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173 Instruction to Authors
It is indeed a great honour to bring out the second issue of our journal. I had tried to pull it out in time, but couldn’t do it entirely to my satisfaction. I apologize sincerely for the delay. I have tried to maintain the same structure of contents in this issue as in the previous issues, as consistency is an important virtue in our long term goal of indexing. I am indeed fortunate to have avid authors who are the bone muscle and skin of this journal and I take this opportunity to bow in front of them.

As usual we have started up with the cover story, this time on trabecular meshwork, which I am sure will throw some light on the etiopathogenesis of glaucoma. This meshwork is the key player in open angle glaucomas. I request all of you to give your suggestions regarding the next cover pictures.

The first major review in this issue is an evidence based review on the management of retinal detachment. The author has very clearly put forth the evidence for and against the various forms of treatments for retinal detachment with an evidence based review of literature. More than the article, the methodology involved in the article should be the way to go about in the management of any disease.

Our next review is on pediatric cataract surgery, an area where a lot of changes are taking place, thanks to advent of newer technology in the surgical procedures, understanding of the nuances of inflammation and advances in the premium IOls. I am sure, this article will give us great insight into the way we treat the pediatric cataracts. Videos are uploaded for you to see the techniques described.

Strabismus is one area where there is a lot of subjectivity in surgery. The author of this review takes us through various points of importance, before, during and after the surgical correction of strabismus. Thus we have incorporated three surgical areas in the major reviews this time, articles which would help improve the practicing ophthalmologist’s wisdom.

Our problem oriented medical diagnosis, this issue focuses on optic disc morphology as a cue to diagnosis. We all know that glaucomatous optic nerve is a great mimicker and can be missed by routine examination. Here we are looking at the reverse where discs look glaucomatous, but truly are not caused by glaucoma. The article goes on to differentiate almost all the differential diagnosis of glaucomatous disc morphology.

We pursue the way to write a research protocol in our Biostatistics made easy. This is the second part of the article. The author has nicely in a very comprehensible manner arranged the things to do before one starts any research protocol. It is a complete article and will help clinicians as well as postgraduates grasp the basics of research methodology.

Managing week zonules during cataract surgery is the theme of surgical corner this time. The article focuses on preoperative work up, intraoperative techniques and postoperative complications in pseudoexfoliation and week zonules during cataract surgery. An excellent video is uploaded for clarity of understanding.

Our common test simplified in this issue is gonioscopy, an important skill that every one needs to master. With a number of pictures and photos, the author mesmerizes us with an article complete with almost all angle pathologies.

Added to these are our brief reports and original articles, all of them are eye openers in their own sense. The journal review, book review and PG corner finishes the list. Let me once again persuade all of you to spare some time to think what you can contribute to our journal. Please feel free to criticize me as that is the only way forward for improvement.

Jai KSOS

Dr Gopal S Pillai
Editor KJO
Trabecular Meshwork – Anatomy And Cellular Organization

INTRODUCTION
Aqueous humour leaves the eye at the anterior chamber angle mostly through the system consisting of trabecular meshwork, Schlemm’s canal, intrascleral channels and episcleral and conjunctival veins. This pathway is referred to as conventional or trabecular outflow. Trabecular outflow in humans accounts for approximately 70% to 95% of the aqueous humour egress from the eye, with the lower values corresponding to younger eyes and the higher values corresponding to older eyes. The other 5% to 30% of the aqueous humour leaves primarily by uveoscleral outflow pathway, with a decline in contribution with age.

DRAINAGE PATHWAYS
Conventional outflow system
Trabecular meshwork ---- Schlemm’s canal ---- collectorchannels of schlemm’s canal ---- intrascleral and episcleral venous plexus (fig 1).
Unconventional outflow system
Aqueous humour ---- through the ciliary body ---- along the interstitial spaces of the ciliary muscle ---- choroid or suprachoroidal space ---- together with the vascular channels of sclera into the connective tissues of the orbit.

TRABECULAR MESHWORK
Trabecular meshwork consists of two parts- non filtering part and a filtering part.

Non filtering part
Anterior most portion of the trabecular meshwork adjacent to that portion of the limbus just posterior to schwalbe’s line. It has got no contact with schlemm’s canal, hence called the non filtering part. Consists of 3-5 trabecular beams covered by small trabecular cells forming elongated bands or rows. They are in contact with keratocytes of posterior lamellae of cornea. Also called as opercular cells as they form a cover over the anterior part of the filtering meshwork.

Filtering part
Filtering part of the trabecular meshwork covers the inner wall of schlemm’s canal. Consists of three morphologically and most probably functionally different portions.
1. Juxtacanalicular or cribriform layer
2. Corneoscleral meshwork
3. Uveal meshwork

Juxtacanalicular layer
Also called as endothelial meshwork or cribriform layer or pore tissue. It is the outermost part of trabecular meshwork adjacent to inner wall endothelium of schlemm’s canal. The inner trabecular endothelium is continuous with the endothelium of corneoscleral meshwork. The central connective tissue layer has variable thickness and is unfenestrated with several layers of parallel spindle shaped cells loosely arranged in
a connective tissue ground substance which appears as “empty spaces” in electron micrographs. The outer most portion of trabecular meshwork, the last tissue that aqueous humour must traverse, is the inner wall endothelium of schlemm’s canal. The surface is bumpy due to protruding nuclei, cyst like vacuoles and finger like projections bulging into the schlemm’s canal (fig 2). Actin filaments are also described in the inner wall endothelium of schlemm’s canal. The intercellular spaces are 150-200A wide and the adjacent cells are connected by a variety of intercellular junctions. Openings in the inner wall endothelium of schlemm’s canal consist of minute pores and large or giant vacuoles. They are now believed to be physiologic structures involved in the transcellular transport of aqueous humour. It is found to be a passive transport as the number and size of vacuoles have been shown to increase with progressive elevation of IOP. An alternative theory to the transcellular transport is the paracellular routes between the inner wall endothelial cells.

**Uveal meshwork**

This inner most portion is adjacent to the aqueous humour in the anterior chamber and is arranged in bands or ropelike trabeculae (fig 3) that extend from the iris root and ciliary body to the peripheral cornea. The arrangement of the trabecular bands create irregular openings that vary in size.

**CELL SYSTEM OF TRABECULAR MESHWORK**

Trabecular meshwork contains three basic types of cells.  
1. Trabecular cells — derived from neural crest cells. Covers the beams of corneoscleral or uveal meshwork. They are predominantly involved in the self cleaning process of trabecular meshwork by phagocytosis and tissue repair.  
2. Cribriform cells — along with the extracellular matrix of cribriform layer beneath the endothelial lining of schlemm’s canal. These cells are derived from perivascular cells. These cells are found to be highly metabolically active.  
3. Endothelial cells of schlemm’s canal —derived from mesodermal tissue. The inner wall endothelium is capable of developing pores, vacuoles and transcellular microchannels through which aqueous humour, particles and even erythrocytes can pass.  

Trabecular meshwork cells produce glycosaminoglycans, extracellular glycoproteins and fibrillar material.

**CONNECTION BETWEEN TRABECULAR MESHWORK AND CILIARY MUSCLE SYSTEM.**

The fibroblasts of the anterior ciliary muscle sheaths are continuous with the cells lining trabecular lamellae. Posteriorly the intertrabecular spaces are continuous with the interstitial spaces of the longitudinal portion of the ciliary muscle system. Longitudinal muscle of the ciliary muscle system taper off into 3 different types of tendon.

Type A- attaches to the scleral spur
Type B – anchors within the posterior corneal stromal layer.  
Type C – attaches to the corneoscleral meshwork and cribriform layer.

**PHYSIOLOGICAL VARIATIONS IN TRABECULAR MESHWORK.**
1. Thickening of trabecular beams.
2. Continuous loss of cells in the corneoscleral and uveal meshwork. The decrease in cell number is slightly greater in the central portion of the meshwork than in the anterior and posterior portion.
3. Cell loss within the cribriform layer.
4. Deposition of extracellular material which appears as irregular clusters, bands or plaques within the cribriform layer. They are derived from the sheath of elastic like fibrenet beneath the endothelial lining of Schlemm’s canal. Hence called sheath derived plaque (SD plaque).

**PATHOLOGICAL VARIATIONS IN TRABECULAR MESHWORK.**

**POAG.**
1. SD plaque deposition in the cribriform layer.
2. Decrease in the vacuolization of inner wall endothelium of Schlemm’s canal.
3. Increased resistance of the inner wall of Schlemm’s canal with collapse of parts of the canal and plugging of collector channels as a secondary effect.
4. SD plaque deposition around the collector channels.
5. “Matrix vesicles” found in the cribriform layer indicating cellular degeneration.

**NTG**
1. Trabecular beams appear somewhat thickened.
2. Large amount of SD plaque material deposited beneath the endothelial layer of Schlemm’s canal and within the cribriform layer.
3. Trabecular cells are stimulated by some unknown factors to produce extracellular material which appear to accumulate within the cribriform layer.

**Pseudoexfoliation glaucoma**
1. Exfoliative material found deposited beneath the inner wall of endothelium of Schlemm’s canal as well as in the cribriform layer.

**Pigmentary glaucoma**
1. Plugging of intertrabecular spaces by pigment granules together with adhesion of trabecular beams would cause decreased outflow facility.

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Management Of Rhegmatogenous Retinal Detachments- Evidence Based Review

Introduction
Rhegmatogenous retinal detachment (RRD) although rare, it is a serious ophthalmic condition that can lead to significant loss of vision or blindness without timely and appropriate management. It has been nearly a century (1918) since Jules Gonin demonstrated the importance of localizing and sealing retinal breaks, a procedure termed ignipuncture.1 Scleral buckling techniques, introduced by Custodis2 and refined by Schepens and later Robert Machemer’s pars plana vitrectomy, revolutionized repair of RRDs. Pneumatic retinopexy introduced in the mid 1980s allowed treatment of retinal detachments as an outpatient procedure in selected retinal detachments 3 Above techniques either alone or in combination has resulted in surgical success rates close to 90%.

For most surgeons, choice of surgical procedure for primary retinal detachment will depend on the individual clinical situation combined with each surgeon’s experience; bias and comfort level with a particular procedure. Recent advances in surgical instrumentation, wide angle visualization, and the use of various intra-operative tamponade agents have made vitrectomy procedures safer and preferred treatment by the surgeons. There is less exposure to scleral buckling for vitreo-retinal surgeons in training and younger surgeons may ultimately prefer vitrectomy as a first choice for repair of retinal detachments. Scleral buckling however still has relevance to this day, but without a randomized clinical trial comparing the 3 modalities, and in various clinical scenarios, definitive answer as to which procedure is superior will be impossible.

Basic Rationale of repair of RRD
The regardless of the procedure chosen, the surgery aims to identify and close all the retinal breaks with minimum iatrogenic damage. Break closure in retinal detachment would involve two steps. First, is to bring the edges of the retinal break into contact with the underlying RPE which is achieved either by bringing the eye wall closer to the detached retina (a scleral buckle) or by pushing the detached retina toward the RPE (intraocular tamponade with a gas/ PFCL(expand) bubble). The second step would be to create a strong chorio-retinal adhesion around the breaks; this may be accomplished with cryotherapy, laser photoacoagulation or diathermy.

This review will try to find answers to some of the common situations in retinal detachment treatment

Asymptomatic retinal detachment
Management of asymptomatic retinal detachments usually identified on routine clinical examination range from conservative observation to prophylactic surgery. Vrabec et al, 4 Byer et al 5 and Cohen et al 6 suggest a conservative management. These authors recommend that asymptomatic RRDs in selected cases may be safely observed for many years with routine examinations and appropriate patient education on symptoms of retinal detachment. Vrabec et al4 demonstrated, demarcation laser photocoagulation of shallow, macula-sparing, RRD without associated PVR to be a reasonable alternative to surgical repair. However, in a case series by Greven et al 7, asymptomatic RRD patients undergoing scleral buckle had good anatomic and functional results. They advocate that surgical management should be considered for asymptomatic RRD

Scleral Buckling:
Scleral buckles usually are made of solid silicone and silicone sponges. They can either be used as explants or as implants. Explants are the most commonly done procedure where the buckle is sutured to the sclera while in the implant technique they are placed in the bed of the dissected sclera. Scleral explant procedure was initially described by Custodis 2 which Lincoff later modified 8 while the implant method was popularised by Schepens 9. Both the above techniques have the following steps in common
- conjunctival peritomy and tenotomy
- Isolation of the recti muscles.
- Localisation of breaks with IDO
- Retinopexy with Cryotherapy /diathermy
- Suture placement / scleral bed dissection and placement of buckle is then performed.
- Subretinal fluid drainage is done based on surgeon preference and case based need.
- Break buckle relation and adjustment of buckle height.
- Careful monitoring of central retinal artery perfusion is done (AC Paracentesis done if required)
- Finalisation of buckle and encirclage
- Closure of the conjunctiva

Contentious issues in Scleral buckling.

Address for correspondence: rajusampangl@hotmail.com, Netraspandana Eye hospital, Bangalore
a) Cryotherapy vs laser photocoagulation
In a randomised clinical trial by Lira et al 39 in eighty six patients with uncomplicated retinal detachment, both techniques of retinopexy were shown to have satisfactory anatomical and functional success. Laserpexy offered faster visual acuity recuperation with fewer postoperative complications but required a second intervention and was costlier than cryotherapy. They opined that laserpexy to be a successful alternative to cryopexy in creating chorioretinal adhesion for scleral buckle surgery.

b) Need for drainage of subretinal fluid?
Drainage of subretinal fluid is one of the debatable issues in scleral buckling. As this step is almost a blind procedure it is not free from potential complications that include choroidal hemorrhage, retinal incarceration and intraocular infection. Drainage in scleral buckling surgery is usually done in bulloxous detachments to visualise the breaks, to make space to allow a large scleral indent without significant increase in intraocular pressure. Hilton et al.10 compared drainage vs. non drainage in a randomised controlled trial of 120 consecutive patients undergoing scleral buckling procedures. He found no significant difference in the primary success rate (87% in the drainage group v 82% in the non-drainage group), final flattening rate (97% in both groups) or visual acuity outcome between the two groups. Decision on whether to drain subretinal fluid was assigned at random preoperatively and all surgeries were done by a single surgeon. It can be concluded that drainage of subretinal fluid is not an absolute necessity and is indicated only in specific situations.

c) Which is the best method of drainage?
Various methods of drainage have been described:
1) Scleral cut-down and choroidal puncture with diathermy,
2) Scleral cut-down and the choroidal puncture by argon laser via an indirect ophthalmoscope or endoprobe.
3) Needle drain where the sclera and choroid are punctured in one stab with a 3 mm suture needle
4) Needle drainage with 26/27 g needle

Three trials have prospectively compared needle and laser drainage. Ibanez et al. 11 in a randomised study of 175 patients comparing laser drainage choroidotomy using an endoprobe, or needle drainage found no significant difference in the complication rate between the two groups (13% v 16%). However, Das and Jalali 12 reported an increased complication rate in the needle drainage group (4/25) than in the laser group however no statistical analysis was provided. Randomised prospective, controlled trial comparing suture needle drainage with argon laser drainage by Aylward et al 13 Argon laser drainage was associated with a lower rate of clinically significant subretinal haemorrhage (4.3% v 28.3%, respectively) and a higher rate (98%) of adequate drainage, compared with suture needle drainage (85%), even though larger sclerostomy created by the argon laser drain was larger it was not associated with any increase in the rate of retinal incarceration. Azad et al 14 compared modified needle drainage with conventional drainage of subretinal fluid (SRF) as described by Schepens in surgery for primary rhegmatogenous retinal detachment. They found the Conventional Drainage group had, more serious SRF drainage complications and opined modified needle drainage is a safe and effective procedure for SRF drainage.

