impacted against the opening of the NLD, fracture may work by stretching open the NLD. It is infractured towards the nasal septum using the blunt end of a Freer elevator (Figure 4).

4] Nasolacrimal duct intubation

Intubation using a silk seton was described as early as 1909, by Berry. Quickert and Dryden described intubation with silicon tubing in 1970. It is recommended that after a failed second probing or when the patient is older than 18 months of age or when there is canalicular stenosis, silicone intubation should be performed. The silicon tube acts as a stent and creates a normal anatomical pathway. Silicone is well tolerated, is flexible and knots easily. Retrieval of the distal end is possible under direct visualization or using a Crawford hook, when stents with olive tips have been used (Figure 5). Performed alone, success rate has been reported to be 82.5%, while in combination with infracture of the inferior turbinate it can be as high as 97%. The tube can be left in place for 3 to 6 months. Complications are maximum in the first 3 months, tube prolapse being the most common. Others include punctual stretching, infection, corneal abrasion, tube dislodgement and breakage. The potential for canalicular trauma and subsequent stenosis, is probably the most important disadvantage.

5] Balloon catheter dilatation

This is a useful adjunctive procedure in cases with incomplete NLD obstructions, where probing has failed especially in children older than 13 months of age.
Fig. 6. Technique of balloon catheter intubation (using Lacricath catheters). The balloon is inflated to widen the NLD and the junction of the lacrimal sac and the NLD.

Becker described this technique in 1991. After punctual dilation, probing is done. The balloon catheter is introduced through the upper punctum. The outer diameter of the balloon is 2 mm in children below 33 months of age and 3 mm in older children. The balloon length varies from 13 to 15 mm. The balloon is introduced until it is in the NLD. The balloon is now inflated to a pressure of 8 atmospheres for 90 seconds and deflated for 10 seconds (Figure 6). A repeat inflation up to 8 atmospheres is done for 60 seconds. The balloon is now retracted 5 mm, to lie at the junction of the sac and the NLD and two more inflations are performed. Complications with balloon dacryoplasty are fewer than intubation. It is minimally invasive, has more than 90 % success rate and does not leave any external scars.

6] **External dacryocystorhinostomy (DCR)**

Indications for DCR include failed probing or intubation and cases of severe craniofacial anomalies. Anatomical considerations include anteriorly placed ethmoid cells, flatter anterior and posterior lacrimal crests, shallower lacrimal fossa, rapidly growing facial bones and the exuberant healing response. It is preferably done beyond the age of 1 year, usually by 3-4 years of age. The ostium should be at least 1 cm in diameter. Silicone stents should be used if there has been a history of acute dacryocystitis or in cases with canalicular stenosis. Failures usually occur because of anatomic obstruction by granulation tissue. Success varies between 79-96%.

7] **Endoscopic dacryocystorhinostomy**

Endoscopic DCR has been performed in children with good results comparable to that obtained in adults. It can be done to avoid an external scar. Success rates of 76-88 % have been reported. The advantage is concomitant sinonasal procedures can be performed.

**Summary**

In children less than 12 months, sac massage should be performed, unless there are strong reasons for early probing. In children reaching about 12 months of age, probing needs to be done. Infracture of the inferior turbinate can be attempted, if the turbinate is impacted against the lateral nasal wall. Repeat probing can be attempted in unresolved cases. If probing fails, silastic intubation can be considered. DCR is recommended where silastic intubation fails, in those with bony obstructions and those with craniofacial anomalies and is preferably done about 3 years of age.

**References**


(Contd. from pg. 190)

Around 1247 Bacon underwent a spiritual transformation and became a Franciscan Friar. His feverish activity, amazing credulity, superstition, and vocal contempt for those not sharing his interests displeased his superiors in the order and brought him under severe discipline. They issued an order which prevented him from publishing without prior permission.

Bacon overcame this order by writing to Pope Clement IV who he had known in Paris before his papacy. He wrote to the Pope that he had suggestions on Mathematics, Languages, Astrology and the Natural World, which he felt would help confirm the Christian Faith. The Pope’s command to be given these suggestions enabled Bacon to work secretly ignoring the order of his superiors.

Between 1266 and 1267, Bacon wrote his three main works: the Opus Majus (Major Work), the Opus Minus (Minor Work) and the Opus Tertium (Third Work).

His Opus Majus is an Encyclopaedia of all Science and deals with Mathematics, Optics, Alchemy and Astronomy. It was written to persuade the Pope of the urgent necessity and manifold utility of the reforms he proposed.

In the Opus Minus he attacked the Church. He argued that in “every town, in every village... there is an infinite corruption, beginning with the highest level.” He claimed that priests were eager to “enrich themselves indifferent to the care of souls... the monks, in their turn, are no better, and I exempt no Order.”.

He wrote many other books including “On the Marvellous Power of Art and Nature”, “On Mirrors”, “Metaphysical” and “On the Multiplication of Species”. In his Perspectiva (Optics) he proposed a model of philosophical study applying linguistic and scientific knowledge to understand Theology.

He rejected the blind following of prior authorities, both in Theological and Scientific study. His writings are a passionate tirade against ignorance. He combined his attack upon the ignorance of his time with suggestions for the increase of knowledge. He set out his own new model for a system of philosophical studies that would incorporate language studies and science studies then unavailable at the Universities.

The death of Pope Clement in 1268 extinguished Bacon’s dreams of gaining for the Sciences their rightful place in the curriculum of university studies.

In 1277, Bacon was condemned to prison by his fellow Franciscans for heresy. He remained chained up in a tiny cell for almost fifteen years. During this period he managed to write only one book, Collected Study of Philosophy which though incomplete shows him to be as aggressive as ever.

Roger Bacon was released from prison just a few months before his death on 11th June, 1292. Despite his denunciation of the Church, he was buried at Greyfriars, the Franciscan Church in Oxford.

After his death, his true greatness was recognized and earned him sobriquet “Doctor Mirabilis” (Wonderful Teacher)
Basic Concepts of Primary Exodeviations

Dr. Leila Mohan MS DO

Introduction
Strabismus occurs among 1-4% of the children worldwide and strabismic amblyopia is a major cause of unilateral visual impairment among all age groups. Though it has been reported by many that esodeviations are seen 3 times more commonly than exotropia in European and American countries, exodeviations or outward deviation of eyes, are more commonly met with in Asia, Africa and the Middle-East with ratio varying from 2:1 to 3:1. Nepal has a reported incidence of exotropia in 76% of strabismus. In India exotropia is more commonly seen than esotropia. Jenkins has proposed the concept that the more equatorial a country is, the higher the incidence of exotropia. The dazzle produced by bright light is supposed to disrupt fusion, inducing tropia. Primary exotropia is more common among females. It is more symptomatic than esotropia since the intermittent form is the commonest and the phoria to tropia phase produces lot of sensory phenomena like diplopia and blurring of vision.

Small asymptomatic exophorias are not uncommon in our clinical practice, in the normal population. Variable transient forms of exotropias are seen in the newborn which disappear by 4-5 months. Exodeviations are much more common in the intermittent form unlike in esotropia. Primary exodeviations are the most common of exotropias, which constitute a progressive spectrum of deviations from exophoria to constant exotropia, through a period of intermittency i.e. intermittent exotropia. The progression from phoria to tropia is influenced by the decreased tonic convergence with increasing age, the development of suppression, loss of accommodative power and increasing divergence of orbit with aging. Von Noorden has reported that 75% of the intermittent exotropes progressed to constant exotropia. Intermittent exotropia constitute 50-90% of exodeviations.

Causes of primary exotropia
Primary exodeviations are caused mainly by a combination of innervative and mechanical factors, added to which there is an interplay of other factors. An innervational imbalance which upsets the reciprocal relationship between active convergence and divergence mechanism has been the basis of classification of Duane, who considered divergence an active process and put forth his theory more than 100 years ago. Later, Weiss and Bielschowsky, showed that anatomical and mechanical factors like growth and depth of orbit, insertion of extraocular muscles etc. also play a role, this being supported by the fact that there is a higher prevalence of exodeviations in craniofacial dysostosis like Crouzon’s disease due to shallow and laterally directed orbits. Current concepts combine these two theories. There are other factors which contribute to progression to constant tropia.

Defective fusion capability
Worth has pointed out the role of absence of fusion faculty in causation of any strabismus.

Refractive errors
Constant understimulation of convergence in uncorrected myopes, anisomyopes and anisoastigmatics
facilitate progression due to absence of fusional reflexes. Even high hypermetropes who have a low AC/A ratio, give up attempts to clear vision and can lead to development of exotropia.

Bitemporal hemiretinal suppression – that is, the ability to suppress temporal field of vision has been postulated to favour progression.

AC/A ratio - when high produces a true divergence excess exotropia and when low, convergence insufficiency.

Heriditary factors-Knapp, Burian, Spivey have all reported the higher incidence of intermittent exotropia in families. The refractive status also may be inherited. Proprioceptive reflexes-Mitsui et al have shown that proprioceptive reflexes from extra-ocular muscles have a role. The demonstration of Magician’s forceps phenomenon is in favour of this postulation. After anaesthetizing the eye, the fixing or dominant eye is held by the nasal limbus with a forceps and forcibly adducted. It is seen that the divergent eye makes an adduction movement with the disappearance of the strabismus. Care should be taken to see that the other eye does not take up fixation.

Classification

There are different classification systems in primary exotropia, all of them taking the distance near disparity into consideration.

Duane’s Classification-

I Basic type where deviation for distance and near are equal
II Divergence Excess type where distance deviation is more than near deviation
III Convergence Insufficiency type where near deviation is greater than distance deviation.

Burian’s Classification-takes into consideration, the exuberant fusional convergence reflexes which may mask a basic deviation for near i.e. produces a simulated divergence excess pattern. This is the more generally accepted classification. Thus

I Divergence Excess pattern where distance deviation is >10pd larger than at near.
II Basic –where Distance deviation equals or is within 10 pd of near deviation.

III Convergence Insufficiency pattern where near deviation is >10 pd greater than distance deviation.

IV Simulated or pseudo-divergence excess-where distance deviation is more than near deviation, but near deviation equals or is within 10 pd of distance deviation after monocular occlusion for 30 to 45 mts. This is due to the tonic fusional convergence reflexes which gives an apparent smaller deviation for near, but which is dissipated by the monocular occlusion.

Kushner’s classification takes into account in addition to the fusional convergence reflexes, the accommodative convergence reflex as well, which is eliminated by the use of +3D lenses after monocular occlusion before measuring deviation again. This gives an etiological evaluation to guide in the management

Kushner classification

<table>
<thead>
<tr>
<th>Distance/near disparity</th>
<th>Diagram</th>
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<tbody>
<tr>
<td>Distance deviation is &gt;10 pd larger than near.</td>
<td><img src="image" alt="" /></td>
</tr>
<tr>
<td>Distance deviation equals or is within 10 pd of near deviation.</td>
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High AC/A ratio-Kushner measured the gradient AC/A and reported that >90% of divergence excess show normal AC/A. The small percentage that did have high AC/A ratio can develop overcorrection for near and may need bifocal lenses postoperatively.

Convergence insufficiency (CI) is characterized by a reduced NPC, a low AC/A ratio, a high exophoria or intermittent exotropia for near and a reduced positive fusional vergence. They have asthenopic symptoms for near.
Characteristics of intermittent exotropia (X(T)) DE

Most DE and basic exotropes are intermittent. Since X(T) is the most prevalent exodeviation-50-90% and since the management is critical before it becomes constant, it is important that we recognize and treat the condition. The age of onset is usually before 18 months. They have an initial stage of phoria which later become intermittent and then progress until about 6 years of age, when it becomes more noticeable. Initially deviation is seen for distance only and later progresses for near also. Only 6% are first observed after 5 years of age. It is more prevalent in females. They have equal and good visual acuity in both eyes, and good stereopsis when ortho and may have ARC when deviated. They have normal NPC and adequate convergence amplitude.

Symptoms

A common complaint for which child is brought to the clinic is for frequent closure of eyes or one eye in bright light especially in the sun. This is due to the breakdown of fusion in bright light experiencing diplopia. It may be described as a photophobia or rubbing of the eyes by the parent and may be taken as an allergic symptom. Parents also complain of deviation of eyes when watching TV. Transient blurring or diplopia and vague discomfort may be complained of. Asthenopic complaints are not uncommon in early decompensating phase. Micropsia is a rare complaint of overconvergence by accommodation which they utilize for correcting the deviation. Symptoms become less as they become constant.

Sensory Adaptations

Though bifoveal fusion with good stereopsis is seen during orthophoric phase, various sensory phenomena occur during decompensation. Temporal Hemiretinal suppression during the tropic phase they may develop temporal hemiretinal suppression. Dual retinal correspondence with ARC during the tropic phase and NRC during orthophoric phase is not uncommon. They may also have panoramic viewing with extension of field during the tropic phase. Monofixation and amblyopia are very rarely seen. Patients who develop deviation after visual maturation, experience diplopia.

Factors contributing to Progression

As we have already seen the contributory factors in the etiology of exotropia influence the progression to from intermittent to the constant exotropic phase. Decreasing tonic convergence with increasing age is a major factor. As the frequency of tropic phase increases, suppression of the temporal half of retina sets in which favours a speedy progression to tropia due to lack of fusion. Loss of accommodative power and increasing divergence of orbits also contribute. Kushner has shown that 75% progress to constant XT.

Assessing Control of Progression

Most clinicians follow the child by assessments of control in the clinic. Calhounz et al has described 4 phases of exodeviations.

Phase I- Exophoria for distance, ortho at near-asymptomatic.
Phase II-Intermittent exotropia for distance, orthophoria or exophoria for near-symptomatic for far only
Phase III-Exotropia at distance, Exophoria/intermittent exotropia for near-Binocular vision for near, suppression scotoma for far.
Phase IV-Exotropia for far and near- no binocularity.
Exophoria for near, exotropia for far.

Since the clinic control does not take into account the duration of tropic phase and the fact that there is no standardization as to when to intervene, a novel method was put forth by H. Haggerty and Richardson.
The **Newcastle Control Score** seems to be useful in grading the severity of intermittent exotropia and as a criteria for surgical intervention.

The Newcastle Control Score takes into consideration the subjective and objective criteria to grade severity and quantify progress. The score is the sum total of scores obtained in home control and in the clinic for near and far. Patients with a score of 3 or more are considered to need surgical intervention. It is a consistent method of rating severity and enables one to easily monitor progress of X(T).

**Newcastle Control Score**

<table>
<thead>
<tr>
<th>Score Component</th>
<th>Home control</th>
<th>Clinic control near</th>
<th>Clinic control distance</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Score</strong></td>
<td>Squint / monocular eye closure never noticed</td>
<td>Manifest only after CT and resumes fusion without need for blink or refixation</td>
<td>Manifest only after CT and resumes fusion without need for blink or refixation</td>
</tr>
<tr>
<td>0</td>
<td>Squint / monocular eye closure seen occasionally (&lt; 50% of time) for distance</td>
<td>Blink or refixate to control after CT</td>
<td>Blink or refixate to control after CT</td>
</tr>
<tr>
<td>1</td>
<td>Squint / monocular eye closure seen frequently (&gt; 50% of time) for distance</td>
<td>Manifest spontaneously or with any form of fusion disruption without recovery</td>
<td>Manifest spontaneously or with any form of fusion disruption without recovery</td>
</tr>
<tr>
<td>2</td>
<td>Squint / monocular eye closure seen for distance &amp; near fixation</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>+</td>
<td>Clinic control distance</td>
<td>Clinic control near</td>
<td>Clinic control distance</td>
</tr>
<tr>
<td>0</td>
<td>Manifest only after CT and resumes fusion without need for blink or refixation</td>
<td>Manifest only after CT and resumes fusion without need for blink or refixation</td>
<td>Manifest only after CT and resumes fusion without need for blink or refixation</td>
</tr>
<tr>
<td>1</td>
<td>Blink or refixate to control after CT</td>
<td>Blink or refixate to control after CT</td>
<td>Blink or refixate to control after CT</td>
</tr>
<tr>
<td>2</td>
<td>Manifest spontaneously or with any form of fusion disruption without recovery</td>
<td>Manifest spontaneously or with any form of fusion disruption without recovery</td>
<td>Manifest spontaneously or with any form of fusion disruption without recovery</td>
</tr>
</tbody>
</table>

The score varies from 0-7; score of 3 or more significant

**Congenital XT**

Differential diagnosis-Unlike pseudoET, pseudoXT, is not common and if a mother complains of eye deviating out, it is unlikely that it is pseudoexotropia. But there are a few conditions which can give an apparent XT. Hypertelorism, large angle kappa due to a temporally dragged macula due to ROP or a toxoplasmic scar are some.

Congenital exotropia-Primary acquired exotropia has to be differentiated from congenital exotropia which is a rare condition with onset before 6 months of age and show large angle of 40-80 pd. They may have associated DVD or IOOA. They are often associated with neurological problems like cerebral palsy, prematurity etc in 60%. So we need to look out for these. They are rarely amblyopiogenic. Here treatment is surgical.

**Resolving cong.IIIrd nerve palsy(Left)**

Congenital IIIrd nerve palsy often resolves to an extent with minimal or no ptosis and may have to be differentiated. Exotropic Duane's syndrome i.e., Duane type II is another condition to be differentiated. Transient exotropia of the newborn disappears by 4-5 months.

**Evaluation**

History of complaints as in any strabismus, age of onset, any glasses, previous surgery, family history, any neurological problems -are all important. Special attention is given to the duration of control at home-whether dissociated for > 50% of the time.

Refraction under full cycloplegia is important. Some children may show pseudomyopia due to overaccommodation to control tropia.

**Evaluation of deviation**

Three factors are important in assessment. Fixation for near and distance, fusional vergence and accommodation. The cover/uncover test, cover test, and the alternate cover tests are invaluable in that they detect presence or absence of bifoveal fusion and characterize vergence movement if present- the information of which is indispensable in diagnosis and management of X(T).The cover tests are done in the primary position, for distance and near.

The Uncover Test-The importance of this test is that in the presence of 6/6 vision in both eyes the presence of a real life fusional vergence is positive proof of bifoveal fixation. The character of refusion movement is observed e.g. whether fusing without a blink etc. This inference does not need further confirmation by sensory tests and can be used for management decisions.

**Alternate cover test**

The fusional vergence in some exophorics may be very strong and difficult to dissipate. Here alternate cover test is done switching between the eyes rapidly to
dissociate any fusion in between. Even if the deviation could not be detected on the first visit, if a mother says his eye deviates outwards on watching TV etc, every effort should be taken to dissociate and do cover test again.

**Measurement of deviation**

Due to variable angle, routine PBCT may not be reliable. Prolonged alternate cover test to suspend tonic fusional convergence is necessary. Measurement should be done for near, distance, ie 6 meters and for distant far i.e. > 50 ft.

**Distant far deviation** It has been found that measurement of distant far as when looking out of the window gives larger deviation than for routine 6 meters and when surgery is planned the maximum deviation for distant far should be targeted.

**Patching**

If after prolonged alternate cover test there is a disparity of > 10 pd between distance near measurements, then a patch test i.e. measurement of deviation for far and near after monocular occlusion for 30-45 minutes should be done. This is to overcome the influence of fusional convergence reflexes. Controversy exists regarding duration of time of patching. Kushner in a comparative study has found no difference by patching for more than 30-45 minutes.

**+3D add test**

After patch test while still dissociated, +3 D add given to both eyes and deviation measured. If XT at near increases by 20 pd or more it is a case of DE due to high AC/A ratio.

**Lateral and vertical incomitance**

Ocular movements and deviations in different gaze positions are evaluated. Size of deviation may differ in lateral gaze. It has been found that 6-25 % show a smaller deviation in lateral gaze. As shown by Repka et al it may be an artifact in measuring deviation in lateral gaze or a true lateral incomitance, in which case the LR recession has to be less on that side or overcorrections can occur. Vertical incomitance with A, V or X pattern may be seen, V pattern being the most common. Oblique overaction may or may not be present.

**Binocularity**

Is tested with Worth 4 dot test-distance test showing central fusion and near testing showing peripheral fusion. Bagolini’s glasses show sensory status under natural viewing conditions and is a better assessment for suppression, suppression scotoma and ARC. BSV can be assessed at the synoptophore and stereoacuity measured.

**Bagolini’s glasses**

Objective methods of assessing distance stereocuity-by Mentor B-VAT random dot and contour circle tests are supposed to be good in assessing binocularity because near stereopsis does not correlate with the degree of control.
Management Decisions in X(T) present a challenge unlike other childhood onset strabismus. Timing of interference is controversial due to frequent fusional capabilities. Decisions to intervene are controlled by the size of the deviation and control of deviation which is the most important.

Nonsurgical methods of Treatment of X(T) are not found to be effective in achieving a cure. But deviations < 20 pd and the very young children are better managed nonsurgically

Maximize visual acuity: Anisometropia, astigmatism, myopia and even hypermetropia can impair fusion. If visual acuity is not affected by a hypermetropia, it is better left alone to stimulate accommodation unless > 4-5 D. Myopia is fully or overcorrected. Treatment of amblyopia may be necessary.

Overcorrecting minus lenses may be used to stimulate accommodative convergence thus favouring fusion. It is particularly useful in patients with high AC/A ratio. Young children tolerate 2-3 D or more. When there is a mixed astigmatism transpose in such a way that maximum minus sphere is given. e.g. + 1 D/-2 Dcyl at 180 may be given as -1/+2 Dcyl at 90 if visual acuity is not compromised.

Orthoptic therapy - is meant as a supplement to surgery and not a substitute. It makes the patient aware of manifest deviation and teaches him to control it. Diplopia awareness, antisuppression therapy and convergence exercises are the modes of orthoptic therapy used in the convergence insufficiency type of XT. Red lens and TV trainers may be used to eliminate suppression scotoma. Occlusion / penalization of the dominant eye or alternate eyes to prevent or eliminate suppression scotoma can be done with caution before surgery.

Prisms- Base in prisms may be instituted to enforce bifoveal stimulation in deviations <20 pd. and in undercorrections. In larger deviations, prisms are used only for short term preoperative enforcement of fusion. Prisms should be used with caution since in the presence of ARC, the deviation may increase due to prism adaptation.

Surgical Treatment

There exists a controversy regarding timing of surgery in X(T) though the definitive treatment is universally considered surgical. They have a better chance for binocularity and stereopsis. Though Knapp and advocates uphold early surgery in children, they do caution that in visually immature children overcorrection can lead to monofixation and amblyopia. Jampolsky advocates delayed surgery for accuracy in measurement and avoiding consecutive esotropia. Most of the intermittent exotropes progress to constant XT.

Signs of progression are loss of fusional control as evidenced by increase in frequency of tropic phase, development of secondary convergence insufficiency, increase in size of basic deviation, development of suppression symptomatically as evidenced by absence of diplopia during the manifest phase and objectively. The Newcastle Control Score can be followed up and if the score is >3 surgery undertaken.