Sub-retinal haemorrhage is the dreaded complication and is problematic if it tracks back under the macula. Most retina specialists practice favours the expediency of a needle drain when the macula is attached and the submacular space is closed, while the safer laser drain is preferred when the macula is off. In case of total retinal detachments undergoing buckling, drainage on the nasal side would reduce chances of sub-retinal bleed tracking under the macula in the event of such a complication.

Pneumatic retinopexy
Pneumatic retinopexy has the major advantage of being an outpatient procedure. The technique was recommended in the management of RD caused by a single break, no larger than one clock hour and located within the superior eight hours of the ocular fundus, or by a group of small retinal breaks within one clock hour, in the absence of grade C or D PVR and uncontrolled glaucoma. Some selected cases with multiple retinal breaks located more than 30° apart can also treated with this technique.

Perfluoropropane (C3F8) and sulfur hexafluoride (SF6) are the most commonly used gases for pneumatic retinopexy. It involves cryopexy of retinal breaks if possible, followed by injection of an intravitreal gas bubble and postoperative positioning to allow the gas bubble to act as a tamponade for the retinal break. If cryopexy is not performed, laser photocoagulation is applied to the retinal breaks after they have been flattened with intraocular gas.

Complications: Complications of pneumatic retinopexy in the treatment include
- New retinal break formation in 4–26% of cases,
- development of new retinal detachment in 15–24% of cases,
- delayed subretinal fluid absorption in 4–21%,
- chronic macular detachment in 4.1%,
- PVR in 3–24%, macular pucker in 2–9%,
- subretinal gas in about 2%, and
- endophalmitis in 1% of cases.

Other rare complications include supra-choroidal gas, extension of the retinal detachment, macular hole formation, and entrapment of gas in the pre-vitreous space anterior chamber 15

In a comprehensive review by Clement et al 16 the updated
average surgical outcomes for the 4,138 eyes in the 21-year period revealed a single-operation successes (74.4%), final operation successes (96.1%), new retinal breaks (11.7%), and proliferative vitreoretinopathy (5.2%).

Pneumatic retinopexy is an effective procedure. Since the success rate is only marginally less than scleral buckling, it is a viable alternative and is of great use in patients unfit for surgery.

Controversial issues in pneumatic retinopexy

Is scleral buckling/vitrectomy better than pneumatic retinopexy?

The evidence

PR vs Scleral Buckling

Several controlled trials have evaluated the results. The Retinal Detachment Study Group conducted a multi-centre trial and compared pneumatic retinopexy with scleral buckling. They reported results at six months 17, and at 2 years 18; there was no significant difference in either first time (82% v 73%) or final (98% v 99%) reattachment rate for scleral buckling and pneumatic retinopexy, respectively. Pneumatic retinopexy group had better visual outcomes. Mulverhill et al.19 in small randomised study of 20 consecutive patients, who met inclusion criteria, to be treated either by scleral buckling or pneumatic retinopexy. Retinal flattening was achieved in one operation in 90% of the pneumatic retinopexies and 100% of the scleral buckles. Visual outcome was comparable between the two groups. However, in a meta-analysis of pneumatic retinopexy compared to primary scleral buckling procedures, scleral buckling was found to have a higher primary success rate than pneumatic retinopexy.20

PR vs. Vitrectomy

Pneumatic retinopexy, was found to have a comparable success rate to vitrectomy with cryotherapy and gas in a prospective randomised controlled trial of 120 cases in 1987.21 However it is not desirable to extrapolate this study findings to present day as the technique of vitrectomy was still evolving at that time.

Is pseudophakia a relative contraindication for pneumatic retinopexy?

Multiple clinical studies have demonstrated the lower success rate of pneumatic retinopexy in repairing pseudophakic detachments compared with phakic ones, with a range of success from 45% to 80%. 22 This has led Tornambe to recommend 360° peripheral laser at the time of pneumatic retinopexy in pseudophakic detachments, placing several laser rows posterior to the vitreous base. This lower success rate might be due to the higher difficulty in detecting pseudophakic breaks. 17,18 The retinal detachment study group found that most aphakic and pseudophakic eyes that initially failed to pneumatic retinopexy ultimately reattached with fairly good vision, hence recommended the use of pneumatic retinopexy independently to whether the eye was phakic or aphakic/pseudophakic. 17,18 However for many clinicians, pseudophakia still remains a relative contraindication to pneumatic retinopexy.

Primary pars plana vitrectomy (with or without scleral buckling)

Vitrectomy is usually indicated in patients with PVR greater than grade C1, giant retinal tears, posterior breaks, multiple breaks at multiple levels, in patients of iridofundal coloboma with RD.

Over the past decade, more and more surgeons have been advocating pars plana vitrectomy for the primary management of retinal detachments probably due to the vitreo-retinal training patterns and better technology in vitrectomy machinery and wide angle visualization.

Technique involves a standard 3-port pars plana vitrectomy with removal of the juxta basal vitreous. During this process, retinal breaks are identified, freed of vitreous traction and marked. Internal subretinal fluid drainage is then performed by one of 3 techniques: either through the causative anterior breaks using perfluorocarbon liquids or through one of the causative anterior breaks using a cannulated extrusion during fluid–air exchange or through a posteriorly created retinotomy during fluid–air exchange. The breaks are then treated with endolaser, either through perfluorocarbon liquids or under air. Some surgeons will only treat the identified retinal breaks, whereas some other will perform 360° peripheral laser, placing several rows posterior to the vitreous base. The air is finally exchanged for a long-acting gas/ silicone oil. The patient has to maintain position postoperatively. One of the major advantages of vitrectomy over scleral buckling is the greater ability of visualizing retinal breaks with the combined use of wide-angle viewing systems and scleral depression. Retinal break detection is also possible with the help of perfluorocarbon liquids and the Schlieren phenomenon. Many retinal breaks undetectable by ophthalmoscopic examination can thus be found during pars plana vitrectomy. The risks of hemorrhage and retinal incarceration associated with external subretinal fluid drainage are not encountered with endodrainage during vitrectomy.

Other significant advantages of vitrectomy over scleral buckling include the

- absence of refractive shift,
- clearance of vitreous hemorrhage/ vitreous floaters,
- less postoperative pain,
- lower risk of postoperative diplopia, and
- Lowered risk of infection.

Removal of the vitreous potentially present in the anterior segment and around the intraocular lens might also be beneficial for postoperative visual recovery.

Disadvantages of pars plana vitrectomy over scleral buckling
include a more
• the requirement for postoperative positioning, and
  the inability to engage in air travel when gas is used for
  intraocular tamponade
• costly surgery, especially if perfluorocarbon liquids are
  used,
• the risk of postoperative intraocular lens displacement,
• Complications:
  ✤ Intraoperative complications include iatrogenic breaks,
  optic capture, and retinal haemorrhage.
  ✤ Postoperative complications include cellophane
  maculopathy, cystoid macular edema, PVR. Some have also
  suggested that compartmentalization of fluid against the
  retinal surface might potentially increase the risk of epiretinal
  membrane formation.

Factors Determining Anatomical and Functional Success
Several factors appear to have an influence in the anatomical
and functional recovery after vitreo-retinal surgery in PRD.
1. Higher reattachment rates have been achieved in cases
in which the macula was attached pre-operatively. Similarly,
patients with less extensive RDs appear to have better
anatomical outcomes after vitreo-retinal surgery.
2. The presence of PVR at presentation appears to be one
of the most important factors determining the anatomical
outcome in PRD, with higher redetachment rates in those
cases in which PVR is present.
3. Poor presenting vision and longer duration of symptoms
before presentation,
the presence of preoperative choroidal detachment, vitreous
hemorrhage, large retinal breaks (≥ 1 clock hour), or breaks
located posterior to the equator and
4. The occurrence of intra-operative hemorrhage appear to
also be variables predictive of poor anatomical success. (23)
5. The length of history of the RD (time between the
occurrence of RD and the surgical repair) appears to have a
major influence in the functional results obtained after
surgery. The shorter the history of visual loss, the better the
visual recovery following surgery.
6. Girard 24, 25 found that the presence of anterior chamber
reaction and preoperative PVR of grade B or greater were
also associated with a poor visual outcome following surgery.

As per the results of the SPR (Sclera buckling vs Primary
Vitrectomy in Retinal Detachment) study, 26 a prospective
randomized clinical trial comparing scleral buckling
surgery (SB) and primary pars plana vitrectomy (PPV)
in rhegmatogenous retinal detachments of medium
complexity, primary vitrectomy combined with scleral
buckling surgery is recommended for the treatment of
rhegmatogenous retinal detachment in pseudophakic and
aphakic patients. It should be noted that these results do
not apply to localized detachments with a single break and
patients with proliferative vitreoretinopathy (PVR) grade B or
higher and giant tears were excluded from the study.

Contentious issues in Vitrectomy
1. Optimal intraocular tamponade agent in the surgical
management of PVR
The Silicone Study group analysed the efficacy and
complications of intraocular gas and silicone oil tamponade
in patients with severe PVR and reported its results in a series
of publications. 27–37 Silicone oil and C3F8 gas were similar
in visual acuity and anatomical outcomes. No difference was
found in keratopathy rates and persistent hypotony was
more common in C3F8 treated eyes (P < 0.05).

2. Need for scleral buckle / encircling band with vitrectomy
In a prospective, nonrandomized, comparative study in
pseudophakic patients Stangos et al 40 did not find any
additional benefit of encircling band.

3. 20g vs 23g vs 25g
In a comparison of 20- and 25-gauge vitrectomy for primary
repair of rhegmatogenous retinal detachment Kobayashi
et al 38 reported good anatomic and functional results with
25-gauge vitrectomy and the outcomes were comparable
with 20-gauge vitrectomy. Similarly in a comparative study
Lewis et al 41 found 20-, 23-, and 25-gauge instruments
to be equally effective for primary repair of pseudophakic
rhegmatogenous retinal detachment.

Conclusions:
Rhegmatogenous retinal detachment (RRD) can have multiple
anatomic presentations. An individualized approach to repair
of RRD is necessary for optimal results than following a single
stereotyped procedure. Scleral buckling can be a valuable
component for repair of retinal detachments as it supports
both the existing tears and the vitreous base. However, the
use of scleral buckling has decreased in recent years due
to the success of vitrectomy alone and the avoidance of
complications associated with scleral buckle that include
buckle erosion and strabismus.
Based on available evidence the following the principles in
management of rhegmatogenous RDs
  • Phakic RRD is best treated by scleral buckling alone,(figure
    1 a-d) unless other problems, such as proliferative
    vitreoretinopathy(figure 2) opaque media, (figure 3 a,b)
    blood in the vitreous, , or GRT(figure4 a,b,c) (figure posterior
    tear, necessitate vitrectomy as well.
  • Pseudophakic RRD is treated with vitrectomy combined
    with/without scleral buckling.
  • Pneumatic retinopexy is reserved for only very simple
RRDs.
Some Common representative situations and management
options are described in the table below.
Figure 1a: High Myopia with Inferior RD

Figure 1b: High Myopia with Inferior RD

Figure 1c, d: Inferior RD in a patient with High myopia managed successfully with scleral buckling

Figure 2: RD with posterior PVR

Figure 3 A: RD with significant vitreous haze

Figure 3 B: RD with significant vitreous haze managed with vitrectomy + silicone oil

Figure 4a: Giant retinal detachment

Figure 4b: GRT with rolled flap and bare choroid

Figure 4c: GRT managed with vitrectomy with silicone oil
<table>
<thead>
<tr>
<th>Clinical situation</th>
<th>Pneumatic retinopexy</th>
<th>Scleral buckling</th>
<th>Primary vitrectomy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Superior Quadrantic detachment with one peripheral break.</td>
<td>First choice excellent candidate for PR, in the absence of contraindications</td>
<td>First choice segmental scleral buckle placed radially, if the tear lies under a vertically acting muscle, a segmental circumferential buckle of solid silicone may be preferable</td>
<td>Second choice Can be considered as first choice if there is significant traction on the edges of the breaks especially in pseudophakics</td>
</tr>
<tr>
<td>Total detachment with one break.</td>
<td>Can be considered as First choice if the tear is in the upper eight clock-hours &amp; patient positioning possible</td>
<td>First choice with /without drainage. Especially if the break is the lower 4 clock hours Encircling band may be needed in the presence of other retinal lesions/traction/early PVR</td>
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</tr>
<tr>
<td>Detachment with multiple breaks at same distance from ora</td>
<td>Usually not an option unless all open breaks are within a 1-2 clock-hours</td>
<td>Can be considered as First choice usually with drainage Encircling band recommended</td>
<td>Can be considered as first choice especially in pseudophakics</td>
</tr>
<tr>
<td>Detachment with multiple breaks at different distances from ora.</td>
<td>Usually not an option</td>
<td>Broad buckle grooved silicone implant is employed</td>
<td>First choice</td>
</tr>
<tr>
<td>“Aphakic detachment” with multiple small ora breaks.</td>
<td>Usually not an option</td>
<td>Buckling with encircling band Can be considered as First choice</td>
<td>First choice Encircling band &amp; A 360-degree peripheral laser photocoagulation is often applied.</td>
</tr>
<tr>
<td>Macula off Detachment with peripheral break and pseudomacular hole</td>
<td>As per situation 2,3,4</td>
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</tr>
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<td>Macula off Detachment with peripheral break and macular hole.</td>
<td>Can be considered but second surgery may be required for macular hole</td>
<td>Challenging surgery, good results reported by some surgeons implant may be sutured to the sclera of the posterior pole.</td>
<td>First choice May require Heavy silicone oil for tamponade</td>
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<td>Detachment due to macular break generally seen in association with high myopia</td>
<td>Can be considered</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Detachment with retinal dialysis.</td>
<td>First choice: drainage optional</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Detachment with giant break (more than 3 clock hours)</td>
<td>Not an option</td>
<td>Usually not considered</td>
<td>First choice with or without a low, encircling scleral buckle PFCL needed to flatten retina</td>
</tr>
<tr>
<td>Detachment with no apparent break. Rule out secondary retinal detachment</td>
<td>Not considered</td>
<td>Contiguous cryotherapy is applied in one or two rows starting just posterior to the ora in all detached quadrants.</td>
<td>Can be considered as first choice as breaks can be identified with use of PFCL that causes the subretinal fluid to exit the subretinal space via the break(s). Staining the subretinal fluid with Trypan blue can also be tried for better visualisation</td>
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- **Scleral buckling**
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### Detachment with outer-layer break in retinoschisis

- Not considered
- outer-layer breaks should be carefully treated with cryotherapy and closed with scleral buckling
- vitrectomy for those cases in which outer-layer breaks are far posterior and their buckling would be difficult

### Detachment with PVR

- Usually not considered
- High encircling buckle for Grade C1 or C2 PVR
- grade C3 or greater, vitrectomy is recommended and is the mainstay of treatment

### References:
29. The Silicone Study Group. Vitrectomy with silicone oil

Dr Raju has completed his postgraduate training in ophthalmology and vitreo-retina residency at the prestigious All India Institute for Medical Sciences. He has published several scientific papers in international peer reviewed journals and presented papers, videos and posters in various international and national conferences. He has previously worked as associate editor of the DOS times ( Delhi Ophthalmic Society) and is currently the chief editor of Chakshu, Journal of the Karnataka ophthalmic society.
Other than cataract surgery, strabismus surgery is the next one to give instant gratification as well as dissatisfaction. Good surgical results depend not only on the surgical methods including proper anatomical knowledge, but also on proper preoperative assessment and proper planning of the surgery.