Type of Surgery Bilateral LR recessions is the standard surgical technique for true divergence excess(DE) XT. In Simulated DE type and basic type either Bilateral recessions or unilateral recession/resection may be undertaken. There are advocates for both procedures. In convergence insufficiency type, bimedial resections are the ideal. In patients with high AC/A ratio, who constitute a small number, posterior fixation – Fadenisation – of MR is done either during lateral rectus recession or during a recess/ resect procedure. This fully corrects the distance deviation at the same time minimizing the risk of consecutive esotropia for near. Intentional overcorrection during the immediate postoperative period has been recommended by Cooper as early as 1966. 5-6 pd of overcorrection is considered ideal and immediate postoperative diplopia is considered even therapeutic to prevent suppression. But in young children it can lead to amblyopia. Also in late onset XT, who already experience diplopia, this may produce severe symptomatic diplopia, so better aim for orthophoria in these patients. For overcorrection, bilateral recessions are better as these tend to straighten out during the early postoperative period unlike in recession/resection procedures where it can persist. Adjustable sutures have an upper hand in this regard. In case of lateral incomitance, the LR recession has to be reduced. In small XT, successful unilateral LR recession has been reported.

Constant XT- Only 20% of all exotropes are constant, and are mostly alternating. Many show ARC.
is surgical. Possibility of diplopia which is usually transient should be explained. In large XT, 3 or more muscle surgery with adjustable sutures ideal.

**A and V patterns** - In the absence of significant oblique overaction, horizontal muscle displacement suffice. If there is Inferior oblique overaction (IOOA) in a V pattern XT, then IO recession is done. In a pattern XT with Superior oblique overaction (SOOA), SO weakening with or without tendon spacers may be necessary. A pattern is rare. An X pattern is not uncommon in longstanding XT due to tethering of lateral rectus and lateral rectus recession suffice.

**Consecutive esotropia** - If the overcorrection lasts beyond 4-6 weeks it becomes a consecutive ET. Upto 10 pd of overcorrection usually straightens out by 6 weeks. Preoperative amblyopia, suppression, recess/resect procedure, all contribute to the development of consecutive ET. In small ET, prisms are given in the immediate postoperative period and limited alternate patching tried. If not responding, surgery will be necessary. Unilateral or bilateral LR advancement or unilateral or bilateral MR recessions are the options.

**Residual XT** - If less than 15 pd, may respond to nonsurgical treatment - fusional exercises, prisms, minus lens etc. >15 pd of deviation will necessitate surgery. Wait upto 12 weeks before reoperating. If < 6 mm bilateral recessions done before, re-recession is better than resection.

**Treatment Outcomes/Goals** - The variability of presentation, type of surgery and lack of standardization as to timing of intervention all contribute to the variable outcome of surgery. The goal of surgery is to restore alignment and to maintain or restore binocular function. Ideal goal is absence of tropia for far and near though success of surgery is defined as alignment within 8 pd of exotropia. Surgery following preoperative occlusion and orthoptic treatment yields the best results.

Kushner and Moton, Morris et al, Scott and associates all have reported an increase in binocularity even in adults as determined by WFD test, or Bagolini’s glasses which was in turn, correlated with longterm post-operative alignment. Since undercorrections are common, maximum deviation for distant far should be targeted and longterm follow up undertaken for 4-5 years.

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A Case of Optic Disc Pit Maculopathy

Dr. Archis Shedale MS, Dr. Mahesh G MS FRCS Ed, Dr. A. Giridhar MS, Dr. Sachin Fegde FRCS Ed, Dr. Savita Bhat MS

Case Report

A 33-year-old woman presented to us with complaints of blurred vision in left eye of 2 months duration. Her best corrected visual acuity was 6/6 N6 in the right eye and 6/36, N12 in the left eye. Anterior segment examination was unremarkable. Amsler grid examination revealed a central scotoma in left eye. Indirect ophthalmoscopy and slit lamp biomicroscopy showed optic disc pit along with associated serous macular detachment (Fig 1A). She underwent fundus fluorescein angiography, which showed pooling of dye with no point leak (Fig 1B). Optical coherence tomography demonstrated convex schisis of the outer retinal layer (Fig 1D and E) and connection of optic disc pit with intra-retinal schisis. (Fig 1D) She received focal laser photocoagulation to the left eye with green laser in the peripapillary region. Laser was applied to the peripapillary retina like a barrage with a spot size of 100 microns and a power of 200 mW. Also 0.3 cc perfluoropropane gas ($\text{C}_3\text{F}_8$) was injected intravitreally under aseptic precautions. She was asked to maintain a prone position with face down for one week. Patient was followed up over a period of 12 months and best-corrected visual acuity remained stable at 6/36, N12.

Fig. 1. A: Pretreatment colour photograph showing optic disc pit with serous detachment of macula and chronic cystoid changes. B: FFA before treatment showing pooling of dye within the serous detachment without any point leak C: Post treatment photograph showing resolution of macular detachment D: OCT before treatment showing schisis cavities within the retina which is suggestive of chronic maculopathy. Also there is connection of optic disc with intraretinal schisis cavities E: OCT before treatment showing the cystoid changes at the fovea with an epiretinal membrane F: OCT after treatment showing reduction in macular thickness and resolution of schisis cavities in the retina
Colour photograph after 12 months revealed flat macula with resolution of serous macular detachment (Fig 1C). Optical coherence tomography was repeated after 12 months which showed few intraretinal cysts with near normal contour of retina (Fig 1F).

**Discussion**

Optic disc pit is a rare congenital abnormality that is frequently associated with macular detachment. The macular involvement occurs in 25% to 75% of eyes with optic disc pit\(^1\). Various mechanisms have been reported to explain the serous macular detachment in patients with optic pits including vitreous\(^2\) and cerebrospinal fluid leakage\(^2,3,4\) through the optic pit and from there into the sub-retinal space.

The best corrected visual acuity on presentation in our case was 6/36, N12 whereas Sobol et al\(^4\) in analyzing 15 patients with optic disc pit, found that most eyes presented with visual acuities of about 6/12 to 6/18. Theodossiadus\(^5\) opined that patients with optic disc pits present later in the course and their macular detachments when their visual acuities are worse than 20/70 and similar observation was made in our case. Hassenstein A and Richard G\(^6\) analyzed 8 patients with optic pit maculopathy by optical coherence tomography. They demonstrated retinal detachment with a typical convex schisis of the outer retinal layer and also neurosensory detachment with and without intraretinal cystoid formation in their cases. OCT of our patient on presentation had similar picture with intraretinal schisis. Recent long term studies confirm the earlier impression that untreated macular detachments caused by optic disc pit have an overall poor prognosis\(^5\). Hence we treated our patient by combining two modalities, namely laser photoagulation and pneumatic displacement of the retinal elevation by gas injection. Several series have favourably compared the outcome of photoagulated eyes with untreated eyes in the resolution of the serous macular detachment and in final visual outcome.\(^8\) More recent attempts to combine photoagulation therapy with posterior vitrectomy and gas fluid exchange have shown more encouraging long-term visual outcomes.

**Conclusion**

Barrage laser photoagulation to the peripapillary retina along with intravitreal gas tamponade followed by prone positioning is a safe and effective method of treatment for optic disc pit with maculopathy.

**References**

Acute Retinal Necrosis Following Herpes Simplex Encephalitis

Dr. Valsa Stephen MS DO DNB, Dr. Biju John C MS DNB FRCS, Dr. Sonia Rani John DNB, Dr. Meena Chakrabarti MS DO DNB, Dr. Arup Chakrabarti MS DO

Acute retinal necrosis is a severe ocular inflammatory syndrome associated with a very poor visual outcome. Though it is known to occur occasionally in association with or shortly after herpetic encephalitis, ARN occurring years after encephalitis has been rarely reported. Here we present a case of unilateral ARN occurring 5 years after herpes simplex virus encephalitis.

Case Report

A 14 year old male presented with redness and watering of his right eye of a week’s duration. He had been on local steroids and mydriatics for 3 days. He gave a history of herpes encephalitis, with post viral demyelination, cystitis and septicaemia 5 years back. At that time he had presented with headache, fever, vomiting, slurring of speech, drooling of saliva and inability to feed. Though blood and cerebrospinal fluid titres of antibodies to herpes simplex virus were negative, he was diagnosed to have herpes encephalitis on the basis of CT scan and MRI findings which showed diffuse brain oedema. He responded well to antiviral therapy and had been asymptomatic since then.

On presentation, the best corrected visual acuity was 6/18, N8 in the right eye and 6/6, N6 in the left eye. Anterior segment showed evidence of anterior uveitis in the right eye with keratic precipitates, 2+ cells and 1+ flare. Left eye was normal. Dilated fundus showed vitritis with large whitish yellow confluent retinal infiltrates and vasculitis in the periphery of the right eye.

(a) showing vitritis and normal posterior pole right eye (b) showing confluent retinal infiltrates and vasculitis in the periphery of the right eye

Fig 1. (a) showing vitritis and normal posterior pole right eye (b) showing confluent retinal infiltrates and vasculitis in the periphery of the right eye

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infiltrates and vasculitis in the retinal periphery in the right eye (Fig 1 a & b). The retina in the left eye was normal.

A diagnosis of acute retinal necrosis was made and he was started on T. Acyclovir 800 mg 5 times and T.Wysolone 60 mg daily along with topical steroids and mydriatics. Investigations performed were IgG and IgM antibody titres for Herpes Simplex virus, Cytomegalovirus, Varicella Zoster and Human Immunodeficiency virus. He was advised review every 2 days. Investigation results revealed positive antibody titres for IgG and IgM for HSV and IgG for CMV. The lesions were found to be remaining status quo without any increase in size (Fig 2 a & b).

He was kept on regular follow up with Acyclovir and steroids in tapering dose once the lesion started resolving.

One month later best corrected visual acuity was 6/9, N12 in the right eye and 6/6, N6 in the left eye. Fundus showed resolved ARN with healing vasculitis Fig (3 a & b).

He was advised to stop T. Acyclovir and Wysolone and to continue topical steroids and topical flurbiprofen eyedrops. However one month later he once again presented with recurrence of panuveitis and vision drop to 6/18 and had to be restarted on steroids and Acyclovir. Despite treatment, the vitritis increased though retinal lesions did not recur. (Fig. 4 a & b). An epiretinal membrane had formed at the macula. A therapeutic pars plana vitrectomy under general anesthesia was done along with peeling of epiretinal membrane (Fig. 5) and prophylactic barrage laser of the peripheral retina. The vitreous aspirate was sent to

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**Fig. 2 a & b showing lesions remaining status quo without increase in size or number**

**Fig 3 a & b showing resolved ARN with healing vasculitis after one month Right eye**
detect IgG and IgM antibodies for Herpes Simplex virus, Cytomegalo virus, Varicella Zoster virus. IgG antibody for HSV1 was found to be positive. Postoperatively the eye was quiet with clear fundus view and normal retina. On review after 1 month the eye remains quiet with no recurrence and a best corrected visual acuity of 6/9, N8. (Fig. 6 a & b)

**Discussion**

ARN (Acute Retinal Necrosis) was first described by Urayama et al. in 1971 in 6 patients with severe intraocular inflammations, retinal vascular sheathing and large white confluent retinal infiltrates which progressed to rhegmatogenous retinal detachment.
Acute retinal necrosis syndrome or ARN is characterised by initial onset of episcleritis or scleritis, periorbital pain, and anterior uveitis. This is followed by decreased vision resulting from vitreous opacification, necrotizing retinitis and in some cases optic neuritis or neuropathy. The retinitis appears as deep multifocal, yellow-white patches in the peripheral fundus which become concentrically confluent and spread towards the posterior pole; the macula is frequently spared. An active vasculitis is present with perivascular hemorrhages, sheathing and terminal obliteration of arterioles by thrombi. This phase of active retinitis usually lasts for 4 to 6 weeks during which time an exudative retinal detachment may occur.

With resolution, pigmentation of the lesions begin at their posterior margins, leaving a scalloped appearance. Retinal breaks may develop at the junction of normal and necrotic retina. A rhegmatogenous retinal detachment or tractional retinal detachment due to organisation of vitreous inflammation and proliferative vitreoretinopathy may occur.

The contralateral eye is involved in about 36% cases usually within 6 weeks of onset in the other eye though it may be affected after as long as 34 years. ARN commonly occurs in otherwise healthy patients of either sex and of any age. ARN can also occur in immunocompromised patients. Immunocompromised patients have been described to have skin manifestations of zoster and CNS involvement.

Considerable evidence points to one or more members of the herpes virus family in the etiology of ARN syndrome. Varicella-zoster virus, cytomegalovirus, herpes simplex have all been implicated. Some studies suggest that Varicella-zoster virus and Herpes simplex virus type-I cause ARN in patients older than 25 years, whereas herpes simplex virus type-2 causes acute retinal necrosis in patients < 25 years.

Retinal necrosis in ARN probably is the result of multiple factors like direct lytic viral infection of retina, immune complex disease mediating an obliterative vasculitis and vitreous inflammation and traction. This case validates the theory of brain to eye transmission of the virus through the optic nerve. Thus, following encephalitis retinal neurons may function as a reservoir for HSV that can be reactivated to cause ARN several years later. The occurrence of ARN would support the etiological suspicion of the previous encephalitis.

Treatment is with acyclovir which has good activity against HSV and VZV. It can hasten resolution and prevent contralateral spread. Dosage is 1V 500 mg/m^2 every 8th hr for 10-14 days or orally 800 mg x 5 times for 6 weeks. Valacyclovir orally 1gm tds or famicyclovir 500 gm tds have the same efficacy and lesser side effects. In case of severe progression despite acyclovir, ganciclovir or foscarnet can be given. Systemic corticosteroid therapy in a dose of 0.5-1 mg/kg is given after 24-48 hours of IV acyclovir.

Aspirin is given in extensive arteritis and retinal vascular occlusion. Other modalities of treatment are intravitreal injection of antiviral, barrage laser photocoagulation, prophylactic scleral buckling with vitrectomy, endolaser photocoagulation and silicone oil tamponade in eyes at risk etc.

ARN resolves spontaneously in 2-3 months with or without therapy. However retinal detachment with large breaks with rapid progression of the disease can occur in untreated cases.

In this case, a previous history of herpetic encephalitis was useful in making the diagnosis of acute retinal necrosis and initiating treatment at the earliest. Thus though the disease is associated with poor visual prognosis, prompt treatment could prevent complications from occurring as in this case.

**Conclusion**

Pediatricians and physicians should be aware of this entity and be alert to recurrences that may be delayed by years. Similarly previous history of central nervous system infection can help to clinch the diagnosis of ARN. Earliest diagnosis and treatment is important to prevent complications of ARN.

**References**


### HUMOUR IN OPHTHALMOLOGY

#### In the lighter vein

Dr. Yarma MS

Dr. Bhikhalal S. Patel’s name may not be in any Ophthalmic Hall of Fame. But I am sure; it will be engraven in the minds and hearts of generations of alumni of B.J. Medical College, Ahmedabad.

Affectionately called “Bhikhu-bhai” by all and sundry, he was the honorary head of the second unit of Ophthalmology of that 128-year old college during seventies and eighties. He was not a great clinician or surgeon; nor was he a great teacher. In fact, he never taught us any Ophthalmology; he taught us about Life. And he taught not by pedagogy; but by his own life. He was the master of patient management. The way he built rapport with; consoled and comforted; cajoled and convinced; humoured and honoured patient after patient was a treat to watch. I once had the fortune to accompany him on a house visit; or so I thought.

One day as I was hurrying back to the OPD after a mid morning coffee break, he asked me to go with him to a nearby house to see an old patient of his. Since only he had an air-conditioned car in the department in 1980, I was quick to grab the chance. He drove to the nearby Asarva Housing Colony and stopped in front of a ‘two room – kitchen’ apartment. There were about fifteen people in the house. Their reaction was astounding. It was as if the Lord God himself had stepped into the house. And as God’s assistant, a little reflected glory fell on yours truly too. When the head of the house assured him that everything was ready, Dr. Patel asked me to fetch ‘that thing’ from his car. ‘That thing’ proved to be a stainless steel basin with five or six surgical instruments and a big ball of cotton in it. While we were served too sweet tea, the basin was filled with some water and set to boil on the stove. Then it struck me! He was going to do a cataract operation!

While I held a feeble torch, and all the family members crowding around, he did an ICCE/PI (knife section, no sutures!) in about fifty seconds flat. As soon as the cataract was out (capsule forceps) he gave it to the nearest relative saying “Lo! Mothiaam” (Here! The Cataract). It was reverently passed round. Then squeezing out the ball of cotton, he put it on as pad and bound up the eye. I would be omitting some truth if I do not add that while going out one of the relatives slipped an envelope (Rs.20/-) in my pocket. Assistant’s fees! Whenever the topic of deteriorating doctor-patient relations come up, I am reminded of my great teacher who inspired such blind faith in his patients.
Sino-Orbital Tumour- Exenteration

Dr. Jayasree Menon P MS, Dr. C.V Anthrayose MS, Dr. (Prof) Alex Joseph MS

History

60 yr old male presented with a history of chronic nasal obstruction caused by a nasal mass diagnosed 3 months previously and associated with headache and difficulty in opening the left eye for the past one month (Fig 1)

O/E The left nasal cavity is filled with a mass lesion, with plenty of nasal discharge and bleeding on probing was also noticed. There was a mass in nasopharynx in (L) choana and the infraorbital margin (L) appeared soft.

Investigations

X ray PNS: (L) Maxillary sinus hazy with destruction of bone

CT Scan: Malignant mass involving sinonasal cavity (L) side with extension to adjacent structures. Evidence of acute right maxillary sinusitis was also noticed. (Fig 2 a & b)

Biopsy

from (L) nasal cavity was carried out and a histopathological evaluation was performed. The histopathological study revealed a Sinonasal non-keratinising squamous cell carcinoma.

Fig 1. Full face view showing narrow palpebral aperture left eye and hypotropic position of the globe on left side

Fig. 2. a & b : CT scan demonstrating mass filling sinonasal cavity (L) side with extension into adjacent structure and evidence of (L) maxillary sinusitis

Fig. 3. a & b : Showing the globe in 15-20° Hypertropia and abduction with gross restriction of adduction and depression
The patient was advised maxillectomy with orbital exenteration after a thorough ophthalmic evaluation. Ophthalmic evaluation revealed a visual acuity of 6/6 in right eye and 6/9 in left eye. The left eye showed 15-20° hypertropia with abduction (Fig 3 a and b). Adduction and depression were absent. Both eyes also had incipient cataracts. Fundus evaluation in the right eye was normal while in the left eye the inferior half of the fundus could not be evaluated as depression was greatly restricted.

A left (L) total maxillectomy with orbital exenteration done under general anaesthesia and the specimen was subjected to histopathological examination (Fig 4 a and b).

The histopathological study showed a sinonasal non keratinizing squamous cell carcinoma.

**Fig 4. a and b: Post operative appearance of the exenterated orbit**

**Discussion**

**Maxillary Sinus Carcinoma**

The commonest paranasal sinus tumour involving orbit is maxillary carcinoma. These produce diplopia, proptosis and visual loss in 23%. Of these, squamous cell carcinoma constitutes 90% and most of them are poorly differentiated with little evidence of keratinization.

International Union Against Cancer (UICC) Staging system for Malignant Neoplasms of the Paranasal Sinuses

**Maxillary sinus**

- **T1** - Tumour limited to the antral mucosa with no erosion or destruction of bone.
- **T2** - Tumour causes bone erosion or destruction, except for the posterior antral wall, including extension into hard palate and / or middle meatus of nose.
- **T3** - Tumour invades any of the following: bone of post wall of maxillary sinus, subcutaneous tissues, skin of cheek, floor or medial wall of orbit, inferotemporal fossa, pterygoid plates, ethmoid sinus.
- **T4** - Tumour invades orbital contents beyond the floor or medial wall including any of the following: orbital apex, cribiform plate, base of skull, nasopharynx, sphenoid, frontal sinuses.

**Prognosis**

The prognosis of maxillary sinus tumour depends upon the staging of the tumour.

Advanced stage is associated with worse prognosis. T1 & T2- 5 yr survival rate is 70% whereas T3 & T4- 5 yr survival rate is only 30%.

**Treatment**

Previously radical excision with orbital exenteration was done for all cases of orbital involvement. Currently, preoperative radiotherapy and / or chemotherapy preferred if there is only orbital wall involvement with preservation of periosteum. But with extension of the tumour mass into the orbital contents, as in this case, orbital exentration is the only option.

**Orbital exenteration**

**Indications**

1. Malignant tumours of eye and ocular adnexa
2. Malignant tumours extending to orbit from cranium or PNS
3. Severe trauma
4. Congenital deformities of eye and orbit
5. Mucormycosis and fungal diseases of orbit
6. Occasional cases of severe orbital contracture

Most common ophthalmic indication for orbital exenteration is basal cell carcinoma of lid extending to orbit in Speath's series of 38 cases. Basic principle of exenteration is to remove all diseased tissue while preserving as much normal tissue as possible. It can range from extended enucleation or partial exenteration to radical procedure.

The simplified method of exenteration is the proposed by Coston & Small if eyelid can be saved.
Technique

The periosteum is incised around the entire superior, medial and lateral circumference of orbit; the inferior segment is excised with the en bloc resection of maxilla. Periosteal elevators, then elevate the periorbita to the apex of orbit. With the globe retracted down and medially, a curved clamp is then passed through the inferior orbital fissure to grasp the Gigli saw. Occasionally fracture of some thin bone will be required to introduce the clamp. If it is not possible to pass the clamp, the lateral orbital rim is sectioned using a sagittal plane saw. Directing the Gigli saw upward and forward transects the lateral orbital rim. The optic nerve is severed midway between the globe and optic foramen or as far as posterior as indicated. Avoid excess traction on the optic nerve because it may produce damage to optic chiasma and subsequent visual field defects in the opposite eye. The ophthalmic artery which is medial to the optic nerve is ligated. Within the fat pads are small vessels that should likewise be ligated.

References

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Blepharophimosis with Bilateral Duane’s Syndrome - An Unusual Combination

Dr. Vijaya Kumari MS

Introduction
Blepharophimosis is a rare congenital disorder, which occurs sporadically or inherited as an autosomal dominant trait. Duane’s syndrome is characterized by limitation of abduction, narrowing of palpebral fissure along with retraction of eyeball and face-turn. A novel coexistence of sporadic blepharophimosis with bilateral Duane’s Type 1 in a male patient is described here.