Always be sure of your diagnosis. Wrong diagnosis leads to wrong surgical planning and poor results. It is important to rule out causes of pseudo-strabismus in any case of strabismus.

**Preoperative Evaluation**

1) A thorough diagnostic work up includes assessment of vision by age appropriate tests, complete cycloplegic refraction, identification of amblyopia and the binocular status.

2) Always look for head posture—Abnormal head posture could be indicative of strabismus.


4) Measurement of deviation—Accurate determination of angle of deviation is of at most importance in achieving good surgical results. It is essential to learn correct placement of prism while measuring the deviation. Plastic prisms should be held in Frontal plane rather than in Prentice position. Errors induced by using Prentice position increase if the deviation is more than 20pd, which in turn alters the surgical results.

It is not for nothing that deviation is to be measured under different conditions to assess the different properties of strabismus. It is mandatory to measure the deviation for near distance and far distance to look for the variation of deviations in varying distances. The deviation has to be measured in all nine gaze positions to rule out any incompetence.

Look for any A/V pattern in each and every case of horizontal deviation.

Do not forget to measure the deviation with and without spectacle correction.

It is to be remembered that only the residual deviation after spectacle correction has to be operated. Plus lenses decreases while minus lenses increases the measured deviation. This is clinically significant with corrective lenses of more than 5 D. This artifact from the peripheral lens of spectacles may also be reduced by using lens in trial frame and moving them until they are centered in front of the visual axis of the deviating eye.

It is always advisable to include a prolonged patch test and examination under +3D lens to counter the fusion and accommodative element associated with the deviation.

5) Ocular motility—Part of strabismus measurement includes ocular motility examination. Both ductions and versions must be checked in all cases as in most of the cases, it might only be an apparent limitation of versions with full limit of ductions present. Always do a nine gaze motility examination. Associated, inferior or superior oblique overaction or underaction should be noted. Pattern deviation may be present with or without oblique overaction in which case surgical treatment is to modified accordingly.

6) Note for any retraction and narrowing of palpable aperture.

7) Do not miss any nystagmus or nystagmoid movements.

8) Do not forget to do a complete anterior and posterior segment examination in all cases. Elaborate systemic investigations are not indicated in most cases of strabismus. Neurological evaluation including neuro-imaging is indicated in cases of paralytic strabismus, acute comitant strabismus or when myasthenia gravis is to be excluded.

**Planning of treatment**

Precise treatment plan individualized to the amount and type of misalignment is needed to improve the outcome. It is important to ensure that optimum optical correction and adequate amblyopia treatment is given before planning further management. Whenever necessary non surgical treatment should be tried before surgical intervention. Orthoptic exercises have a role in certain cases of intermittent squint.

While planning surgical treatment, it is to be remembered that only the static and not the dynamic deviation is to be operated upon.

Plan the eye to be operated. Usually the surgery is decided based on the fixing eye (dominant eye) and the deviated eye is operated. But in cases if the paralytic eye is dominant, the secondary deviation needs to be corrected. At times both eyes have to be tackled at the same time.
Choice of surgery

The procedures in strabismus surgery are either weakening or strengthening procedures in one or more muscles horizontal or vertical in one or both eyes.

It is not necessary to do symmetric procedures in the two eyes but to establish symmetry between the eyes. So our aim is to do symmetrizing procedures.

Large deviation requires three muscle surgery. The amount of surgery may be preferably split between the two recti to be operated rather than accomplishing large muscle surgery in one muscle alone. Extensive recession/resection in one eye may correct the deviation but can induce incomitance in extreme lateral gaze and alter palpebral aperture.

Not more than two recti have to be operated in one eye in the same sitting. If so surgery has to be spaced by six weeks.

The choice of adjustable/fixed sutures should be decided before hand.

FDT needs to be done preoperatively in cases where a restrictive pathology is suspected. It also needs to be repeated at various times during surgery.

Some of the children having squint may have associated cardiac or neurological problem which should be evaluated by the anesthetist. He should be aware of the occurrence of the oculocardiac or oculorespiratory reflexes and malignant hyperthermia more common in strabismus surgery.

Communication with patient/parent

The communication between the patient and the surgeon should be encouraged to answer any queries or fears. Ensure that the patient does not hold any undue or impossible expectations. Fear about anesthesia especially in young children is a common concern. The eye or eyes to be operated should be discussed. More so in cases deviating from the usual protocol, lest the patient feels that the wrong eye has been operated. At times both eyes are operated and the patient taken into confidence to avoid any litigations.

If the surgery is for cosmetic reasons the fact that visual improvement is not expected should be conveyed in clear terms.

The need for stepwise surgery or resurgery should be explained. The possibility of post operative diplopia is another aspect that should not be neglected.

Surgical Principles

The knowledge of anatomy of extraocular muscles is essential prerequisite for any strabismus surgeon. The distance of each rectus muscle from limbus is different for each individual and in infants. The two ends of the muscle also differ from the midpoint in muscle limbal distance. While recessing one should also measure this distance and take it into account. The width of the muscle(9-10)mm should be restored after suturing for normal physiological actions.

The surgeon should be aware of the torsion of the eye induced in anesthesia which may cause confusion between horizontal and vertical muscles. The vertical recti has a slightly oblique insertion. Their temporal ends are further receded than nasal while horizontal recti have ends equidistant from limbus. Other than medial recti all the three have an association of oblique muscles which should be noted.

The sclera is thinnest (0.3mm) at the insertion of recti. So at least 0.5 mm stump should be left for resection resuturing.

Muscle sheath should always be preserved. Tenons capsule 8 mm posterior to muscle insertion should not be violated to prevent fat prolapsing and cause adherence syndrome.

Measurement for recession is made from the upper and lower ends which are never handled with forceps. This prevents any induced shift. The fixation forceps hold the insertion at the middle.

While passing sutures on the muscle on the muscle in recession, it should be placed no more than one mm from the end of the insertion. Placing the sutures too posterior will impose a resection effect on a recessed muscle.

Spatulated needles should pass paralimbal away from each other at a mark about 8-10 mm width. Suture bites should not be too deep (to avoid scleral perforation) or too superficial (to avoid slippage).

While working too posterior especially in supramaximal recession, avoid penetration of globe and injury to vortex vein.

While performing vertical transposition, keep the muscle insertion concentric to the limbus. An essential prerequisite before a transposition is attempted is absence of any restriction of antagonist as seen by FDT.

A faulty technique of hooking may cause splitting of muscle fibres causing laceration and fibrosis. A proper dissection to ensure that full width is hooked is important.

A loose suture or partial thickness suture may cause more weakening than required.

Improper apposition of conjunctiva allows tenon to prolapse and cause implantation cysts.

Post operative

Mild emesis is more common in children. It may be related to muscle handling or effect of certain drugs. Newer anesthetic drugs and techniques can reduce the incidence to a great
extent.

Bunching up of conjuctiva especially after medial rectus resection may tend to cause corneal dellen due to local disruption of tear film. It disappears on rehydration.

Anterior segment ischemia thorough rare is a serious disorder, which if promptly recognized is amenable to treatment though after a prolonged clinical course.

About 10% undercorrection and 5% overcorrection may occur even in expert hands.

This may occur in immediate postoperative period or delayed due to postoperative drift. Other than marked overcorrection usually due to muscle slippage, which is managed without delay, resurgery for overcorrection is done after 6 weeks or three months. This is evaluated after observing version for any incomitance and FDT. Small residual strabismus is easily managed by orthoptic fusion vergence or by manipulating the spectacle correction.

The need for use of spectacles even after surgery in appropriate cases, and maintenance of amblyopia therapy and the need for regular follow up are never to be underestimated in any case.

Precision is the key word in strabismus. Though a hundred percent precise and predictable outcome is a dream rarely fulfilled, a thorough understanding of the underlying principles, proper planning and execution of the procedure is sure to bring optimal results and prolonged gratification both for patients and surgeon.

Suggested Reading

Dr Anitha Balachandran did her MS from RIO Trivandrum.
Presently working as Associate Professor at RIO Trivandrum
In Charge of the Paediatric Ophthalmology Unit For The Past 12 Years.
Her area of interest is Paediatric Ophthalmology & Strabismus
Pediatric Cataract Surgery: Consensus And Controversies

Abstract
Pediatric cataract surgery has improved significantly in terms of outcome over the last 2 decades primarily due to improvements in microsurgical instrumentation and techniques and the management of posterior capsule at the time of cataract surgery itself. The essential steps that can ensure a good outcome surgically are adequately sized anterior and posterior continuous curvilinear capsulorhexis, a limited but sufficient anterior vitrectomy and intraocular lens insertion in the capsular bag. Surveillance and care during the immediate postoperative period for intraocular inflammation is of paramount importance. Yet, some issues need to be resolved and refined in pediatric cataract surgery such as at what age can intraocular lenses be safely inserted and what is the best power to put in a growing eye. The most important aspect of pediatric cataract surgery remains the aftercare with appropriate long term addressal of residual refractive error, amblyopia and possible complications like secondary glaucoma.

Key words: pediatric cataract, surgery, capsulorhexis, intraocular lens.

Pediatric cataract surgery has come a long way with the outcome improving dramatically over the last 2 decades with advancements in microsurgical techniques and instrumentation. Yet, taken as a whole, management of a child with cataract remains one of the most complex and difficult tasks confronting an ophthalmologist. This article seeks to discuss the status of management as it stands today, both the issues which appear settled and some where the jury is still out.

Timing of surgery:
Regarding the timing of surgery, the general rule is that earlier the visual axis is cleared, the better the visual outcome. However this does not mean that all pediatric cataracts are best removed as soon as they are identified. Weighing the risks with the benefits, the indication for surgery would be cataracts centrally located and greater than 3mm in size that obscures the red reflex on distant direct ophthalmoscopy. In order to assess visual status of these young children with media opacities, it is helpful to quantify visual acuity when possible with Cardiff or Teller acuity cards or Lea symbols. Presence of squint or nystagmus suggests that it is already late for intervention and these children should be taken up for surgery on an urgent basis. Unilateral cataracts need more urgent intervention as compared to bilateral cataracts as they are more amblyogenic. As regards the earliest age to take a child up for cataract surgery, the main consideration to be kept in mind is the anaesthetic risk, which seems to be quite minimal even in the immediate newborns. Current evidence seems to suggest that the window period we have from the amblyopia perspective is 6 weeks from birth for unilateral congenital cataracts and 3-4 months in bilateral cases.

Investigations
Half the bilateral cases are idiopathic in origin and 1/3rd are hereditary without systemic associations, while most cases of unilateral congenital cataract are idiopathic. Hence there is no need to waste precious time and money on unnecessary investigations, especially when they wouldn’t influence management greatly. Unless there are clear evidences to suggest specific diseases or syndromes, it would be judicious to restrict investigations to hemoglobin (anaesthetic considerations) and blood sugar.

Surgery
Pediatric eyes need to be handled a little differently from adult eyes due to the following reasons:
1. Pediatric eyes have reduced scleral rigidity and so tend to collapse more easily during surgery and to leak postoperatively.
2. Anterior capsule is more elastic and therefore needs more force to both fracture and tear. To make matters worse, it has a constant tendency to run out to the periphery.
3. There is more violent inflammatory reaction postoperatively.
which sometimes results in fibrin formation towards the end of surgery itself.

4. Mitotic lens epithelial cell proliferation is much more active in the pediatric eyes and so visual axis obscuration (VAO) by posterior capsule and/or anterior vitreous face opacification is invariable unless managed primarily by posterior capsulorhexis and anterior vitrectomy.

Technical aspects of surgery:

Wound construction:
A valved incision of the smallest length necessary for instrumentation would help maintain anterior chamber which has a constant tendency to collapse. For the same reason bimanual irrigation aspiration and a 2 port vitrectomy is useful as opposed to irrigation from the same port. It is preferable to suture all wounds in children below 10 years of age.

Anterior capsule management:
The anterior capsulotomy size, shape and edge characteristics are crucial for both immediate and long term centration of IOLs in the capsular bag. Various options for anterior capsulotomy in pediatric cataract surgery are manual continuous curvilinear capsulorhexis (CCC), vitrectorhexis, radio-frequency diathermy and Fugo plasma blade. Among these manual CCC was found to produce the most extensible opening with best edge characteristics. Though it is the most difficult to perform and control in pediatric eyes for reasons enumerated earlier, it remains the gold standard for anterior capsulotomy in pediatric eyes (Figure 1).

A high molecular weight viscoelastic agent like Healon or Healon GV and capsular staining with Trypan Blue is helpful while performing CCC. While tearing the capsule, the direction of pull on the capsular forceps should be centripetal. Repeated regrasping of the capsule for short tears and aiming for a smaller capsular opening prevent running out of the capsular tear to the periphery (see video - http://www.youtube.com/watch?v=ulU7Q0LSf5Q). The edge of the lamellar cataract often serves as a guide to the ideal size to be aimed for. For the less experienced pediatric surgeon, it is safer to err on the side of a smaller rhexis and then to make it of the appropriate size after IOL insertion as shown in the video (http://www.youtube.com/watch?v=EipthArLSOM).

A small rhexis in the postoperative period runs the risk of capsular phimosis and capsular contraction syndrome due to the violent inflammatory response characteristic of children. Manual CCC is especially difficult to perform in very young infants because the capsule is highly elastic. Here vitrectorhexis is a good option as owing to the highly elastic capsule, the edge remains regular and resists radial tearing. In the older children, the capsulotomy edge of a vitrectorhexis is scalloped and never as regular as a CCC.

Lens matter removal
Being soft, lens material only needs aspiration and none of the nucleus management techniques of adult phacoemulsification surgery. It can be done either with a symcoe cannula or by automated irrigation-aspiration handpiece of the phacoemulsifier. Very rarely only, if the cataract is harder, it becomes necessary to resort to the larger diameter of the phaco probe for irrigation aspiration or actual ultrasound energy.