Case report
A fourteen-year-old male came with complaints of drooping of upper lid since birth. Patient also complains of inability to close the eyes fully. There is no corneal exposure on attempted closure of the eyes. There is no significant head tilt or face turn. Visual acuity is 6/6 bilaterally. His abduction is partly restricted in both eyes. Retraction of the eyes on attempted adduction is present in both the eyes. There is no strabismus in primary position. Apart from this, there is hypoplasia of superior orbital rim, flat nasal bridge and distichiasis. There is moderate degree of ptosis in both the eyes with the upper lid covering half the pupil. Levator function is only 4 mm bilaterally. Lid crease is also noted to be absent. There is also ectropion of the lower lid, telecanthus and epicanthus inversus, which completes the picture of blepharophimosis syndrome. Anterior segment and fundoscopy is normal.

Shows ptosis in primary position (1), eye retraction and abduction restriction on side gaze (2, 3)
Discussion

Blepharophimosis ptosis epicanthus inversus syndrome (BPES) is a distinctive congenital eyelid malformation, which can occur sporadically or inherited as an autosomal dominant fashion. Both types of BPES have been mapped to chromosome 3q23 and mostly due to mutation of a fork head transcription factor FOX-L2 gene, which locates at this region.¹

Primary amenorrhea is described in most of the affected women.² Thus, BPES is characterized by facial dysmorphology combined in some cases with primary ovarian failure.³ High degree of menstrual irregularities and infertility are common in affected women. Early recognition of this condition may allow appropriate counseling and treatment including egg donation.

During the period of four to eight weeks of embryonic development the cranial nerves, their nuclei and corresponding innervation to extra ocular muscles undergo development and differentiation. This coincides with period of time that FOX-L2 is strongly expressed in developing eyelid and surrounding tissues.⁴

Mental subnormality can occur at times in sporadic cases. Seventy percentage of BPES patients have refractive error. In a study by Kyung. S et al ⁵ in Samsung medical centre, Seoul, Korea, out of 20 BPES patients, 45% had amblyopia, 25% unilateral and 20% bilateral. 67% of those with Amblyopia has coexisting strabismus ⁶.

In our patient, the only complaint was ptosis. There was no squint in primary position, refractive error or head tilt. Since the levator action is poor, the patient is advised sling surgery at a later date. Understanding this rare and complex syndrome will aid us in better management and counseling of such cases when need arises.

References

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3. Loffler.K.A, Zarkower.D. Etiology of ovarian failure in BPES, FOX-L2 is a concerned early acting gene in vertebrate ovarian development; Endocrinology. 2003 Jul 144(7); 3237-43
In the year 1900, Terson described the presence of a dome shaped subhyaloid haemorrhage associated with intracranial haemorrhage. Vander Linder and Chisholm (1974) described the occurrence of a sub ILM component in the premacular haemorrhage. Review of subsequent literature gives descriptions of perimacular folds (Keithen & Meiler: 1992) and also correlates a higher incidence of epiretinal membranes with dense vitreous haemorrhage and bleeding under the Internal Limiting Membrane.

This photo essay contains essentially intraoperative still photographs taken during vitrectomy procedure for a 21 year old engineering student with traumatic Terson Syndrome following a road traffic accident. The preoperative ocular evaluation revealed a visual acuity of hand movements on the right side with accurate projection and dense vitreous haemorrhage with subtotal posterior vitreous detachment on B-scan ultrasonography. The photographs depict the stages of induction of posterior vitreous detachment, presence of sub ILM bleed and demonstrates the technique of peeling the elevated ILM and draining the sub ILM blood. (Fig. 1 to Fig. 5)

Discussion

Terson Syndrome is characterized by pre, intra or subretinal haemorrhage associated with any form of intracranial haemorrhage. Other associated findings and long term sequela include epiretinal membrane (ERM) formation, pigmentary macular changes, cystic intraretinal changes (CIRC); epimacular membrane formation, macular hole and retinal detachment; ERM formation is the commonest sequela following Terson Syndrome and occurs in 40% of cases. A higher incidence of ERM formation has been described in eyes undergoing conservative management for the intraocular bleed associated with Terson Syndrome. The exact pathogenesis of this sequela is not known and it is postulated to be caused by the direct trauma to the ILM during the seepage of blood beneath it, with resultant glial proliferation.

Perimacular retinal folds are circular folds spanned by glistening membrane which on electron microscopy proved to be Internal Limiting membrane of the retina. These folds are hence associated with denser vitreous haemorrhage with sub ILM bleeding. Studies on the treatment options in Terson Syndrome favour an early surgical intervention in the form of pars plana vitrectomy with drainage of sub ILM bleed, to reduce the incidence of sequela like ERM formation.

References

Fig. 1. Dense organized vitreous haemorrhage

Fig. 2. PVD induction using Flute needle

Fig. 3. Demonstrates the presence of Sub ILM Bleed

Fig. 4 a & b  Stages of ILM Peeling

Fig. 5 a, b, c  Drainage of Sub ILM blood using Flute needle


Post Trabeculectomy - Good ‘Bleb’ with Poor IOP Control: Management Options

Compiled by Dr. V. Sahasranamam, Dr. Thomas George
Panelists: Prof. Dr. V. Velayutham, Dr. Noel Moniz, Prof. Dr. Andrew David Braganza

CASE HISTORY

A 42 year old gentleman presented to our glaucoma clinic with history of Primary Open Angle Glaucoma, diagnosed 2 years back and presently on Timolol and Bimatoprost eye drops. The IOP was 30 mm Hg BE with applanation on his present medications. Fundus examination showed 0.8 glaucomatous cup in the right eye and 0.9 cupping in the left eye. Field studies revealed typical glaucomatous field loss in both eyes. Visual acuity was 6/9 (uncorrected) in both his eyes and improved to 6/6 with a correction of +0.50 D sph.

He had a strong positive family history of glaucoma with his father and elder brother having the disease. Father was blind due to the disease from his 60th year of age. Since his IOP was not getting controlled with the present topical medications, considering the family history and his age, trabeculectomy was planned. Trabeculectomy, (with intraoperative topical antimetabolites) was performed in the left eye. On the first and third post operative days, the IOP was 12 mm of Hg in the operated eye, with a good bleb. He was discharged with advice to continue antiglaucoma medications in the RE and antibiotic steroid combination and atropine in left eye. One week review showed IOP at 12 mm Hg left eye. He was asked to review after 2 weeks with plan to do surgery in the right eye. At 3rd week review IOP in the operated eye was 25 mmHg. Eye was quiet with a good bleb.

How will you approach the case at this stage?

Prologue

When we brought up this case for discussion, we left a few lacunae so that our teachers can question and initiate a discussion. We expected that our experts' panel would be asking us:
1. What was the anterior chamber depth like?
2. Was the trabeculectomy ostium open?
3. Can a “good bleb” be associated with raised IOP, in the usual course?
4. Was the patient a steroid responder?
As expected, majority of the experts covered all these lacunae and discussed the case at length. Here is what the panel had to say........
Prof. Dr. Velayutham
Here is my opinion on the clinical situation you have mentioned in your letter
1. It is not mentioned whether the anterior chamber is normal, deep or shallow. It is not clear in the case report whether the bleb is functioning or not. If the bleb is functioning then the filtration is not sufficient. You can try laser suturolysis.
2. A Gonioscopy must be performed, the internal ostium must be viewed, and a note as to whether there is any iris periphery incarceration partially occluding the ostium is made. In that case laser over the peripheral iris may be attempted.
3. If the bleb is cystic, needling of the bleb is to be attempted.
4. Patient also needs to be put on anti-glaucoma medication
5. Do an Ultrasound to find out if there is any choroidal haemorrhage and if the AC is shallow an UBM is to be done to rule out Malignant Glaucoma. After successfully managing the left eye, the right eye can be taken up for trabeculectomy with antimetabolites and releasable sutures.

Dr. Noel Moniz
I assume that this is a case of POAG and that narrow angles have been excluded by indentation gonioscopy. First of all, we must try to find out the cause for the failure. I would do a gonioscopy to check the internal ostium and see if any iris, vitreous or debris is plugging the ostium. Look for any excessive conjunctival scarring which could also be a cause for a failing bleb. I would have very much liked to see a photograph of the bleb. A UBM would tell us of the type of bleb and any chance of the bleb getting encysted at this stage is highly unlikely. A digital massage may be tried which is unlikely to work at this stage. Then the option would be to go in for bleb needling which may be repeatedly done to break the encystment phase. Failing this, an augmented bleb revision is the choice. Both these methods help us to preserve the conjunctiva for further procedures. If one does repeated needlings or bleb revision the IOP should be kept under control all the time to prevent further damage to the optic nerve. One possibility of the patient being a steroid responder should be kept in mind.
If this also fails then I would discuss with the patient the possibility of resurgery or continuing on topical medication to obtain the IOP optimal for him which should be in the low teens. If I decide on resurgery, I like to see the patient on first postoperative day and thereafter on day 4, day 7, day 14, day 21. I either like to do a digital massage or a releasable suture. The option of laser suturolysis is a modality I am not very fond of.

Dr. Andrew Braganza
This is a very unusual situation. Failure of a filter in the early postoperative period is unlikely to be associated with a “good bleb”. I think a few more details are required. Is the bleb diffuse and avascular or localized? What is the anterior chamber depth and configuration? Is the patient a documented or suspected steroid responder? What is the gonioscopic appearance of the internal ostium at this stage? All these are relevant and will influence further management.
If the bleb is localized and/or vascular, the rise in pressure indicates failure of the filter either due to restricted flow through the ostium or fibrosis around the bleb with incipient encapsulation. Gonioscopy would show whether there is pigment, iris, or even vitreous obstructing the internal opening. (Yes, I have seen trabeculectomies in phakic patients with vitreous in the internal opening; possibly the surgeon was over enthusiastic in grasping the iris during the PI and ruptured zonules as well) It would also confirm adequate excision of the block. Why wasn’t the pressure rise seen earlier if there was obstruction to flow at this level? Postoperative ciliary “shutdown” may have masked the failure due to relative hypotony. Some flow through the filter may have allowed the bleb to stay formed. Or, if iris or vitreous is blocking the ostium, this may be a recent event. Even a partial block would result in elevation of IOP without complete flattening of the bleb. Careful attention to the appearance of the bleb would give a clue to this. Gonioscopy is mandatory, preferably with a Sussmann or equivalent indentation gonioscope which can be used safely even on the first postoperative day. A YAG laser can be used to clear the ostium if blocked.
If the bleb is in the process of getting encapsulated, needling and 5 FU injection is indicated. If it is felt that obstruction at the level of the scleral flap is the problem, laser suturelysis or release of a releasable suture if present could be done first. If needling becomes necessary, the surgeon has the choice of puncturing or
lifting the scleral flap during the procedure.

The next possibility is malignant glaucoma. This can occur at any time and is more common in eyes with angle closure glaucomas rather than POAG. It has been well described and I have seen it occur even while patients are on atropine. It is also important to realize that hypotony with choroidal effusions can subsequently lead to aqueous misdirection caused by rotation of the ciliary body. The configuration of the anterior chamber offers a clue. If this diagnosis is suspected, my preference is to perform Chandler’s four step diagnostic and therapeutic procedure. In any case, whatever surgery is performed, additional injections of 5 FU are mandatory if the long term success of the operation is to be improved.

The most difficult situation to diagnose, prove and handle is a steroid response following trabeculectomy. Withdrawing the steroid is not really an option here, as the resultant rebound inflammation is likely to cause the filter to fail completely. Also, challenging the other eye with steroids to establish the diagnosis is dangerous in the presence of advanced glaucomatous damage. My preferences in a situation like this when I suspect a steroid response is to do everything possible to make the filter work better. This starts with direct and indirect massage and 5 FU injections and goes on to suturelysis and needling as indicated. In my experience, the fear that hypotony will result when steroids are eventually withdrawn is not borne out by fact. There is always the option of restarting topical medications. Timolol, alone or in combination with dorzolamide is what I would use. However, starting anti glaucoma medications means we are giving up on the trab which I feel is too early.