Posterior capsule management
Visual axis obscuration by posterior capsular opacification is the commonest cause of poor outcome following pediatric cataract surgery. Maintaining a clear visual axis continually is of paramount importance in the amblyogenic age group. It is well accepted now that the posterior capsule is best managed by primary posterior capsulectomy and

Figure 1: Post operative photograph on the 3rd day of a 2 year old boy showing both anterior and posterior capsulorhexis and a PMMA IOL in capsular bag

Figure 2: Three month post operative photograph of an 18 month old boy showing opacification of anterior capsular rim, early opacification of posterior capsule and clear visual axis due to a PCCC and anterior vitrectomy
anterior vitrectomy during the cataract surgery itself (Figure 2, 3). Like anterior capsulectomy, among the various options available, a manual PCCC is the gold standard. The ideal size of the posterior capsulotomy opening is 3.5-4 mm. A smaller opening runs the risk of closing off (Figure 4). Most surgeons perform PCCC before IOL insertion (http://www.youtube.com/watch?v=ulU7Q0LSf5Q and http://www.youtube.com/watch?v=EipthArLSOM) while others prefer to lift the IOL with the non-dominant hand and perform it after the IOL is inserted.

Anterior vitrectomy
In young children, below 6-8 years, a posterior capsulectomy alone would not ensure a clear visual axis. The anterior vitreous face would in such cases invariably be covered by migrating epithelial cells and metaplastic cells in the post-operative period. Therefore a primary limited anterior vitrectomy is recommended in young children. The current recommendation seems to be a PCCC with anterior vitrectomy in children below 3 years, a PCCC alone between 3 and 7 years and leaving the posterior capsule intact in older children and who are likely to cooperate for a YAG Capsulotomy later on.

Pars plana vitrectomy
Some surgeons prefer to perform anterior vitrectomy through pars plana route after IOL insertion. This technique has the advantage of making IOL insertion less risky and also making possible a larger posterior capsulotomy. It is especially useful in resurgeries to remove posterior capsular and vitreous opacifications behind the IOL.

IOL implantation
Primary IOL implantation is the recommended practice in children older than 2 years. In children below 2 years it is controversial. IOLs seem to increase the chances of posterior capsular opacifications (PCO)13-15. Yet with increasing use of Acrysof IOLs, more and more surgeons are implanting lenses in infants as young as 3 months of age. The absolute contraindications to IOLs are microcornea and microphthalmos while relative contraindications include uveitis, glaucoma, aniridia and persistent hyperplastic primary vitreous. The important determinants of outcome related to IOLs are material, position and size of IOLs. PMMA has the longest track record but current opinion seems to favour hydrophobic acrylic16-24. PCOs following Acrysof IOLs set in later and are also predominantly proliferative as opposed to fibrotic in PMMA cases. VAO produced by Acrysof lenses are less severe than those caused by PMMA lenses and so are less amblyogenic25. IOL sizing was a major issue with PMMA lenses in smaller eyes especially in infants but seems to be a lesser problem with Acrysof. The haptics are extremely flexible and have excellent...
memory and single piece Acrysof lenses adapts well to the smallest capsular bag without becoming decentered. In the bag placement is the best position to ensure long term stability and centration of IOLs. This is why a well made CCC is crucial to the success of pediatric cataract surgery.

Optic capture of IOL in the PCCC, championed by Gimbel, was introduced to obviate the need for primary anterior vitrectomy. While it produces excellent centration of the IOL, it causes increased inflammatory response leading to opacification of vitreous face and so is currently not very popular.

IOL power selection

This is an area where pediatric surgeons have different opinions and protocols. In general, an IOL power which produces emmetropia in the immediate post operative period will eventually end up causing myopia as the eye grows in axial length. Younger the child, more the myopia. To eventually aim for emmetropia, the child will have to be left hypermetropic immediately after surgery, which is amblyogenic and which has to be managed appropriately with reducing power of plus lenses. There are many tables available for IOL power selection, but it is not easy to predict the rate of growth of an individual eye and its final refractive outcome. It is influenced by several factors like normal growth rate, age at surgery, visual input, presence or absence of IOL, laterality and genetic factors. A recent study showed that the residual refractive error after cataract surgery does not influence the myopic shift. Some surgeons advocate aiming for emmetropia in children older than 2 years of age in order to avoid potentially amblyogenic residual hypermetropia. But this is likely to lead to significant myopia eventually. Predicting rate of growth and the resulting refractive change remains a major challenge of pediatric cataract management.

Contact lens

In children who are not implanted with an intraocular lens due to any cause, contact lens is a good option for visual rehabilitation. But compliance and safety are major issues in developing countries with the result that more and more pediatric ophthalmologists are implanting IOLs at a younger age.

At what age can IOLs be safely implanted?

As mentioned earlier, the current recommendation is still for IOLs after 2 years of age where the axial length of the child’s eye approaches 90% adult dimensions. There have been a few studies comparing visual rehabilitation with contact lenses and intraocular lenses, with the largest being that of the Infant Aphakia Treatment Study Group (IATS). In a randomized, multicenter (12 sites) clinical trial, 114 infants with a unilateral congenital cataract were assigned to undergo cataract surgery between 1 to 6 months of age either with (57 infants) or without (57 infants) primary IOL implantation. Contact lenses were used to correct aphakia in patients who did not receive IOLs. Grating visual acuity tested at 1 year of age by a masked examiner showed no statistically significant change between the 2 groups. Additional surgeries were required more frequently in the IOL group and the authors concluded that caution should be exercised when performing IOL implantation in children aged 6 months or younger given the higher incidence of adverse effects and the absence of improved short term visual outcome compared with contact lens use. Nevertheless several things should be noted in this study as highlighted in the discussion by the authors themselves. One, the contact lens group had a very high compliance rate (average more than 80% of waking hours). This was ensured in the study by providing free contact lenses and patches and frequent monitoring of compliance by telephone calls. This is difficult to duplicate in societies like ours and so what this study estimates is the efficacy (benefit under ideal conditions) rather than effectiveness (benefit under usual conditions). Secondly, assessing the risks and benefits of IOL implantation at 1 year of life may lead to premature conclusions. The real benefit of IOL implantation may occur later, especially if children in the contact lens group become less compliant with contact lens use as they become older. If this is true, it is possible that the children in the IOL group will have an increasing visual advantage with their pseudophakic correction alone as they become older and approach emmetropia. The study plans to retest the visual acuity of these children when they are aged 4 years using the Amblyopia Treatment Study–HOTV acuity test.

The main difference between the 2 groups was in relation to number of additional surgeries and the adverse effects. The main adverse effects noted are lens proliferation in the visual axis and pupillary membranes which were the reason for the additional surgeries. There was no significant difference in the incidence of secondary glaucoma. Cataract surgery in infancy is itself a risk factor for glaucoma, but IOL doesn’t seem to increase the risk. In fact, some studies even suggest that IOLs have a protective effect against secondary glaucoma. We can look forward to the IATS study which is supposed to evaluate the children again at 5 years for secondary glaucoma.

So on current evidence, in the debate on IOLs versus contact lenses in the very young, what is in favour of contact lenses is the lesser incidence of post operative VAOs while this advantage might be offset by a lower visual acuity related to poor compliance issues.

Postoperative treatment and follow up

A good job on the surgical table is less than half the job done in case of pediatric cataracts. In fact the treatment would
have just started with the surgery. The immediate post operative period is vital because if the inflammation is well controlled then, the anatomical results remain good. It is therefore important to see the child very frequently during the first few post operative days and to be aggressive with topical and, if necessary, systemic steroids. The next main concern is amblyopia for which appropriate spectacles and patching in unilateral cases is to be started as soon as possible. These children need to be followed up regularly and monitored for vision and change in refraction. Aphakic and pseudophakic glaucoma is a well recognised complication and can occur at any time after cataract surgery. Therefore these children need to be monitored for life.

**Conclusion**

Surgical management of pediatric cataracts is different from adult cataracts. The reduced scleral rigidity, elastic lens capsules and positive vitreous pressure make surgical manipulations more difficult. The high rates of posterior capsular opacifications make PCCC and anterior vitrectomy mandatory in the younger age group. Ocular growth makes selection of IOL power a difficult choice. However outcomes have improved greatly in the last few decades and with better microsurgical instrumentation and techniques and improved understanding of pediatric eye growth, we can hope for more.

**References**


Dr Satish Thomas had his post graduate training from Dr. Rajendra Prasad Centre for Ophthalmic Sciences, AIIMS following which he has also done DNB in ophthalmology. He has subspecialised in Pediatric ophthalmology and Strabismus, having done a long term fellowship at Sankara Nethralaya, Chennai and a short term ICO fellowship at Children’s Hospital, Westmead, Sydney under Prof. Frank Martin. He is currently Professor of ophthalmology at Christian Medical College and Hospital, Ludhiana. His areas of special interest include pediatric cataract, complex strabismus and retinopathy of prematurity.
Traditionally the diagnosis of primary open angle glaucoma (POAG) is a triad of increased intraocular pressures, characteristic optic nerve head (ONH) changes and corresponding visual field changes. Recent definition of glaucoma excludes intraocular pressure as a defining factor (1). The diagnosis of glaucoma depends on functional psychological tests and imaging technology of ONH morphology. Structural changes precedes that of functional changes. Despite the advent of new technology to assess ONH changes with Heidelberg Retinal Tomogram (HRT), Scanning Laser Ophthalmoscope (SLO) and Optical Coherence Tomography (OCT) available for research, clinical acumen in detecting glaucomatous features of ONH is imperative in routine clinical practice. Very often in our busy outpatient practice, large disc associated with large cup and similar lesions are over diagnosed and labeled as “glaucoma”. It is therefore imperative to differentiate glaucoma from non glaucomatous disc anomaly. Several conditions pose as ONH mimickers and these lesions often reiterates the need of careful clinical examination of ONH.(2,3). We present a series of three such cases.

Case-I

A 40-year old male presented with complaints of defective vision for distance. On examination, best corrected visual acuity in both eyes was 6/9 N6. Right eye - -9.00 Dsph and left eye -8.00/-1.00 Dcyl x 90º. Applanation tonometry in the right eye and left eye was 20mmHg and 23mmHg respectively. Extra ocular movements, pupil and anterior segment was within normal limits. Fundus picture showed tilted optic disc with prominent inferonasal margin as in Fig.1.

Fig. 1

Visual field examination done in 2002 showed corresponding arcuate defects emanating at the disc and enlarging beyond it as seen typically in glaucoma (Fig.2).

Fig. 2

However, the fields remained stable on the following visits. Fields in 2003 showed no progression (Fig.3). The diagnosis of congenital optic disc syndrome was made.

Points to remember regarding congenital tilted disc syndrome are as follows:

♦ Bilateral
♦ Situs inversus of retinal vessels
♦ Inferior or inferonasal crescent or conus -Fuch’s coloboma
♦ Prominence of inferonasal fundus
♦ Posterior inferonasal Staphyloma 6-9 diopters
♦ Refractive error – astigmatism/myopia
♦ Field defects refraction scotoma or bi-temporal hemianopia which may need neuro imaging. (4,5)

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Case-II

A 52 year old male presented with complaints of eye strain in 2005. On examination, his unaided visual was 6/6 in both eyes and near vision was N6. Extra ocular movements, pupil and anterior segment were within normal limits. Applanation tonometry in both eyes was 14mmHg. The diurnal variation of tension in the right eye was 12-18 mmHg and left eye was 12-16mmHg. Fig(4) shows abrupt absence of normal optic disc structure with inferior disc pallor and corresponding wedge shaped nerve fiber layer defect better seen in red free photograph.

The fields in Fig(5) shows characteristic superior defect which remained the same and did not progress (Fig.6) as in a typical glaucoma.

Case-III.

A 48-year old man came for a routine ophthalmic evaluation for renewal of driving license. On examination, best corrected visual acuity was 6/6 N6. Extra ocular movements, pupil and anterior segment were normal. Applanation tonometry in both eyes was 12mmHg. Fundus picture in Fig (7) shows a typical double disc sign with bridge of retinal tissue called bridge coloboma and shows an abrupt absence of normal optic disc feature.

Note the large size of optic discs in both eyes with normal structure of superior discs. Also, left eye has an optic nerve pit. Fields in Fig.(8) in the right eye showed enlargement blind spot and left eye shows typical superior arcuate scotoma.

Red free photograph of left eye confirms inferior NFL defect in this area. (Fig.9)
Case II and III are different spectrum of ONH coloboma. Case III left eye also had ONH pit. The characteristic features of optic disc coloboma are:

- Non closure of fetal fissure
- Bowl shaped excavation occupying a large disc
- Excavation decentered inferiorly
- Inferior neuro retinal rim is thin or absent
- No glial tissue
- Normal peripapillary retinal blood vessels
- Familial and bilateral
- Serous macular detachment is common
- Iris, ciliary and retinal coloboma are associated

- PAX 2 gene mutation
- Visual acuity affected with involvement of papillomacular bundle
- Common in females, as hemiamnionopia with associated trans sphenoidal basal encephocele, mid facial anomalies and clefting syndrome
- Retinal detachment
- Sub-retinal choroidal neovascular membrane.

The systemic conditions associated with colobomas are:
- Charge Syndrome (coloboma, heart defect, atresia choanae, mental retardation, genito urinary abnormalities, ear defects)
- Goltz syndrome
- Lenz microphthalmia syndrome
- Meckel – Gruber syndrome
- Walker Warburg syndrome.
- Goldenhar’s syndrome

Optic disc coloboma is classified as an excavated optic disc anomaly which are as follows:
- Morning glory disc anomaly
- Optic disc coloboma
- Peripapillary Staphyloma

The features of Morning glory syndrome are:
Morning glory disc is akin to the flower of the same name in Figure 11. Also seen are the straight course of vessels in the angiogram.
The ophthalmic differences between morning glory disc and peripapillary coloboma are - .

Peripapillary staphyloma is usually unilateral with deep fundus excavation surrounding the disc. There are posterior scleral defects. The normal cup is placed within an atrophic area. There is no glial tissue. Pigmentary changes are present. Retinal vessels are normal. Vision is normal but slightly myopic. Scotoma or enlarged blind spots may be present. No systemic association.

Ophthalmic differences between peripapillary Staphyloma and morning glory disc.

**OPTIC DISC PITS**

Optic disc pits are found in 1 in 10,000 cases. They are unilateral and affected disc is larger in size. The pit is present in the temporal part of the disc and often the disc has a cilio-retinal artery. 25-75% of eyes are associated with serous macular detachment and called Kranenberg’s syndrome. Paracentral arcuate scotoma with enlarged blind spot are common and field defects poorly correlate with location of pit. Pathogenesis is uncertain and has no systemic association.

Therefore, different optic disc anomalies should be considered before diagnosing glaucoma in routine practice. The common optic disc anomalies mimicking glaucoma are -

- Normal variants include a congenital deep cup, large physiological cup, myopic disc
- congenital tilted disc.
- Morning glory syndrome
- Optic disc coloboma,
- peripapillary Staphyloma,
- optic disc pit.

Table 1 gives ophthalmic differences between Morning glory disc and optic disc coloboma.

<table>
<thead>
<tr>
<th>MORNING GLORY DISC</th>
<th>OPTIC DISC COLOBOMA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Optic disc lies within the excavation</td>
<td>Excavation lies within the disc</td>
</tr>
<tr>
<td>Symmetrical defect (Disc lies within the excavation)</td>
<td>Asymmetrical defect (Excavation lies inferiorly within the disc)</td>
</tr>
<tr>
<td>Central glial tuft</td>
<td>No glial tuft</td>
</tr>
<tr>
<td>Severe peripapillary pigmented disturbances</td>
<td>Minimal peripapillary pigmented disturbances</td>
</tr>
<tr>
<td>Anomalous retinal vasculature</td>
<td>Normal retinal vasculature</td>
</tr>
</tbody>
</table>
Table 2 enlists ocular and systemic differences between morning glory disc and optic disc coloboma.

<table>
<thead>
<tr>
<th>MORNING GLORY DISC</th>
<th>OPTIC DISC COLOBOMA</th>
</tr>
</thead>
<tbody>
<tr>
<td>More common in females, rare in black</td>
<td>No sex or racial predilection</td>
</tr>
<tr>
<td>Rarely familial</td>
<td>Often familial</td>
</tr>
<tr>
<td>Rarely bilateral</td>
<td>Often bilateral</td>
</tr>
<tr>
<td>Not associated with iris, ciliary and choroidal colobomas</td>
<td>Iris, ciliary and choroidal colobomas common</td>
</tr>
<tr>
<td>Rarely associated with multisystem genetic disorders</td>
<td>Often associated with multisystem genetic disorders</td>
</tr>
<tr>
<td>Basal encephalocele common</td>
<td>Basal encephalocele rare</td>
</tr>
</tbody>
</table>

Table 3 enumerates differences between peripapillary staphyloma and morning glory syndrome.

<table>
<thead>
<tr>
<th>PERIPAPILLARY STAPHYLOMA</th>
<th>MORNING GLORY DISC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deep cup shaped excavation</td>
<td>Less depth funnel shaped excavation</td>
</tr>
<tr>
<td>Relatively normal, well-defined optic disc</td>
<td>Grossly anomalous poorly-defined optic disc</td>
</tr>
<tr>
<td>Absence of glial and vascular anomalies</td>
<td>Central glial bouquet of vascular anomalies</td>
</tr>
<tr>
<td>Embryological: fifth month</td>
<td>Embryological: four weeks</td>
</tr>
<tr>
<td>Defect in posterior sclera</td>
<td>Defect in distal optic stalk</td>
</tr>
</tbody>
</table>

Table 4 enlists the difference in glaucomatous and non-glaucomatous features of ONH

<table>
<thead>
<tr>
<th></th>
<th>Glaucomatous</th>
<th>Non-glaucomatous</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>Usually elderly</td>
<td>Younger age group with congenital anomalies</td>
</tr>
<tr>
<td>Laterality</td>
<td>Primary glaucomas are bilateral</td>
<td>May be unilateral or bilateral</td>
</tr>
<tr>
<td>Presentation</td>
<td>Asymptomatic</td>
<td>Incidental or on evaluation of headache</td>
</tr>
<tr>
<td>Visual acuity</td>
<td>Good until late stage of disease</td>
<td>Poor if papillomacular bundle involved</td>
</tr>
<tr>
<td>Colour vision</td>
<td>Correlates with acuity and maintained until end stage disease</td>
<td>Correlates with involvement and extent of the lesion</td>
</tr>
<tr>
<td>Optic disc features</td>
<td>Normal to small size</td>
<td>Usually large sized</td>
</tr>
<tr>
<td>---------------------</td>
<td>----------------------</td>
<td>---------------------</td>
</tr>
<tr>
<td></td>
<td>Focal or uniform</td>
<td>Unaffected part of the disc appears healthy</td>
</tr>
<tr>
<td></td>
<td>affliction of glaucoma</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pallor proportionate to the cup</td>
<td>Pallor out of proportion to the cup</td>
</tr>
<tr>
<td>Field defects</td>
<td>Nasal step, arcuate scotoma</td>
<td>Central, cecocentral hemianopia or arcuate scotoma</td>
</tr>
<tr>
<td>Progression</td>
<td>Invariably progresses</td>
<td>Will not progress</td>
</tr>
<tr>
<td>Systemic associations</td>
<td>Absent</td>
<td>May be present esp with congenital anomalies</td>
</tr>
<tr>
<td>Family history</td>
<td>May be present</td>
<td>Absent</td>
</tr>
</tbody>
</table>

**References:**


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Dr Savita Bhat after her DO,MS, DNB and initial training at CMC Vellore is senior consultant at Giridhar Eye Institute, Kochi
(6) Preliminary Work done

While writing the protocol it is always necessary to include a write-up on the work already done by the investigator in the topic of the research project. For funding projects this is very much necessary to justify the importance & relevance of the topic based on the results obtained from the preliminary work done and to assess the expertise of the investigator in the topic of research on which the protocol is prepared. This need not be based on a planned project w.r.t. sample size requirements and other aspects of a fullfledged research project. The investigator can highlight the results obtained from the preliminary study and justify its importance and relevance and his/her knowledge in the area of research and the facilities available for conducting the research and the shortcomings, if any, in the conduct of the research work done, suggesting the possible ways and methods of improving them and thus making them scientifically acceptable. One of the important questions asked in this context is the availability of budget required for carrying out the preliminary study. Normally this has to be arranged by the investigator himself/herself, either from the Institute, Organization in which he/she is working or from some other possible sources. Some Institutes like, All India Institute of Medical Sciences, New Delhi has some provisions for financial assistance to the faculty/researchers in AIIMS for carrying out the preliminary work on the chosen topic. Many funding agencies/Organizations like ICMR, WHO, DST and DBT include this requirement in their Form meant to be used for requesting for financial assistance for carrying out research projects.

(7) Justification of the study

The Principal Investigator (PI) has to convince the Funding agency with proper justifications for the study he/she would like to carry out and financial assistance is requested for from them. Advantages, both specific and general w.r.t. the population of patients/subjects from which appropriate samples are to be included in the study have to be highlighted. Benefits in terms of the effect of the treatment and its implications on health, social and economic components have to be explained clearly. In other words, proper justification has to be given for the funding requested for from the Funding Institution. In this context one of the questions which may be asked by the Funding agency could be that whether any other similar study was going on or not and if yes, whether the requested study could be merged with the ongoing study in any way. Though this may not be practically feasible due to a variety of reasons it might be relevant to include proper reasons for not being possible to merge the requested project with any ongoing similar studies. Appropriate reasons for convincing the Funding agency that the requested project is essential to be carried out independently will help a lot to convince them to consider for financial assistance to the requested project.

(8) Study Designs

Identifying and selecting the appropriate Study Design is very important in any research project. Many researchers confuse the study design with the sample size. Study design and sample size are entirely different. Study design is the method by which a plan is designed keeping in view the objectives of the study. It enables us to estimate the magnitude of the problem (prevalence of complete blindness), to identify/confirm its causative factors or to find out appropriate treatment modalities. Selection of the appropriate study design is one of the major components of any research study. A description of the study designs is given below:

The very basic study design is – case history. For example when the first case of HIV was detected the doctor described the case and published it. That is the starting point. When a group of HIV cases were identified – Case series analysis was planned which describes the cases available in summary forms, like mean age, sex ratio, percentage of cases in different socio-economic groups etc. These two designs are planned in the very preliminary stages of the study when the first patient and later a series of patients of the same disease were spotted. The classical designs for the different types of studies are explained below:

Basically there are two types of designs:

(A) Non-intervention / Observational / Descriptive studies: Observes, Collects and Describes

(B) Intervention / Experimental studies: Intervene with some intervention material / method -measures the outcome and compares between groups

(A) Non-intervention Studies

(b) Explorative Studies:

Such studies are Small studies with a shorter duration and are...
planned when very little is known about the problem (Case study, Case series analysis, explained above)

(b) Descriptive Studies:

This design is planned for the systematic collection of data on a disease/condition (for example, diabetic retinopathy) to get clear picture of the problem which will enable the researcher to estimate the magnitude of the problem, to identify the possible causative factors, or to find out whether the different lab parameters are correlated or not with the clinical parameters. These types of studies fall under the following designs:

Cross-sectional studies:

Planned to estimate the Prevalence (total cases prevalent at a specified period of time), Incidence (new cases identified during a specified period of time) of the diseases/conditions under study or to study whether different lab parameters are correlated or not with the clinical parameters, socio-demographic and behavioural variables (habits of smoking, use of alcohol, life style, etc.) or to study knowledge, attitude and practice (KAP) w.r.t. any problem, say, viewing TV continuously for a long time and the knowledge, attitude and practice w.r.t. its harmful implications.

Diagrammatic representation of the cross-sectional design is given below

![Diagram of-cross-sectional design]

- Disease Present
- Disease Absent
- Risk Factor Present
- Risk Factor Absent

(c) Comparative OR Analytical Studies:

Though the cross-sectional studies enable us to identify/attempts to establish the possible causative factors for the occurrence of a disease/condition, its role in the causation of the disease cannot be confirmed from such studies. It only helps us to determine the frequency & burden of the disease under study and to generate research questions and develop appropriate research hypotheses: For confirming the role of the specific causative factors comparative studies have to be designed. Two important designs adopted for this purpose are:

- Case-Control Studies
- Cohort Studies

(1) Case-control studies

In case-control studies, a certain number of cases and non-cases are selected from the corresponding populations and data on the presence/absence of possible causative factors among them are collected. For example, cases may be children with low vision and control may be children with normal vision. Sample size in each group has to be estimated based on the results observed in the past studies and the amount of confidence and power required and using a statistical formula. Principles & methods of estimating minimum sample size for the study depending upon the design of the study and other aspects of the study will be explained in a separate chapter, in a later issue. The diagrammatic representation of this design is given below.

Statistical significance of the role of the risk factor can be studied by applying appropriate statistical analysis. The advantages of case-control studies are: results can be obtained quickly, less expensive and very useful in case of rare diseases. Main disadvantage is that only odds ratio for the risk factor can be estimated and not the relative risk. In any study on the risk factors of the disease, it is ideal to find out the relative risk of the causative factor. It means the risk...
(2) Cohort studies

In case of Cohort design, a certain number of subjects with the factor (say, those who view TV continuously for long duration) and a certain number of subjects without the factor (those who view TV for shorter duration) are selected from the corresponding populations and they are followed up for a certain period and see how many in each group develop the disease/condition. For example, two groups of children—one group of children viewing TV continuously for a long time daily and another group viewing TV only occasionally. Sample size in each group has to be estimated based on the results observed in the past studies and the amount of confidence and power required and using a statistical formula. The diagrammatic representation of this design is given below:

(A) Population with the presence of the Risk factor
(B) Population with the absence of the Risk factor (much higher than A)

Sample

Risk Factor present
Risk Factor absent

Follow up period

Disease occurs
Disease does not occur

Follow up period

Disease occurs
Disease does not occur
The advantages of the cohort design are: It confirms the role of the risk factor with more strength (relative risk) compared to Odds ratio in case of case-control studies. However, the major disadvantage is that if the end point occurs after a long time, results will be available only after a long time and depending upon the length of the follow-up cohort design may not be feasible. For example, in case of studying the role of smoking in the causation of lung cancer, the follow-up time will be very long and in such diseases, it may not be feasible to conduct the cohort study. Also, since there is a follow-up period, the problem of dropouts may be there and exposure status at the time of enrollment in the study may change over a period of time. Hence depending upon the requirement, the purpose of the study, time period and finance available for the study and the knowledge already available on the confirmation of the role of the risk factor in the causation of the disease, appropriate design (cross-sectional, case-control or cohort) design can be planned.

Guidelines which may be used for selection of the appropriate design (either case-control or cohort) are:

1. If no or very less information is available on the magnitude of the disease and its possible causative factors—CROSS-SECTIONAL STUDY
2. In case of rare diseases—Longer the interval between suspected cause and outcome—Financial constraints—CASE CONTROL STUDY
3. In case of Common diseases—Shorter the interval between suspected cause and outcome—No Financial constraints—COHORT STUDY

**B) Intervention (Experimental) Studies**

(a) Clinical trials

This type of design is planned to prove the efficacy of an intervention or to establish a statistically and clinically better intervention method (treatment with a drug, surgery, health advice like exercise or changing the life style such as following a particular diet pattern). Essential Features of an intervention design are: Component of subjects (say, patients), intervention material as indicated above and the response such as cured/not cured or improved/not improved or alive/expired or a decrease or increase in the value of a parameter. Another important part of the intervention design is that there will be a control group with no intervention to anybody in that group. For example, a clinical trial may be planned to compare the efficacy of a specific eye drops compared to washing the eyes with clear water for testing the efficacy of the eye drops for curing conjunctivitis. Sample size in each group has to be estimated based on the results observed in the past studies and the amount of confidence and power required and using a statistical formula. The diagrammatic representation of this design is given below:
(b) Prevention (Prophylaxis) trials

This design is adopted to study the efficacy of a method in the prevention of the occurrence of the disease. Classical example of prevention is through vaccination, immunization or health education. This design can also be used in assessing community health programmes such as nutrition, use of contraception and use of alcohol, & use of hard drugs and smoking.

The basic difference between clinical trials and prophylaxis trials is that while the former one is normally done in a hospital set-up, the latter one is done in a community set-up, usually on a much larger number of subjects.

(9) Estimation of minimum sample size & the method of selection

What should be the sample size in my study—a common question asked by any researcher. The answer for this is not very simple, for any researcher. Many researchers think that the sample size for their study can be readily got from a statistician just by asking for it—just like a commodity from a shop. But, the researcher should know that it is not a magic number and also not a universal figure. Sample size varies from study to study depending upon many criteria. Some researchers think that 30 will be adequate sample size for any study. This number may be ok theoretically based on a statistical theorem (Central limit theorem), but, not alright for a research study. Sample size of 10 may be adequate for some studies, but, 3000 may not be adequate for another study. The pertinent question is how much should be the small portion is determined by computing the Minimum Sample size. Sample size has to be estimated based on several information such as, the design of the study, type of study variables (whether measurable (pulse rate, weight, BP, eye pressure etc.) or categorical (presence / absence of cataract, blind / not blind, improved / not improved etc.), whether the study aims at only for estimation of a parameter (prevalence of blindness, mean value of eye pressure) or it aims at testing the statistical significance of a research hypothesis (Prevalence of cataract is significantly higher in diabetics than non-diabetics) and the required precision, confidence & power of the test (The concept of precision, confidence & power of the test and the method of estimating the minimum sample size for any particular study, depending upon the study design, type of study variable, precision, confidence & power required will be explained in a later chapter).

Once the minimum sample size is estimated the next question is how to select the required number of study subjects from the population of subjects. Population for a research study is defined as the total sampling elements (units) in the defined area at a particular period. For example, for the estimation of the prevalence rate of visual problems in school children (5 to 15 years of age) in Kerala, all the school children in Kerala studying in the schools at that time period form the population. A small portion of the population which truly represents the population with respect to the study characteristics is called ‘sample’. Most of the research studies are concentrated on samples than to the complete population due to the reasons such as much higher expenses, the time it might take to complete the study covering all the children and the feasibility of the study. The required number of study subjects have to be selected from the list of subjects in the population called sampling frame by random sampling method. There are many types of sampling methods which are commonly used for the selection of sample from the population, some of them for increasing the precision of the estimate (reducing the error in estimation) and some others for convenience in the community studies. These will be explained in detail in a later chapter.

(10) Research Tools

The tools to be used in the study should be explained clearly in the protocol. They could be a set of questionnaires, proformae, lab tests, clinical examination, investigations like X-ray, MRI, Snellen chart for measuring visual acuity, stereopsis, slit lamp, tonometry etc.