What about the other eye? Well, it is not a good idea to rush into surgery in the other eye when we have a situation that we have failed to control here. Settle and stabilize this eye, then go on to the other is my preference.

A final word to the wise. Doing a trabeculectomy is not terribly difficult. Understanding the changes in aqueous dynamics and monitoring the patient with appropriate interventions postoperatively is a major challenge. This is why I generally ask my trabeculectomy patients to stay admitted for a week after the surgery and monitor them closely for a few weeks thereafter.

**Epilogue**

Now we shall discuss the line of management adopted for this patient.

The anterior chamber depth was adequate and gonioscopy showed open angles with the trabeculectomy ostium open. The PI was patent and the root of iris included in the PI on gonioscopy. The eye was quiet with very little congestion.

In the absence of inflammation and obstruction to out flow via the trabeculectomy ostium, a raised IOP occurring about three weeks after surgery and topical steroids, we presumed this to be a steroid responder. We gave the patient 250 mg Acetazolamide and the IOP was 18 mm Hg at two hours. As the IOP was no longer at very dangerous levels we maintained the patient on Acetazolamide 125 mg thrice daily (we preferred this because, for short term, systemic acetazolamide is a good drug and on stopping, it washes out within 72 hours).

Also we switched to a lower potency steroid with negligible propensity for rise in IOP, viz., Fluorometholone and kept the patient on follow up every 48 hours. The IOP was 14 mmHg after 10 days and we withdrew Acetazolamide. By three weeks the IOP was still 14 mmHg. As it was now 6 weeks post op and the eye was not congested, we tapered off the topical Fluorometholone over 2 more weeks. By then IOP was 14 mmHg with an adequate bleb and well formed AC. And we have scheduled surgery for the other eye in a few week’s time. Being an advanced POAG inadequately controlled with maximum medical therapy, surgery is indicated in that eye also. This time we intend to use Fluorometholone in the postoperative period.

In this case as steroid response was picked up early, stopping Prednisolone acetate drops could reverse the raised IOP. This emphasizes the need to check IOP at timely intervals to look for a steroid response in any patient on whom steroids are started. If situation is allowed to go on for some time then the IOP may not normalize on withdrawing steroids alone.

**References**


Work Related Musculoskeletal Disorders in Ophthalmologists: A Silent Epidemic?

Dr. Meena Chakrabarti MS DNB, Dr. Valsa Stephen MS, Dr. Sonia Rani John DNB, Dr. Arup Chakrabarti MS

Occupational injury and illness have been reported in health care workers in high rates, almost twice the reports from other service industries. The ophthalmic health care provider is at risk of developing work-related musculoskeletal disorders from exposure to ergonomic hazards of the working environment, inexpedient medical and surgical equipment design, awkward static postures, repetitive tasks and stress ever present in the various postures adopted at work. A substantial volume of epidemiological research exists which provides a strong cause-effect relationship between high levels of exposure or simultaneous exposure to the various ergonomic workplace hazards and the development of musculoskeletal disorders (MSD's) in the workplace. However, not much data is available on the relationship between the magnitude of exposure and the severity of complications of MSD's on ophthalmologists and optometrists.

Two unpublished surveys of 3700 ophthalmologists, one in the North East, and the other, nationally in USA reported a 66% and 85% incidence of work-related MSD. No single ophthalmic subspeciality had more problems and, no body site was more affected than others. The fact that only one third of ophthalmologists who received the survey questionnaire responded to it, pointed towards a lack of awareness of the occurrence of MSDs. It also brought out the fact that Ophthalmologists will have to scale a mountain of self-denial before addressing their own risks of becoming injured at work. However, once the risks are acknowledged, it is usually a simple matter to work differently to protect oneself or cope with a lingering work-related disability.

Routine ophthalmic practice involves excessive musculoskeletal workload in the cervicobrachial region. Performance of tasks which have a high level of “visual”, “manipulative” and “reach” demands on head, neck, and arms, results in increased muscular tension in the cervicobrachial muscle complexes. The problems are related to awkward static posture with upper cervical extension, lower cervical flexion, unsupported extremities and a kyphotic hunched posture.

Thus the presentations of MSD in eye specialists are

**Upper Extremity Neuropathies**
- Carpal Tunnel Syndrome
- Ulnar Neuropathy
- Rotator Cuff Tendonitis and Shoulder Impingement

**Neck & Back:**
- Neck and back pain
- Radiculopathy
- Muscular and ligamentous injury
- Neural encroachment due to bulging herniated disc.
- Degenerative Spondylolytic changes

Table-1 gives the results of a meta analysis of 2 major surveys on prevalence and type of MSD in ophthalmologists.

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Aggravating factors reported in both these surveys included using the indirect ophthalmoscope, performing long operations and poor posture. The most common diagnosis included nonspecific muscle strain (25.8%), herniated disc (14.3%) and unknown (13.3%). 7.5% of the physicians required surgical treatment. Certain modification in the postures adopted at work has been suggested to reduce the strain on the cervicobrachial muscle complexes.

Ophthalmologists have been practicing since ages, and the common query is “Why are these problems cropping up now in epidemic proportions?”

The answer may be categorized as

1. The rise of the **Nintendo World** – that is an increase in the tasks done chiefly with the key board and VDU screens requiring finely controlled and repetitive motor skills.
2. Unergonomic equipment and design of workstation
3. Adoption of awkward posture at work.
4. An emphasis on productivity which demands more in a shorter time period.
5. Excessive job specialization making it necessary to repeat same jobs several times.

There is a greater strain on all ophthalmologists to see more number of patients at a faster rate. Imagine yourself sitting at the slit lamp for a prolonged period of time, seeing patients, one after the other, with your neck flexed or extended and your arms stretched beyond the safe ‘reach envelope’. Repetitive tasks can cause cumulative damage to your neck, back, shoulders and wrists. The risk for injury is further heightened when the risk factors are combined such as use of slit lamps, indirect ophthalmoscopic laser delivery systems and operating microscopes.

None of these injuries will occur over a short period of time. They are the result of cumulative insults accumulated over years of practice. Hence our goal should be to identify these activities and modify them while the practicing ophthalmologist is still early in his career.

Using electromyography, photography and computer modeling, “risky work patterns” in ophthalmic practice have been identified. These include

1. Slit lamp biomicroscopy
2. Slit lamp laser use.
3. Indirect Ophthalmoscopy (Surgery / OPD)
4. Indirect Ophthalmoscope laser delivery system

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TABLE-1 Meta Analysis of 2 Surveys on MSD’s in Ophthalmologists
5. Operating Microscope use.

The correct posture to be adopted at work is detailed below:

At the slit lamp:
- Position the patient and slit lamp in such a way that your back stays straight.
- Your back should be supported by the chair back.
- Relax your shoulder and do not flex or extend your neck.
- Your arm should be supported and use an elbow rest made up of soft material (Foam padded material or viscoelastic surface).
- Using a standard pincer grip increases the pressure in the carpal tunnel. Use a pencil grip whenever possible.
- Avoid pronating your hand or forearm during gonioscopy. Avoid flexion of wrist.
- Relax your muscles and stretch your arms between patients and procedures.

While performing Indirect Ophthalmoscopy:
- Do not bend over. Elevate the patients’ chair so that you are comfortable.
- Limit neck flexion and sideways bending.
- Use an indirect ophthalmoscope with light weight fiberoptics to decrease the compressive and shear load on the discs of your neck.
- Support your hand on the patients’ head while performing scleral depression. (Fig. 2)

In the Operating Theatre:
- Use an ergonomic chair with a proper seat and back support as well as adjustable arm rest.
- Do not elevate and keep your shoulders abducted.
- Tuck your elbows gently into your sides to help support your arms.
- Position your wrist rest so that when you’re holding heavy hand pieces your wrist is not flexed.
- Reduce the duration of awkward postures involved in vitreoretinal surgery by staging procedures. Be aware that problems can occur while performing fine precise movements while using the pincer grip, prolonged seated posture, holding a vitreous cutter or scissors for prolonged periods and having to exert force against a cryoprobes’s stiff cord.

Adjust the OT table to an optimal height so that your back and arms are supported and your back and neck aren’t bent. The ideal sitting posture includes an anteriorly tilted pelvis, maintenance of lumbar lordosis,
neutral thoracic kyphosis, lower cervical lordosis and upper cervical kyphosis. With correct posture muscle tension is reduced and compressive loads are more evenly distributed throughout the spine, away from weaker posterior structures (Fig. 3).

In The Office

You should adjust your work station so that at work you have adequate back support, horizontal thighs and vertical lowerlegs. Your feet should rest on the floor or on a foot rest. While working in the computer your wrists should be supported and in a neutral position (Fig. 4). These minimal modifications suggested in the work posture will certainly go a long way in reducing incidence of work related musculoskeletal disorders. This article demonstrates the common ergonomic risk factors seen in ordinary ophthalmic practice and the cumulative consequence of improper posture on the musculoskeletal system. Modification of these factors could definitely reduce the risk of musculoskeletal disorders. In addition, consultation with a physical therapist to learn quick and simple exercises that can be performed in between surgeries or patient examination in what is termed ‘working out at work’ will also prove very useful.

It has been stated that an epidemic of cervical spine disease is looming over the nations’ eye professionals. If we’re aware of the existence of such a disease entity and are willing to try out modifications in our work posture, rest assured that – we’re well protected!
References


Narrow Strip Conjunctival Auto Graft for Treatment of Pterygium


Recurrence of pterygium is the most common complication of pterygium surgery. Although recurrence rate are more favorable with a conjunctival auto graft than with bare sclera techniques no single approach has demonstrated absolute effectiveness against recurrence.

The aim of this study, which was conducted at Cole eye institute, Cleaveland, Ohio was to determine the efficacy of narrow strip conjunctival auto graft surgery in the treatment of pterygium. It was designed as a retrospective non-comparative interventional case series study. 21 consecutive interventions were studied in 20 eyes of 18 patients for primary (n =17 cases) or recurrent (n =4). Pterygia exhibited at least 3 mm of corneal extension with progression towards visual axis in 19 out of 21 cases. The rate of limbal/ corneal recurrence at 12 months after conjunctival auto graft was defined as primary outcome measure and was assessed by the primary surgeon.

All surgeries were performed under retrobulbar anesthesia. Pterygium excision was performed as usual and anterior margin of conjunctival wound was sutured directly to sclera anterior to the rectus muscle insertion with 10-0 vicryl to form the posterior margin of bare sclera zone. A narrow 2 mm wide free superior conjunctival epithelial auto graft was fashioned with Wesscott scissors. A 1 to 2 mm zone of undisturbed conjunctiva was preserved posterior to limbus to ensure that limbal stem cells were not violated. The graft then was transplanted to pterygium excision site and was sutured to limbus and sclera anteriorly and posteriorly with 10 –0 vicryl. Suturing of graft and conjunctiva in this manner allowed a 2 to 3 mm zone of bare sclera between the two.

At one year and all the points there after 18 out of 19 (94.7%) cases were free of recurrence. The lone recurrence occurred inferiorly in an eye that had undergone an adjacent narrow strip conjunctival transplantation 6 months for a recurrent temporal pterygium previously and that remained recurrence free after a second surgery.

According to authors, the narrow strip technique appears to offer many advantages over other conjunctival auto graft techniques. Harvesting a narrow conjunctival graft may be less traumatic to the superior bulbar conjunctival procurement site than harvesting a larger graft. This approach also preserves limbal stem cells from the donor site. The concept of an intervening water shed zone may provide additional protection against limbal /corneal recurrence. The direct scleral fixation of graft and conjunctival wound may promote active migration of epithelium over bare sclera which in turn provides a barrier to fibro vascular proliferation by augmenting epithelial scleral adhesion and eliminating the potential subepithelial/ Tenon’s space that might provide the avenue for pterygium recurrence. One could argue that this effect is no different from bare sclera excision technique. However it is possible that the epithelium emanating from graft is different and likely healthier than that originating from conjunctival wound at the cut edge of pterygium.
Diurnal Variation of Central Corneal Thickness and Goldmann Applanation Tonometry Estimates of Intraocular Pressure


Elevated intraocular pressure (IOP) is a major risk factor for the development and progression of glaucoma, a disease responsible for 12.3% of blindness worldwide. IOP undergoes a natural diurnal variation of approximately 2-6 mm Hg in normal eyes that may be higher in glaucomatous eyes. Due to this variability, and evidence that suggests the risk of glaucomatous damage is related to the maximum IOP or magnitude of fluctuation during the diurnal cycle, clinicians are advised to conduct multiple measurements over the course of a day to determine the IOP profile of at-risk patients.

Goldmann applanation tonometry is the “gold standard” instrument for the assessment of IOP, but the accuracy of this device is believed to be influenced by corneal properties such as central corneal thickness (CCT) and hydration. CCT has shown to increase overnight as a function of hydration by approximately 2.9% to 5.5%, and return to baseline within 1 to 2 hrs of awakening. It may be hypothesized that the diurnal variation of CCT and corneal hydration may influence the accuracy of measurement of diurnal variation of IOP made using the Goldmann tonometer, but previous studies have indicated that the diurnal variation of CCT is too small to have a significant effect.

The aim of the study was to determine whether there was a temporal correlation between the diurnal variation of CCT and IOP, as measured by Goldmann applanation tonometry in young healthy adults, with an emphasis on the time period after the eye opening. The presence of a relationship may indicate that the accuracy of tonometric estimates of IOP is influenced by the diurnal variation in CCT.

This study is from school of optometry and basic science, University of New South Wales Sydney. Twenty five eyes of 25 young healthy normal participants were examined in this prospective observational cross-sectional study. IOP, CCT and corneal curvature were measured using standard clinical techniques over a 24-hour period, and the temporal interrelationships between these parameters were examined.

The results showed that overnight change in IOP measured by Goldmann tonometry was 3.1 +/- 2.4 mm Hg (p<0.001), CCT was 20.1 +/- 10.9 micrometer (p=0.016), with no statistical change in central corneal curvature (0.05mm, p=0.477). Both IOP and CCT were highest on awakening at 7.00 then dropped rapidly to baseline levels by 9:00 and these two parameters were highly correlated (r=0.978, p<0.001). After 9:00, there was no correlation between these parameters (r=-0.453, p=0.260). The peak value of IOP was recorded at or near eye opening in the majority (17 of 25, 68%) of participants whereas the minimum value tended to occur mid-afternoon.

The results of this study indicates that IOP and CCT fluctuate in distinctive patterns over a 24-hour period; all were significantly higher on eye opening than immediately before sleep, decreased rapidly for up to 2 hours, then became relatively stable for the remainder of the day. According to authors this is the first study to show that CCT and IOP were highly correlated during the 2-hour period after eye opening.

The overnight increase in IOP is thought to be due to the supine position adopted during sleep, reduction of ambient illumination, the change from light to dark conditions, the stage of sleep, and various circulating chemicals such as cortisol. These factors are removed on awakening and thus may at first seem to explain the subsequent rapid decline in IOP between 7:00 and 9:00. However the participants in this study were required to awake and up right for 15 minutes before...
IOP measurement with the intentions of avoiding the IOP changes associated with waking process. The cornea increases in thickness overnight due to the relative hypoxia, decreased osmolarity and increased temperature that occur under the closed eyelid during sleep, and as anticipated, there was a significant diurnal fluctuation in CCT in this study. A limitation of this study may be the selection of young, healthy participants with no ocular pathology, because the results may not necessarily be applicable to the glaucomatous population, as there may be specific corneal changes in older participants and in glaucomatous participants. The results of this study may have a substantial influence on the clinical assessment of IOP. Although it is inappropriate to make absolute conclusions in the absence of being able to measure the true IOP, it seems likely that the diurnal variation of CCT interferes considerably with the accuracy of IOP measurements for the first 2 hours after waking. As a result, clinicians should interpret IOP results obtained during this period with caution, as there may be errors resulting from variations in CCT if patients are seen outside normal working hours, or in patients with unusual sleep patterns such as shift workers.

Atropine for the Treatment of Childhood Myopia

Wei-Han Chua, Vivian Balakrishnan, Yiong Huak Chan, Louis Tang, Donald Tang.
Ophthalmology 2006; 113:2285-2291

Myopia is the most common eye disorder in humans. Studies indicate that the incidence rates of myopia in Asia are rising. The widespread prevalence and the rising rates, the associated visual morbidity and consequent diminution of quality of life and social disability, and the substantial costs incurred for its correction make myopia a significant public health concern. Recent clinical trials of a variety of interventions, such as progressive addition lenses and rigid gas-permeable contact lenses, have yielded disappointing results or positive results of marginal clinical significance.

To date, only topical atropine, a non-selective muscarinic antagonist, has been demonstrated through relatively small randomized trials to have some clinical effect on the progression for myopia. However, these atropine studies suffered from various methodological shortcomings such as lack of regular and detailed follow-up examinations, absence of appropriate clinical controls, and absence of masking of participants and investigators.

Aim of the study, which was conducted at Singapore National Eye Center, Singapore, was to evaluate the efficacy and safety of topical atropine, a non-selective muscarinic antagonist, in slowing the progression of myopia and ocular axial elongation in Asian children. It was designed as parallel group placebo controlled randomized double mask study were 400 children aged 6 to 12 years with refractive error of spherical equivalent of −1.00 to −6.00 diopters (D) and astigmatism of −1.50 D or less participated. Participants were assigned with equal probability to receive either 1% atropine or vehicle eye drops once nightly for 2 years. Only one eye of each subject was chosen through randomization for treatment. All children regardless of their treatment allocations were prescribed photocomatic glasses for correction of their refractive error. To minimize the observational bias neither the study participants nor the investigators were aware of the interventions given. The atropine and the placebo were packed in identical packets and both pupils were dilated when the children were presented before investigators.

The main efficacy outcome measures were change in spherical equivalent refraction as measured by cycloplegic autorefraction and change in ocular axial length as measured by ultrasonography. The primary
safety outcome measure was the occurrence of adverse events.

Three hundred forty-six (86.5%) children completed the 2-year study. After 2 years, the mean progression of myopia and of axial elongation in the placebo–treated control eyes was \(-1.20 +/- 0.69\) D and \(0.38 +/- 0.38\) mm, respectively. In the atropine–treated eyes, myopia progression was only \(-0.28 +/- 0.92\) D, whereas the axial length remained essentially unchanged compared with baseline (-0.02 +/- 0.35 mm). The differences in myopia progression and axial elongation between the 2 groups were \(-0.92\) D (95% confidence interval, \(-1.10 \text{ to } -0.77\) D; \(p<0.001\)) and \(0.40\) mm (95% confidence interval, \(0.35 \text{ to } 0.45\) mm; \(p<0.001\)), respectively. No serious adverse events related to atropine were reported.

The results in the study show that once nightly dose of atropine 1% drops achieved a reduction in progression of low and moderate childhood myopia compared with the placebo treatment that is both statically and clinically significant. Over a period of 2 years treatment achieved approximately 77% reduction in mean progression of myopia compared to placebo treatment and this finding is strongly corroborated by the concomitant findings in ocular biometry. Authors claim that compared to other similar studies this study stands apart because of its design and presence of several controls.

Much like the cause of myopia the mechanism of action of atropine in retarding the progression of myopia and axial elongation is not understood clearly. Further research is required to elucidate the mechanism of action, to evaluate the safety and efficacy of bilateral atropine treatment beyond 2 years, and to identify the characteristics of children who will derive maximum benefit from this treatment.

Reviewed by Dr. Alex Baby DO DNB. Little Flower Hospital and Research Center, Angamaly.
Clinical Techniques in Ophthalmology

Edited by Simon N Madge, James P Kersey, Matthew J Hawker, Meon Lamont
Published by Churchill Livingstone, Elsevier – Edinburgh London

Ophthalmology is extraordinarily complex, both technically and clinically, and induces bewilderment in the uninitiated when presented with a slit lamp or a page of orthoptic notes. This book seeks to unravel those mysteries and cover the whole range of ophthalmic examination skills, procedures, investigations and instruments that will be faced by the trainee in those first years of mastering the subject.

This is a book written by trainees for trainees. The style is refreshing, the explanations clear and the advice practical. Tips and pitfalls reflect those gems that have been passed down from generation to generation of ophthalmologists. The authors have accumulated and condensed a wealth of information together with beautiful diagrams and illustrations ... and if you are no longer a trainee, it will do no harm to go back to the beginning again and read this book.

In these pages you will find almost every aspect of ophthalmology covered, from writing up notes to understanding refractive surgery techniques, and from mastering biometry to the basics of ocular therapeutics; subjects that will also be relevant for other healthcare professionals.

This book has 6 sections like Basic clinical optics, Ophthalmic equipment, Examination of patients, Investigations, Ophthalmic procedures, An introduction to ophthalmic drugs.

This book cuts through the bewilderment felt by the new trainee and presents the practical, clinical skills and techniques of ophthalmology in a clear and accessible manner. Highly suitable for trainees and residents in ophthalmology, it will also be useful for nurse, optometrists and all those working in the field of ocular health.

101 Pearls in Refractive, Cataract, and Corneal Surgery

Edited by Samir A. Melki, MD, PhD., Dimitri T. Azar, MD
Published by Slack Incorporated, NJ, USA, 2006.
Price: $82.95

The present volume provides specialists in the various areas an opportunity to digest reports from throughout the world and format the information according to their own considerable experiences. Each chapter focuses on a specific ‘how to do it’ in a very practical way. The authors are also able to sort out enormous controversies in the field and to superimpose their own good judgment. Considering the enormous worldwide interest in and application of the type of surgery covered, there is no question that this text will be much sought after in the future.
Looking for quick and user-friendly surgical tips for refractive, cataract, and corneal surgery? Need all of the most important and practical information to be at your fingertips at a moment’s notice? The updated Second Edition of *101 pearls in Refractive, Cataract, and Corneal Surgery* will rise to the challenge and be your only go-to surgical guide.


Drs. Meli and Azar follow the same format as with their previous edition in that the information is organized as a condensed summary of key pearls and pitfalls of challenging surgical procedures in refractive cataract and corneal surgery.

Focusing on practical tips rather than theoretical aspects of surgical procedures and including clear illustrations of techniques, this book is a user-friendly, straightforward guide to surgical success. Each pearl will enhance surgical outcomes, reduce surgical time, minimize complications, or simplify a complicated step. Both novice and advanced surgeons will benefit from this unique, no-nonsense book on practical tips.

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**Ocular Inflammatory Disease**

*Edited by Jack J Kanski, Carlos E Pavesio, Stephen J Tuft*

*Published by Elsevier Mosby*

*Printed in china 2007, Price: £ 64.99*

Effective management of inflammatory eye disease rests on the correct identification of the clinical picture. Distinguishing infectious from non-infectious disease is of paramount importance since therapy will be radically different. Familiarity with the clinical features of the many different manifestations of inflammation is essential.