(11) Standardization of research tools

The tools which are going to be used in the study should be defined and explained clearly without any ambiguity. If more than one researcher / technician is going to be included in data collection / recording (for example, collaborative studies encouraged by ICMR), training of them should be held, preferably in a common place with the support of written guidelines. For example, assume that one item in the proforma in a community study is ‘income’. If it is not defined clearly, it could be taken as: (a) income of only the head of the household, or (b) income of all earning members in the household, or (c) income of all earning members in the household + income from agriculture, property, etc. None of them will give the correct indication of the economic status of the family. The correct method of getting the required information will be defining per-capita income, since the economic status of the family depends upon not only on the total income, but also on the number of members in the family. Similarly all the equipments & instruments which are going to be used in the study should be checked for any defect and the same should be rectified and calibrated so as to get the correct values on the test. Hence standardization of
each & every aspect of research tools is very important in any research study,

(12) Data Collection Methods

Basically there are two types of data collection methods; Primary Methods / Secondary Methods

1) Primary Methods:
   (1) Questionnaires  (2) Proformae (3) Clinical Examination
   (4) Recording of Laboratory, Social, Demographic and Behavioural Parameters

2) Secondary Methods

Secondary data (Published reports and papers, Annual reports Hospital, Census data, Doctors’ Clinic Records, School health records & reports, Communications from the concerned experts etc.)

It is always preferable to collect the data by primary method. ie; the investigator plans the study and collects / records the data directly based on a defined plan & format, which will be more scientific since the investigator has full control over the methods and correctness of information collected. But in case of secondary data, the reliability of information recorded in the report can be questioned since they are collected by another person/organization for a different purpose. Also, utmost care should be taken while collecting data on sensitive information like, use of drugs which are abused, use of alcohol, sexual & contraception habits etc. It is more difficult to establish the reliability of information on such aspects.

(13) Data Analysis Plan & Methods of statistical analysis

A paragraph indicating how the data is going to be analysed has to be given in the protocol. Due to the easy access to the computers and statistical softwares, data analysis is normally done using the computers. First of all, the data collected/recorded in the proforma/questionnaires should be carefully checked for any mistake in entering the information. Also, care should be taken while entering the data in a specific format consistent with the analysis in the computer. Data checking for consistency, abnormal values and wrong entries should be checked very carefully. For example, the age of the child in a studying a paediatric problem is 6 years and if it is wrongly entered as 60 years and if this is not checked & identified, the analysis will give wrong results. Methods of checking these aspects have to be clearly stated under data analysis plan.

Some investigators simply write that data analysis will be done using standard statistical methods. This is not sufficient. What specific methods are going to be used to achieve the various objectives of the study should be stated here. For example, it may be written as:

1) Percentage prevalence rate of complete blindness will be computed
2) To test the statistical significance of the difference in the prevalence rates between poor and high socio-economic classes chisquared test will be done
3) To test the statistical significance of the difference in age mean visual acuity between children from public schools & Govt. schools, student’s ‘t’ test will be done
4) To study the correlation of eye pressure and age, Pearson’s correlation coefficient will be computed etc. The help of a statistician may be sought for writing these aspects

(14) Consent form

An appropriate consent form is very essential for any research study, especially in experimental studies. The consent form should clearly give all the relevant details indicating the aim of the study, the method and material of intervention, possible side effects of intervention, the structure of intervention material and all other relevant details. This is a legal and ethical requirement in any intervention study. Even in observational studies, consent form is required to indicate that the study subjects do not have any objection in participating in the study by giving the required information and by subjecting themselves for any lab test. The consent form needs to be got approved by the Institutional Ethical Committee

(15) Expected outcome

Expected outcome of the study in terms of the benefits and, both direct and indirect, to the patients / common people, both immediate and in the long run should be given. For example, the results on the magnitude of the problem w.r.t. the factors studied and identification / confirmation of the risk factors for the occurrence of the problem or how the confirmation of the efficacy of a new treatment modality in comparison to the existing standard methods is going to be beneficial to the patients specifically and to the Government, in general w.r.t. the social and economic aspects should be spelt out.

(16) Logistics-Resources and Facilities available and required-Budget details

Detailed yearly budget for various components has to be given with proper justifications. Facilities available in the place of research study, in terms of expertise and infrastructure should be given. Personnel, like research and
other staff required, non-consumable items like equipments & instruments and consumable items like pharmaceutical & chemical material, required with the estimated budget for the same should be clearly stated with full justifications. Budget for printing proformae / Questionnaires, travel and contingencies for stationery, local travel etc. should be given with proper justifications. Depending upon the funding agency certain amount may be included as overhead costs as per rules. Both Yearly and total budget should be given.

(17) Time Schedule

Time plan w.r.t. the different components of the study should be a part of the protocol. This should indicate how the total time period indicated for the study is going to be utilized for the different aspects of the study. This is very important to start the study at the correct time and to complete it within the time period indicated. The time period for the various components should be strictly adhered to so that the study can be completed within the indicated time. For example, if the duration of the study is 2 years, the time plan could be as follows:

(A) --- 3 months

(1) Preparation of Study Tools (Proformae / Questionnaires)
(2) Procurement of Equipments / instruments
(3) Standardization of Study Tools / Equipments / Instruments

(B) Data collection / recording --- 12 months

(C) Data entry in the computer and data editing & cleaning --- 3 months

(D) Statistical analysis of data and interpretation of results --- 3 months

(E) Report writing --- 3 months

(18) References

Books / Publications referred in the protocol should be given in the standard format like Vancour style or any other stipulated style of the funding agency. It would be ideal to include the latest publications / books rather than very old ones.

(19) Annexures

Appendices / Enclosures / Attachments such as Proforma(e) Questionnaires, Consent form Important Documents related to the study, Bio-data of the Investigators and any other relevant papers should be included in the protocol.

(20) Summary

A summary of the protocol highlighting the relevance and importance of the study and indicating the objectives and brief write-up on material & methods (study design, sample size, study population, study tools) and the expected outcome of the study should be given in the protocol.

Books for further reading:

(1) Practical guide for health researchers, WHO Regional publications Eastern Mediterranean Series-30, Mahmoud F Fathalla, WHO, Regional office, Cairo, 2004
(2) Medical writing-a guide to clinicians, educators & researchers, Taylor, Robert B, 2011
(4) Medical Statistics-Principles & Methods, Sundaram KR, Dwivedi SN, Sreenivas V, BI publications, Delhi, 2009
(8) Clinical Epidemiology - The Essentials: Robert W. Fletcher, Suzanne W. Fletcher, Lippincott Williams, 2005
(9) Statistics for Epidemiology: Nicholas P. Jewell; Chapman & Hall (CRC), 2004

Prof Sundaram was previously the Head of Biostatistics at All India Institute of Medical Sciences. Currently he heads the Department of Biostatistics at Amrita Institute of Medical Sciences, Kochi.
Strategies For Pseudoexfoliation And Weak Zonules (Video Assisted Skill Transfer Section)

Please click on the link http://youtu.be/YZakv9KTSBI

The surgical video shows the difficulties and complications involved in the management of pseudoexfoliation and week zonules during phacoemulsification. Please read the article along with the video.

Pseudoexfoliation (PXF) syndrome is an age-related disease that is thought to be a systemic disorder in which hyaline material of an unknown etiology accumulates in the ocular tissue. The ocular manifestations involve all of the anterior segment, including conjunctiva and orbital structures. There is increasing evidence of an etiologic association between PXF and cataract formation. Glaucoma occurs more commonly in eyes with PXF, the most common identifiable cause of open-angle glaucoma. Approximately 20% of open-angle glaucoma cases are associated with PXF, and approximately 30% to 50% of patients with PXF develop glaucoma.

In cataract surgery, PXF is associated with a higher rate of intra- and postoperative complications. Intraoperatively, posterior capsular tear, zonular dialysis, vitreous loss, and dislocation of lens matter occur more frequently in eyes with PXF than in eyes without the condition; postoperatively, complications such as intraocular pressure (IOP) spike, corneal edema, iritis, pigment dispersion, cystoid macular edema, posterior capsular opacification (PCO), anterior capsular fibrosis, and subluxation or dislocation of the IOL-capsular bag complex are more frequent in eyes with PXF than in the general population.

The implementation of modern phaco surgery has reduced complication rates significantly; however, complications still occur as a side effect of PXF syndrome. Hence, a meticulous preoperative work-up is needed to diagnose the condition at an early stage and allow the surgeon to formulate an appropriate surgical strategy to minimize complications and optimize outcomes.

PREOPERATIVE WORK-UP
Preoperative work-up for cataract surgery should achieve the following:

1. Identify the presence of risk factors for intraoperative complications;
2. Identify the presence of associated ocular comorbidities;
3. Facilitate appropriate patient counseling.

Risk factors for intraoperative complications.
Nondilating pupils and zonular instability are significant risk factors for intraoperative and postoperative complications. I routinely perform mydriasis evaluation to ascertain whether a small pupil strategy will be required. A viscoadaptive ophthalmic viscosurgical device (OVD) such as Healon 5 (Abbott Medical Optics Inc., Santa Ana, California) can be used to attempt viscomydriasis; however, I prefer to use iris hooks for nondilating small pupils. Good results may also be obtained using other modalities such as the Malyugin Ring (MicroSurgical Technologies, Redmond, Washington), the Beehler Pupil Dilator (Ambler Surgical, Exton, Pennsylvania), the Morcher Pupil Dilator (Morcher GmbH, Antony, France), and the Perfect Pupil Injectable (Milvella Ltd., Sydney, Australia).

I perform thorough slit-lamp biomicroscopy to detect subtle signs of early PXF and zonular weakness. Pupillary ruff defects or pigment dispersion caution me to look more carefully for the presence of PXF in a given case. In established cases, the condition is easy to diagnose: It appears as a powdery deposit in the anterior segment including on the iris, the pupillary margin, and the surface of the crystalline lens (Figures 1 and 2). Postoperatively, these powdery deposits may also be detected on the IOL surface and the anterior capsule (Figures 3 and 4). In frank cases, the pathognomonic “double bull” appearance on the anterior capsule is difficult to miss.

It is important to remember that the extent of PXF deposits may not correlate with the degree of zonular instability. Minimal iridotomies is a subtle sign of early zonular instability, and an asymmetrical anterior chamber depth is an important indicator of zonular weakness. I look for
asymmetry between the patient's two eyes and also among different quadrants of the same eye. Phacodonesis and frank subluxation (after pupillary dilatation) confirm the presence of zonular weakness. Mild tilt or a slightly eccentric central nucleus picked up in the slit beam may indicate the presence of subluxation in patients with a nondilating pupil.

**Associated ocular comorbidities.**
The presence of comorbidities such as advanced nuclear sclerosis, reduced endothelial count, and open-angle glaucoma compounds the challenges faced by the cataract surgeon. I routinely perform gonioscopy in all patients with PXF to check the status of the anterior chamber angle, as the occurrence of intraoperative complications may require implantation of an anterior chamber IOL.

I advocate early surgery for these patients, most specifically before their nuclear sclerosis has reached an advanced stage. In my experience, the majority of PXF patients present with advanced brunescent or white mature cataracts, and if this is the case I perform B-scan ultrasonography to rule out significant posterior segment pathology.

**Patient counseling.**
I advise patients to consider an early surgery so that the outcomes can be maximized by avoiding to operate on cataracts with harder nuclei. Patients are also made aware of the increased incidence of intraoperative complications in these challenging eyes. Postoperative course is more intense and they are required to report for more frequent postoperative visits than is the routine for standard cataracts. Patients are told that PCO induced drop in vision may occur early necessitating Nd-YAG capsulotomy. I advise all these patients to report for any visual symptoms at the earliest so that subluxation of IOL-Capsular complex can be picked up early and fixed thereby perhaps avoiding a major intervention.

**INTRAOPERATIVE TECHNIQUES**

**Anesthesia.** I prefer topical anesthesia for routine cataract patients, but a complex PXF case with weak zonules, subluxation, small pupil, and advanced nuclear sclerosis merits surgery under peribulbar anesthesia. I always use the soft-shell OVD technique for endothelial protection in these challenging situations.

**Capsulorrhexis.** I routinely stain the anterior capsule with trypan blue (0.06%) dye in eyes with PXF. Because in PXF cataracts are often advanced and provide a poor red reflex, the dye is administered to enhance anterior capsular visibility and facilitate the capsulorrhexis. The PXF material and the central zonular attachments also stand out when stained with trypan blue.

Given that the zonules may be weak, the initial puncture of the capsule is made with a 26-gauge bent needle. In patients with significantly lax zonules, radiating wrinkles appear on the anterior capsule at the time of capsular puncture. A central rhesis with a 5.5-mm diameter is attempted. A capsulorrhexis that is too small may complicate phacoemulsification by limiting access to the nucleus, whereas a rhesis that is too large may make subsequent use of capsular support devices more difficult. However, it is safest to make the rhesis diameter larger if the nuclear sclerosis is greater. A bimanual technique may have to be employed in cases with significant subluxation.

**Hydrodissection.** Properly performed cortical cleaving hydrodissection is crucial. I routinely perform a multiquadrant hydrodissection because it efficiently mobilizes the nucleus and greatly helps minimize zonular stress while the nucleus is rotated during phacoemulsification. I exercise extreme caution in this step when emulsifying brunescent and white cataracts associated with PXF for fear of posterior capsular blow-out. The initial rotation is achieved asatraumatically as possible employing a bimanual technique. Visco dissection with an appropriate OVD may help in capsular-cortical cleavage and facilitate insertion of a capsular tension ring (CTR) should it be required at an early stage of surgery.

**Phacoemulsification.** Current phaco techniques and newer-generation phaco technology using newer power modulation and better fluidics have resulted in considerable improvement of surgical results in eyes with PXF. A correctly performed chopping maneuver minimizes trauma to the zonules and is my technique of choice for nuclear disassembly.

The benefits of a larger rhesis are appreciated at this stage, enhancing access for nucleus manipulation and transmitting less stress to the capsulozonular apparatus. I avoid cracking techniques because these maneuvers may relax the zonules in the axis perpendicular to the cracking (provided there is no imbalance of the cracking forces) but stretch the zonules in the axis of chopping.

All maneuvers should be performed slowly in the center of the pupillary zone, using a low-flow, low-vacuum technique. Anterior chamber depth should be maintained at all stages, with no sudden shallowing or deepening.

**Cortex removal.** Another critical step is cortex removal, during which care should be taken to avoid stressing the zonular apparatus. Tangential stripping and aspiration of the peripheral cortex minimizes zonular stress, and manual aspiration can also be helpful in difficult cases. In some
challenging cases with fragile or lax zonules, I have even waited to remove the cortex after IOL implantation. Properly performed cortical cleaving hydrodissection helps by leaving behind minimal cortex for subsequent removal.

**Stabilizing the capsular bag.** Capsule and iris retractors, CTRs, modified CTRs, and capsular tension segments (CTSs) are helpful adjuncts that come to the rescue when one is confronted with a weak zonular apparatus in these challenging situations. When used appropriately, such devices improve outcomes and avoid complications encountered with capsular bag.