This book provides a succinct and practical guide with clear descriptions of the important clinical presentations. The text has been richly illustrated with clinical details and examples. A description of the diagnostic features, the recommended investigations, differential diagnosis, and management of each condition is presented.


Page after page, Ocular Inflammatory Disease delivers the information you need

- Covers in-depth descriptions of anterior segment inflammatory disease and intraocular inflammation
- Features the chronological aspects of disease processes.
- Includes the latest medical and surgical management strategies such as complications of uveitis, chemical burns, biopsies, HIV-related disease, and corneal rejection.
- Presents clinical pearls throughout that emphasize important clinical facts.
- Contains over 800 full-colour illustrations that depict virtually every inflammatory condition.

From expert guidance to the very latest in the field, this must-have reference answers all of your clinical questions.

This is a comprehensive, practical guide that provides a clear overview of ocular inflammatory disease. It contains outstanding visual guidance, a lucid writing style and concise descriptions of clinical signs, investigations differential diagnosis, and management.

*(Dr. C. V. Andrews Kakkanatt, Jubilee Mission Medical College, Thrissur)*
Welcome to KSOS

All new members to this community are welcome to use the KSOS website that is feature rich and informational. The website address is www.ksos.in

How to register as in the website?

All members are requested to provide the webmaster of KSOS, their details such as
1. First Name 2. Last Name 3. email id

What the webmaster will do:

The webmaster will add these details into the member list. The system will generate the User Id and Password, which will then be emailed to you.

Once the members receive their user ids and passwords, they are requested to visit the website and log in using the Member Login area (top right of the website). Then use the "View My Account" link to see your account details. Please update your details in that section.

You are also allowed to change your User Id and Password, to your liking. Please use names that you can always remember, for your user id. If you change your password, do memorise the new password.

Online facilities for the Members

KSOS members have access to very good online resources. All members can then take advantage of the advanced facilities provided such as

1. KSOS journals: Members alone can access the KSOS journals online. You have to login to access the journals.

2. Message Board Members alone can feed messages into the message board for the rest of the KSOS members.

3. Discussion Forum for members All members are also requested to take part in the discussion forum (a new feature) in the website. For this, you are requested to register separately. The instructions for this have been given in the “News Update” section of the website.

4. Video Streaming has been added to the website
This feature has been added to the KSOS website, making the website even more vibrant. It features streaming video content of medical surgeries and procedures.

5. Useful links The KSOS Website provides links to useful and important websites across the world.

6. Member search KSOS members can access the contact details of any other member, using the website. All members are requested to visit the website and see the features in the website and get accustomed to the online system.
CME Programmes

STATE CONFERENCES

“POT POURRI”
8th July
Chakrabarti Eye Care Centre
Dr.Valsa Stephen
Ph: 0471-2555530

RESEARCH METHODOLOGY
28th July
Little flower Hospital , Angamaly
Hotel Renaissance , Cochin

OPHTHACON 2007
Cataract Surgery
22nd July 2007
Ahalia Foundation Eye Hospital
Palakkad
Dr.V.R.Jayaram
Ph: 9447671716

AUGUST OPHTHALMICA
5th August 2007
Cochin Ophthalmic Club, Cochin

TACOPSIA
23rd September 2007
Thrissur Academy of Ophthalmology
Thrissur

DRISHTI 2007
23rd-25th November 2007
Hotel Indraprastra
Palakkad
Organising Secretary: Dr.Anup Chirayath
Ph: 9447774439

NATIONAL CONFERENCES

Annual Conference of the Intraocular Implant and Refractive Society
14th & 15th July 2007
Hotel Taj Coromandal, Chennai

EROVISION -2007
55th Conference of Tamilnadu Ophthalmic Association
10th – 12th August, 2007
Maharaja Ararangam, Erode
Organising Secretary: Dr.Paneer Selvam
0424-2227019/20/21

NETHRA DARSHAN-2007
2nd Oasis & 31st AP Ophthalmological Conference
28th – 30th September, Tirupathi
Organising Secretary: Dr.V.Krishnamoorthy
Ph: 9912366393

7th ALL INDIA UVEITIS CONFERENCE
1st and 2nd December 2007
International Convention Centre
Sri Sankaradeva Nethralaya
Guwahati, Assam
Organising Secretary: Dr.Dipankar Das
Ph: 91-3612228879/80

GLAUCOMA COMMUNIQUE 2007
Annual Meeting of Glaucoma Society of India
7th,9th December 2007
BB Eye Foundation, Kolkata

INTERNATIONAL CONFERENCES

WORLD GLAUCOMA CONGRESS
July 18-21, 2007
Singapore

XXV Congress of the European Society of Cataract and Refractive Surgeons (ESCRS)
September 8th -12th, 2007
Stockholm International Fairs & Congress Centre.
AAO -2007
Annual Meeting of American Academy of Ophthalmology
New Orleans
Evaluation of a Visually Inattentive Baby
Dr. Meena Chakrabarti, MS, DO, DNB

- **VISUAL DEVELOPMENT IN A NEW BORN CHILD**
  - Snellen Equivalent in a Newborn : 20/1000
  - At 1 Month : 20/600
  - At 4 Months : 20/200
  - At 1 year : 20/50

- At 6 weeks “fixation & following reflexes” should be present.

- **Causes for poor vision in babies with normal appearing eyes.**
  - Lebers Amaurosis
  - Optic Nerve Hypoplasia
  - Ocular Albinism
  - Achromatopsia
  - CSNB (Central Stationary Night Blindness)
  - X-linked Juvenile Retinoschisis
  - High Ametropias
  - Cortical Blindness
  - Oculomotor Apraxia
  - Delayed Visual Maturation

In all babies with suspected blindness a complete ocular examination, assessment of ocular motility, electrophysiological workup (EEG, ERG,VER), CT/MRI should be performed. Close co-operation with paediatric neurologist, paediatrician and geneticist is indicated.

Postpone a definitive answer till clinical picture is clear and do not label a baby as blind unless there is enough evidence to support it.
**GENERAL INSTRUCTIONS TO AUTHORS**

The Kerala Journal of Ophthalmology (KJO) is a quarterly; peer reviewed one, devoted to dissemination of the latest in ophthalmology to the general ophthalmologists as well as to specialists in the various subspecialties of this discipline. It invites submission of original work dealing with clinical and laboratory materials.

Authors submitting materials to this journal are requested to adhere STRICTLY to the norms laid down below. The matter must be typed on one side of the paper. A margin of 1" must be left all around and the material must be double-spaced. A page should contain not more than 25 lines. Two copies of the text in paper and one copy in a CD must be submitted to the Editor and the corresponding author is advised to keep another copy with him. The corresponding author must give it in writing in his covering letter that the same matter will not be submitted elsewhere if accepted. He must also enclose the copyright transfer of his work to this journal. The papers sent will be subjected to peer review. The accepted manuscripts become the permanent property of this Journal. The author is informed that, if his work is returned to him for correction / clarification after peer review, he should effect the same and send the manuscript back to the Editor within one month. Each manuscript component mentioned here under must begin with a new page and the pages are to be numbered at the right tip corner starting from the Title page.

1. **TITLE:** The title of the work must be brief and precise. It should not exceed two lines and 40 characters (including comma, period) Author (s) full name (s) must be given along with his (their) degree and the affiliations. Corresponding author's name, correct address (including e-mail and Fax, if available) and phone number must be mentioned at the bottom left hand corner of the first page.

2. **ABSTRACT:** The abstract is to be given in the beginning itself. It should not exceed 200 words. It must contain the aim, methodology, results and conclusion. For case report, summary / conclusion alone is to be given.

   **KEY WORDS** (maximum five) in capitals are to be included at the end of Abstract.

3. **INTRODUCTION:** Describe the aim of the study, along with the hypotheses that were tested. Only necessary references are to be given

4. **METHOD:** Give in detail the materials used and the methods employed. Describe the type of study. Pharmacological names only must be mentioned for the drugs used and, if proprietary name is used, then the manufacturers name must be given in parentheses. Except for standard, well-accepted abbreviations (Including SI Units), all others must be introduced in parentheses when the full term is used for the first time in the article.

5. **RESULTS:** Give only the results obtained by the study under discussion. State the statistics in the correct scientific form (P value, mean etc). Results based on assumptions must not be given. Indicate in the text the place where the tables have to be inserted

6. **DISCUSSION:** The discussion should be to the point and relevant to the subject under discussion. This section can be combined with the previous one if the author desires. Avoid speculations. Use only standard abbreviations or the abbreviations already introduced.

7. **ACKNOWLEDGEMENT:** This is to be made only to those who were directly and scientifically involved with the preparation of the paper. Permitting authorities, technicians, photographers who assisted in the work need not be mentioned.
8. REFERENCES: The references should be given in numerical order in which they first appear in text and not in alphabetical order (Citation Order System). It should be numbered consecutively in the text. The references will not be checked by the Editor or by the Peer reviewer and hence the author is solely responsible for its completeness and the accuracy. Period should not be employed anywhere in the references. Personal communication, unpublished data and poster references, if mentioned, should be in the text itself and the source mentioned in parentheses. References should be in the following form:-

Journal reference: Author(s) full title, Journal name (as abbreviated in Index Medicus), volume number, pages and year. If there are more than three authors, then mention the first three authors and then 'et al'.

Book reference: Authors(s) (& Editor, if any), title of book (and chapter), publisher, place of publication, page number (s) of the cited portion and year.

9. THE LEGEND: The legend for the illustrations (and tables, if necessary) must be given in a separate sheet of paper and should be typed double-spaced.

Illustrations: The photos and figures should be prepared in glossy prints with good contrast and of the size 6” x 4”. Only salient details should be included. On the back of the illustration, the figure number in text, title of the paper, the first author’s name and the top side (marked with an arrow) must be specified. Except for arrows, no text is to be on the photos. It is the duty of the author(s) to get the patient’s written permission when the subject is identifiable in the photo. Submit two sets of illustrations. Illustrations from other Journals and books are usually not accepted. If used, it rests with the author(s) to get the copyright permission from the original author / publisher and this permission letter must be sent to the Editor at the time of submitting the manuscript. For Histological figures the stain and magnification used should be noted e.g.: - H & E Stain x 70.

10. TABLE: It should be in double space. Each table must have an Arabic numeral (except for single table) and a title both in a single line. Each column in the table must have a short heading. If a table is large, then it must be continued in a second page, which also must have the table number and the title. Avoid vertical lines in the tables. Two sets must be submitted.

The manuscripts are to be sent to The Editor by Courier Mail or by Registered post. The corresponding author will receive communication from the Editor within two weeks of receiving the manuscript.

11. All manuscripts are subjected to editorial board review.

12. Other Categories of Manuscript
   a) Original Articles should generally not exceed 3,000 words or 12 double – spaced pages.
   b) Review Articles: can be on topics of relevance to clinical practice, research methodology, community ophthalmology or investigative work, of relevance to visual science. These articles should include up to date review of existing literature, and summarize the current status / preferred practice for that particular topic.

Brief reports are short communication of new instruments, new laboratory techniques or surgical techniques as well as interesting case reports with unique findings. These should not exceed 1000 words with a maximum of 2 illustrations. They should follow the format - introduction, case, and discussion. No more than 8 references should be cited. Each brief report must begin with a 75-100 word summary that highlights the significance of the articles.
APPAA AUTOPERIMETER

Model: AP 901H
Compact Design
External Fixation Target
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