The zonulopathy in PXF is diffuse and progressive. Hence, appropriate modification of surgical steps and utilization of adjunctive devices are called for when dealing with these cases. In PXF cataracts presenting with no clinical manifestation of zonular weakness, in addition to adopting a zonule-friendly cataract surgery, some surgeons have suggested the routine use of CTRs to reduce or delay the incidence of late postoperative complications such as capsular phimosis and dislocation. CTRs may also facilitate scleral fixation of the IOL or capsular bag complex if symptomatic subluxation develops.

I use CTRs only in the presence of clinically manifest zonular weakness. In these cases, they offer two unique advantages. First, the device helps to redistribute the pressure 360º around the capsular fornix, including areas where the zonules are dehiscent. Second, the CTR keeps the capsular bag expanded throughout the procedure, thereby rendering the surgery much safer.

The timing of CTR insertion is controversial. The device can be inserted at any stage of the cataract procedure; however, if insertion is attempted before the nucleus has been removed (especially if the nucleus is hard) the process may be more traumatic to the zonules. Therefore, I place the CTR after emptying the capsular bag.

Use of an injector is a more precise and less traumatic method for CTR insertion than a manual technique. If intraoperative stabilization of the capsular bag is required, I prefer to use dedicated capsular hooks to support the loose capsular bag. The Ahmed CTS (Morcher GmbH) may also be helpful at this stage. At the conclusion of phacoemulsification, I suture a Cionni ring (Morcher GmbH) to the sclera with 9-0 polypropylene to permanently stabilize the bag.

Cortical clean-up can be a struggle if the CTR has been placed at an earlier stage during phaco. The Henderson CTR (Morcher GmbH), with eight equally spaced indentations, has been found to be of great help in these situations.

**IOL choice and lens implantation.** If the capsular bag is stable—with or without a sutured capsular stabilization device—I implant a one-piece hydrophobic acrylic posterior chamber IOL in the bag. I avoid plate-haptic IOLs, because management becomes difficult should postoperative subluxation occur. If the bag is unstable or absent, scleral suturing or gluing-in of a posterior chamber IOL can be considered. A current-generation anterior chamber IOL with a quadriflex design can be yet another option if the angle is open.

**POSTOPERATIVE COMPLICATIONS**

**IOP spike.** Postoperative IOP spike is a concern in patients with PXF. I routinely prescribe an appropriate topical antiglaucoma medication for a few days after surgery. A systemic carbonic anhydrase inhibitor can be used if necessary. I have found that releasing aqueous through a paracentesis is an effective strategy to decompress the eye. The IOP spike may be blunted by careful removal of the lens matter and thorough evacuation of the OVD at the conclusion of surgery.

**Late complications.** These can include changes to the posterior or anterior capsule and spontaneous IOL subluxation. Posterior capsular opacification is more frequent in patients with PXF than in the general population. When it becomes clinically significant, it should be treated with Nd:YAG capsulotomy.

In the anterior segment, postoperative anterior capsular contraction can occur early in eyes with PXF due to an imbalance of forces caused by zonular weakness. Progressive centripetal contraction can result in progressive zonulysis. If unchecked, this may lead to capsule phimosis with or without IOL decentration. The prophylactic measures that I consider for these challenging cases include the following:

- Creating an optimally sized, round, central capsulorrhexis (with a 0.5-mm overlap on the optic edge of the IOL);
- Vacuuming and polishing the undersurface of the anterior capsule prior to lens implantation;
- Employing a zonule-friendly surgical strategy; and
- Using a CTR or modified CTR.

I consider treatment at an early stage the moment I detect the beginning of capsular contraction. I perform Nd:YAG laser relaxing anterior capsulotomy through the annular capsular band at three or four symmetrically placed locations on the capsulorrhexis margin, which greatly reduces the chance for progression to capsular phimosis, IOL tilt, and decentration.

**Spontaneous IOL subluxation.** This late complication associated with PXF is increasingly encountered because of the expanding pseudophakic population. This problem
can arise any time between several months and 16 years postoperatively, with an average of 8.5 years after IOL implantation. Several surgical techniques are reported in the literature to manage this condition, including IOL exchange, IOL repositioning, and suturing the IOL to the sclera or iris. In a symptomatic patient, I prefer to intervene at an early stage to achieve surgical correction with minimum manipulation.

**CONCLUSION**

PXF cataract can pose significant challenges for the cataract surgeon. With a proper preoperative work-up, intraoperative strategy, and postoperative follow-up, the surgeon can optimize outcomes, even in these difficult eyes.


Figure 1. Nondilating pupil due to pseudoexfoliation.

Figure 2. Pseudoexfoliation deposits on the anterior capsule in a mature white cataract before (A) and after (B) staining with trypan blue dye (0.06%).

Figure 3. Pseudoexfoliation deposits on the anterior capsule in a pseudophakic eye.
Figure 4. Pseudoexfoliation deposits on the anterior IOL surface in a pseudophakic eye.

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The angle of the anterior chamber needs to be assessed in all glaucomas. It usually holds the secrets to pathogenesis in that particular case. It may also reveal pathology in other cases in the form of a retained intraocular foreign body, small hyphemas, small hypopyon, etc. The object of this write up is to guide one through the examination procedure.

Due to total internal reflection we are unable to see the angle of the anterior chamber without compensating for the air cornea interphase. This can be achieved by direct gonioprisms used in surgeries (not dealt in detail in this treatise) and by indirect goniolenses that use a mirror to see the angle.

In general we use Goldman two or single mirror lenses as well as Sussman or Zeiss 4 mirror lenses in diagnostic gonioscopy with slit lamp examination.

**The Procedure**

First we need to explain the procedure to the patient. Under topical anaesthetic (Proparacain) both the patient and the surgeon are to be seated comfortably at the slit lamp. For the surgeon’s comfort one has to place the elbow rest in a convenient position prior to inserting the goniolens.

**Placing the contact lens**

The Ziess or Sussman lens requires no coupling fluid. These lenses are placed onto the patient’s eye with the patient looking straight ahead (fig 1). (Great care is taken so as not to put undue pressure on the eye which can cause the angle to open up with indentation, cause corneal folds that obscure visualization of the angle and make the patient uncomfortable due to vagal stimulation.

When using a Goldmann lens we need to fill the contact lens partly with a coupling fluid (one drop of our surgical viscoelastic / surgical methyl cellulose or even a couple of drops of artificial tears will do). I say partly fill because once on the eye there is need of only about 1 - 2 drops. The excess always flows down the patient’s cheek during the procedure making it uncomfortable for the patient. It would also mess up your slit lamp. After instilling the coupling fluid we can ask the patient to look down, retract the upper lid with your nondominant hand and place the goniolens onto the eye using the edge of the goniolens to retract the lower lid. Now one can ask the patient to look forward and move the goniolens with the eye (fig 2). Alternatively we can ask the patient to look up and place the goniolens over the inferior sclera. Now when the patient looks straight we rotate and place the lens onto the eye (fig 3)

**Slit lamp adjustments**

Now we need to adjust the slit lamp. The beam is kept vertical and the illumination housing slant at about 30 degrees. To assess occludability the slit beam is made as thin and short as possible to view structures in the angle (this will avoid light from going into the pupil and constricting it). This position will allow for good visualization of the upper and lower angles.

To visualize the nasal and temporal angles one needs to make a few other adjustments. The illumination and microscope housings are aligned to be coaxial (0-2 deg). The housing is now tilted so that the beam is directed at about 15 degrees.
from below. The slit beam is made horizontal by rotating the lamp housing. This will allow for enough parallax to assess the angle structures without glare from the proximal glass cover of the goniolens. (Fig 4)

Magnification. Use just enough magnification to visualize the angle. Higher magnification makes one lose depth of focus and can be confusing. If a specific feature like new vessels in the angle is to be looked at closely, the change magnification for that instant alone.

Gonioscopy
The questions to be asked in gonioscopy are:
1. Grading
2. Is the angle occludable?
3. How much can it potentially open up to?
4. Is there any other features? (e.g. Secondary glaucoma features)

Anatomical landmarks
For grading we need to be clear about landmarks in the angle. The anteriormost landmark is the Schwalbe's line. This is the edge of the Descemet's membrane and can be identified by following the corneal parallelepiped to its end where all lines coincide (the epithelial, endothelial and iris lines of illumination) (Fig 5)

Schlemm's canal is sometimes seen if blood has refluxed into it as a pinkish line in the middle third of the trab meshwork. This would be the middle third of the trab meshwork or the anteriormost part of the filtering part of trab meshwork. The trabecular meshwork itself has a granular appearance. Pigmentation can vary a lot in normal patients.

Posterior to this one may see a glistening white line – the scleral spur. This is often a broken line than a continuous one. Behind this would be the grayish ciliary body band. The width of this is very variable and needs to be compared to the contra lateral eye in suspected angle recession. Beyond this would be the root of the iris.

Grading
For grading I prefer an anatomical system of grading as developed at RP center by Dr Madanmohan. (Fig 6). This eliminates subjective assessment of the angle entry from interfering with the grading system. This note however about the angle recess should be mentioned as one’s judgment of “whether this patient has an occludable angle or not?” (i.e. is this patient prone for primary angle closure?)

Grading
- Anatomical Grading Preferred
  - Madanmohan's Grading
    0 - No Dipping Of Beam (False Angle)
    1 - Schwalbe's Line Seen
    2 - Anterior 1/3 Of Trab
    3 - Middle 1/3 Of Trab (Schlem's
    4 - Posterior 1/3 Of Trab (filtering part)
    5 - Scleral Spur
    6 - Ciliary Body Band

- Add note on occludability

Peeping into the angle recess
To see into the angle recess one can peep over the hill. This is done when the lens is jutting into the AC obscuring the angle recess. The goniolens is tilted towards the angle in question taking great care not to indent the angle open. (Fig 7).
A case
Now let me take you through gonioscopy of a particular patient (Fig 8). Top left picture is with the slit beam not hitting the pupil and the angle appears totally closed. The corneal parallelepiped has not joined up at schwalbe’s line - grade 0. The beam is now widened and the pupil starts to constrict causing the angle to open up. Top right picture of the same angle shows up the schwalbe’s line and with the beam length also increased the anterior trab meshwork is seen in the bottom left picture. The bottom right picture shows the angle to be fully open on indentation - Grade 6. Thus if we had not gone step by step we could go wrong here. If we had full illumination and a little pressure on the eye, then the angle would have been fully open and the patient labeled as POAG/NTG requiring life long therapy. When we go step by step it turns out to be an appositional PACG requiring a onetime peripheral iridotomy.

Indentation. (How much can this angle potentially open up?)
Indentation with a Sussman lens or Zeiss lens is straightforward pressure on the corneal apex (Fig 9). When the corneal apex is indented, the limbal ring gets stretched. The peripheral cornea is pushed outward and the iris root rotates backwards. The zonules are stretched pushing the lens (lens iris diaphragm) backwards. All these contribute to the angle opening up fully.

The same can be achieved with a Goldman lens. We need to ask the patient to look towards the mirror we are looking at. At the same time we resist this movement with the goniolens and thus use the edge of the lens to indent the angle open. (Fig 10).

This is less efficient when one uses a Goldmann lens than when one uses a Sussman or Zeiss lens. Therefore if there is a doubt confirm with one of these lenses.

The Goldmann 3 mirror lens is too big to do indentation gonioscopy. The lens does not allow one to tilt at all and even peeping into the recess is impossible. Furthermore the mirror angulation is different and hence one would call an angle more closed than it really is when using a three mirror lens. I would strongly advise all to maintain the three mirror lens as a retinal contact lens and invest in a dedicated goniolens for gonioscopy.
Once we have got grading out of the way we look for telltale signs of angle closure – peripheral synechia, coarse pigment deposits etc. Any features of secondary glaucomas, developmental glaucomas are also noted. This entire information is put down in the case record for each quadrant. (Minimum grade and maximum grade of the angle opening, ocludability, secondary features)

Synechiae (Fig 11)
Peripheral anterior synechiae are adhesions between the iris and angle structures or peripheral cornea.

Synechia of an appositional closure tend to be a smooth anterior edged bump with some areas more open on indentation than others. i.e. synechiae have varying height.

Where as for creeping angle closure the synechia seems to have a uniform height and again has a regular anterior edge.

Inflammation leads to patchy synechia. These have an irregular anterior edge. Often they are point synechiae that look triangular (teepees – as they are called in comparison to the red Indian tents). Inflammatory synechiae are more often in the inferior angle whereas angle closure synechiae are more often in the superior angle.

These need to be differentiated from iris processes that are strands of iris and not full thickness areas of iris plastered on to the angle.

Plateau Iris (Fig 12)
The peripheral iris seems to drop off as in a plateau. The central parts of AC are deeper than one expects from the look of the angle. On indentation one can see the sine wave sign. The iris goes back the up over the ciliary processes and back again before coming up along the convexity of the lens.

Pigmentation (Fig 13)
Pigmentation of the trabecular meshwork varies a lot in normals. But it assumes significance in the presence of pseudoexfoliation on the lens surface and features of pigment dispersion syndrome such as Kruckenberg spindle. Dense pigmentation of the posterior trabeculum tends to be significant compared to uniform pigmentation of trabeculum. Coarse clumps of pigment in an ocludable angle may suggest previous apposition even in the absence of synechiae.

Angle recession (Fig 14)
Always compare width of ciliary body band between the 2 eyes. Look for additional features of trauma. Here the ciliary body band is widened. The width often varies a lot in different parts of the same eye in recession. One may note torn iris processes when present. (one half on iris and the other on trab). There may be old blood in form of hemosiderin balls (black in Colour not brown – that would be iris pigment).

Cyclodialysis (Fig 15)
Invariably here there is recession in the angle. One sees sclera through a cleft like opening. This opening widens and narrows when one presses with goniolens slightly (on indentation). An associated feature would be low IOP.

Neovascularisation (Fig 16a and Fig 16b)
New vessels in the angle may precede new vessels on the iris in neovascular glaucoma. These vessels arise from the iris root and brach in an arborising pattern onto the trabecular meshwork surface. These later contract and cause pulled up synechiae with vascular anterior edges.

**Axenfeld anomaly** (Fig 17)
There is ridge like posterior embryotoxon. Iris strands attach to this as bridging synechia (with a gap behind).

**Iridocorneal Endothelial syndromes** (Fig 18)
There is some corneal edema in all 3 forms of ICE. The synechia are broad and have a pulled up appearance due to the contracting membrane.

**Post Trauma progressive inferior corneal edema** (Fig 19)
This is often due to a retained foreign body in the anterior chamber angle.

**References:**

Visual Outcome And Surgical Complications After Phacoemulsification In Fuch's Heterochromic Uveitis

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Abstract

Aim: To evaluate the visual outcomes and surgical complications of Cataract surgery by phacoemulsification and foldable lens implantation in Fuch's heterochromic Uveitis

Material and Methods: 15 patients who presented to Comtrust Eye Hospital during the period from June 2008 to January 2011 and were diagnosed as Fuch's heterochromic uveitis with complicated cataract were evaluated preoperatively, intraoperatively and postoperatively. Their visual outcomes were assessed during follow up.

Results

Age ranged from 31 to 49 yrs. 9 were females and 6 were males. All patients showed heterochromia irides. 6 (40%) patients showed blood in Schlemm's canal during gonioscopy. 1 (6.6%) showed Amsler's sign-filiform haemorrhage during gonioscopy. 6 were done under topical anaesthesia and the rest under peribulbar. 2 (13.3%) showed blood in the anterior chamber after peribulbar block before starting the surgery. 3 (20%) showed hyphema immediately after entering the anterior chamber (Amsler's sign). 6 (40%) developed hyphema on the first postoperative day. None had ongoing hyphema. Deposits on the posterior capsule was seen in 4 (26.6%). 6 (40%) were given postoperative short course systemic steroids. Postoperatively a visual acuity of 6/12 and better was achieved in 13 (86.6%).

Conclusion: Phacoemulsification with foldable IOL is the best option for Fuch's. They need close follow up during immediate postoperative period. Topical anaesthesia is a better option for surgery.

Introduction

Ernst Fuchs in 1906 described low grade chronic anterior uveitis occurring in one eye and characterised by heterochromia iridis in individuals between 20 to 40 years with equal sex preponderance. The classic triad of heterochromia, keratic precipitates and cataract was first described by Kimura and is taken as the diagnostic criteria. Iris stromal atrophy and defective posterior epithelial layer leads to the heterochromia and the moth eaten appearance of the iris. Symptoms of iritis are minimal and they often present with defective vision due to the complicated cataract, glaucoma or floaters due to vitreous opacities or are diagnosed during routine examination. Keratic precipitates are classically round or stellate distributed all over cornea. A flattening of anterior iris architecture and moth eaten appearance is classic. Neovascularisation of iris and angle of the anterior chamber over the trabecular meshwork may be seen and these vessels at the angle may bleed when IOP is suddenly reduced during surgery or paracentesis. Amsler and Verrey in 1946 observed filiform haemorrhages in the angle of the anterior chamber, seconds after paracentesis and Verrey has shown that this blood comes from Schlemm's canal.

The trigger for inflammation is not established, though several postulates including infectious—Toxoplasma, Rubella(7), HSV, and recently Chikungunya(4)—, an immune dysfunction, a sympathetic neurogenic factor—all have been put forth.

90% develop cataract which progresses rapidly. The surgical outcome is good except in situations where glaucoma is associated or vitreous opacities are present. Poor pupillary dilatation and rubeosis can produce surgical problems. Small incision, clear corneal section is preferred to avoid vessels at the angle. Slow decompression of the globe reduces risk of haemorrhage from the angle. An acrylic in the bag placement of lens is preferable.

Aim: To evaluate the visual outcomes and surgical complications of Cataract surgery by phacoemulsification and foldable lens implantation in Fuch's heterochromic Uveitis.

Material and Methods: A prospective study was done in 15 patients who were seen in Comtrust Eye Hospital during the period from June 2008 to January 2011 and were diagnosed as Fuch's heterochromic uveitis with complicated cataract and were referred for surgery. They were evaluated pre-operatively, intraoperatively and postoperatively. All of them had phacoemulsification and 4 were implanted with hydrophobic acrylic IOLs whereas 11 had hydrophilic acrylic...Their visual acuity, intraocular pressure and dilated fundus examination findings were noted. A detailed slit lamp examination was done and gonioscopy with particular attention to the schlemm's canal and any neovascularisation. B-scan was done where there was no fundus view. Blood workup including Mantoux was done in all and Toxo titre was done in 6. The diagnostic criteria were low grade anterior uveitis, absence of acute exacerbations, diffuse fine keratic precipitates, diffuse iris atrophy with loss of iris.
architecture, and absence of posterior synechiae. Their visual outcome were assessed and followed up for 6 months to 3 years.

**Results:**

**Demography**

There were 9 (60%) females and 6 (40%) males.

The mean age group was 40.6 years ranging from 31 to 49 years. All were unilateral. Defective vision was the main complaint in all the patients and 7 (46.6%) complained of floaters as well. Re was involved in 8 cases and LE in 7. None of the patients had positive Manteaux. 1 patient out of the 6 tested showed a high IgG toxo titre.

The other eye of all patients were normal and had 6/6 vision with correction. Slit lamp examination showed fine, round and stellate keratic precipitates in all the patients, which were scattered all over the cornea and changed pattern during the course of days. There was no aqueous flare in any of the case. Vitreous floaters were seen in the anterior vitreous in 7 cases (46.6%). 5 (33.3%) cases presented with total white cataract, 8 (53.3%) as posterior subcapsular and 2 (13%) presented as cortical cataract.

6 (40%) patients showed blood in Schlemm’s canal, out of which one showed a linear haemorrhage during gonioscopy. No neovascularisation was seen. None of the patients had glaucoma.

All had phacoemulsification with foldable lens. 6 were done under topical anaesthesia and 9 were under peribulbar block. 10 had hydrophilic acrylic lens, 4 had hydrophobic acrylic lens and 1 had bifocal acrylic lens.

One patient showed mid-dilated pupil. Intraoperatively, 2 patients showed hyphema after peribulbar block and massage. 3 patients showed blood coming from the angle of the anterior chamber after paracentesis - ie Amsler Verrey sign was seen in 6 cases altogether (40%) of the cases.

Large vitreous floaters were seen in 8 (53%) patients. 4 patients showed white deposits on the capsule. All these patients had large vitreous opacities as well. Postoperatively, on day one, 3 patients showed mild diffuse hyphema and 3 patients showed a streak of blood clot in the anterior chamber which cleared completely by 4 days. None of the patients who were operated under topical anaesthesia, developed hyphema. 7 (46.6%) patients had VA 6/12 or better on the first postoperative day. None of the patients showed iritis postoperatively. One patient who had hyphema, developed mild corneal oedema postoperatively and had raised IOP was given oral acetazolamide and timolol topically, which cleared after one week and the IOP became normal in 2 weeks. 6 patients who had large vitreous floaters were given a short course of oral steroids postoperatively for 10 days. At 6 weeks postoperatively, 13 i.e. (86.6%) had VA 6/12 or better and 10 (66.6%) had VA 6/9 or better.
One patient who had VA 6/24 NIG, had macular oedema which was treated with topical NSAID, Bromfinac and one patient with VA 6/18 had large vitreous opacities obscuring vision. 12 patients were followed up for 1 year and 3 had YAG capsulotomy after 6 months.

**Discussion**

All cases of Fuchs’ uveitis who were operated for cataract were unilateral. There was a predominance of females in this study, though many studies (2, 5, 10) have shown equal sex prevalence. But Gordana Zlatanovicia et al. (3) showed a female preponderance. Vitreous floaters and opacities were seen in 7- ie47% cases. Only 1 patient showed visual disturbance postoperatively due to the vitreous opacity. Vitreous opacities have been reported to be a major factor producing visual deterioration after surgery necessitating vitrectomy (9). In our series of patients 6-(40%) showed Amsler’s sign. One during gonioscopy, 2 after peribulbar block and 3 after paracentesis. I.S. Begg Sheffield (6) and B. Michael (5) has shown the constant occurrence of Amsler’s sign in these cases though many other studies have not. D. R. Sherwood (13) reported 100% occurrence of bleeding into the anterior chamber during paracentesis. Iritis was not seen as a significant postoperative finding in this study. Iritis from 15 to 20% has been reported (11). No difference in postoperative complications or visual outcome was found in the different type of lens used. Glaucoma has been found to complicate the condition from 6-30% of cases (1, 8, 10, 12, 14) but in our study none of them had glaucoma but for a transient rise in IOP during immediate postoperative period in 1 case which was controlled by medication and recovered after 2 weeks. We had 1 case of macular oedema producing defective vision. Many report as having no incidence of macular oedema (12).

**Conclusion**

Cataract surgery by phacoemulsification and foldable IOL gives good results in Fuchs heterochromic uveitis. Topical anaesthesia is better to avoid hyphema. Slow decompression during paracentesis is less traumatic to the fragile vessels to prevent hyphema. Close follow up during the postoperative period is necessary to look for ongoing hyphema or glaucoma.

**Reference**

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**After her studies at Calicut Medical Collage and in UK, presently she is working as senior consultant at Comtrust Eye Hospital, Calicut**
Purpose: To study the role of non invasive imaging modalities like autofluorescence imaging and spectral OCT to identify the leakage site in patients with CSCR and study the feasibility of avoiding FFA.

Methods: Retrospective analysis of 117 eyes of 99 patients seen between February 2009 and February 2011 with acute CSCR were included. All these patients had undergone autofluorescence imaging (zeiss filter), spectral OCT and FFA imaging. FFA images were superimposed on the autofluorescence images and the site of leak analysed. Similarly the OCT characteristics corresponding to the site of leak was also analysed.

Results: Hypoautofluorescence was found in 105 eyes (89.7%) corresponding to the leakage points on FFA. Hypoautofluorescence corresponding to the areas of subretinal fluid accumulation was seen in majority of the eyes. On the OCT, RPE abnormalities in 92 eyes (78.6%) corresponding to the site of leak was seen- 50.4% with PED and 28.2% with a bumpy RPE site. Subretinal fibrin seen as reflective deposits in the subretinal space was seen in 73 eyes (62.4%) and sagging/dipping of the posterior layer of the neurosensory retina above the leakage sites were seen in 42 eyes (35.9%). An RPE defect within the PED and intraretinal fluid was observed in 1 eye (0.9%).

Conclusion: Noninvasive imaging tools like AF imaging and spectral OCT can be used as an alternative to FFA to identify the leakage site in acute CSR.

INTRODUCTION

Central serous chorioretinopathy (CSCR) is a common cause of visual deficit especially in the younger age group that we see commonly in our day to day practice. Eyes with acute CSCR have focal leakage at the level of the retinal pigment epithelium (RPE) seen on fluorescein angiography. Evaluations using indocyanine green angiography in eyes with CSCR have shown multifocal islands of inner choroidal staining and choroidal vessel hyperfluorescence. It is now believed that these exudative changes within the inner choroid to be the primary event in the disease. The subsequent changes at the RPE allow the fluid to enter the subretinal space, and those changes are thought to be reversible because spontaneous resolution of the subretinal fluid (SRF) is not uncommon. However many patients require laser treatment or photodynamic therapy to help resolution of the SRF early. Until recently fundus fluorescein angiography had been the only available investigation to study the RPE. Angiography is an invasive test with a risk of adverse events even though small. With the advent of non invasive tests like optical coherence tomography and autofluorescence imaging detailed study of the outer retina and RPE is now possible. Morphologic changes in eyes with CSC have been reported using optical coherence tomography (OCT). This imaging technology records the various features of CSCR, including subretinal fluid (SRF), fibrinous exudation, pigment epithelial detachment (PED) and cystic changes within the retina. RPE changes corresponding precisely to the leakage points on FFA have been evaluated on OCT and minute pigment epithelial detachments (PEDs)/RPE protrusions have been observed. Also recent enhanced depth imaging optical coherence tomographic evaluation of the choroids have showed an abnormally thick choroid in eyes of CSCR, even in unaffected fellow eyes, which is consistent with the choroidal vascular hyper permeability theory. Autofluorescence photography provides functional images of the fundus by employing the stimulated emission of light from naturally occurring fluorophores, the most significant being lipofuscin. Because accumulation of lipofuscin occurs in retinal pigment epithelial cells because of their unique metabolic role9 autofluorescence imaging may provide clues to the pathobiology of central serous chorioretinopathy especially with regard to the RPE. Various studies have reported the findings of autofluorescence imaging in eyes with acute and chronic CSCR.

In the current study, we examined the OCT images and fundus autofluorescence images (FAF) from patients with CSCR where FFA was done and tried to correlate the findings corresponding to the sites of leakage. This study is a retrospective study of 117 eyes of 99 patients diagnosed to have CSCR between February 2009 and February 2011 who have undergone FFA, OCT and Autofluorescence imaging as part of evaluation for the disease. A diagnosis of CSCR was made based on the presence of a serous detachment of the neurosensory retina, focal dye leakage on FA, and the duration of recent subjective symptoms within 3 months. Eyes with recent CSCR with definite ink blot or smoke stack leaks on the angiography were included in
the study. Exclusion criteria included patients with chronic disease, those with sick RPE disease and in those in whom autofluorescence imaging was unclear or poor quality. Eyes with other macular abnormalities such as Age related macular degeneration (ARMD), polypoidal choroidal vasculopathy (PCV) and other causes of maculopathy were also excluded from the study.

All these patients had fundus examination, measurement of the best-corrected visual acuity (BCVA), and spectral domain OCT imaging performed at baseline and at every subsequent visit whenever possible. FFA was done usually at 4 weeks of poor resolution or earlier in special situations. Poor resolution was defined as significant persistant fluid as defined by the surgeon at 4 weeks after the onset of the disease. FFA was done earlier if the presenting visual acuity was poor, in patients with recurrent disease and in those who were not prepared for a conservative waiting period due to their occupational demands. Indocyanine green angiography was performed whenever necessary especially in older patients suspected to have PCV.

OCT was done using the ZEISS cirrus system on all patients. Both 5 line raster and 512x200 cube data were taken and analysed. After obtaining the FFA and identifying the site of leakage the corresponding site on the OCT SLO fundus image selected with the tracker and the corresponding OCT image which comes on display was analysed. This was possible with the cube data. The higher resolution raster line scans were also then studied corresponding to these sites.

We performed FAF photography with a modified filter provided by ZEISS (excitation light with bandwidth of 535–585 nm and a matched barrier filter having a bandwidth of 605–715 nm). FAF images were enhanced with increasing contrast and then analysed. The fundus photograph/angiography were loaded beside the FAF photograph and the analysis done.

RESULTS
117 eyes of 99 patients were part of this analysis. The mean age of the patients was 34.7 years (range, 29–45). At presentation the duration of symptoms ranged from 1 to 86 days (mean, 16.0). In 80 patients the disease onset was for the first time atleast symptomatically, in 19 patients it was a recurrent disease in the same eye, and 3 patients had CSCR history in the fellow eye. Out of these 22 patients with known past history of CSCR, 10 patients had undergone laser photocoagulation for persistant leakage. The mean BCVA at baseline was 0.84 (range, 0.2–1.5). 106 eyes had single point leakage while 11 eyes showed more than 1 point leakage. 91 eyes had an inkblot leakage pattern, and 26 eyes showed a smokestack leakage pattern on FFA. FFA of the other eye also revealed RPE window defects in 43 eyes and leakage points in 18 eyes many of whom were asymptomatic.

On SD OCT examination, a detachment of the neurosensory retina was confirmed in all patients. The OCT feature observed at the site of leak is tabulated in Table.1. An RPE defect within the PED and intraretinal fluid was observed in 1 eye (0.9%). Thus an RPE abnormality- focal RPE thickening (Fig; 1)or PED (Fig; 2) was seen corresponding to the site of leak was seen in 79% of eyes. In the other eyes there was evidence of fibrin leading to the site of leak with or without the corresponding site of the neurosensory layer showing sagging/ dipping (Fig;3,4). 7 eyes (5.9%) had no abnormality detected corresponding to the site of leak on the OCT. Thus majority of the eyes (100/117- 85.5%) had some abnormality or the other at the site of leakage on the OCT.

<table>
<thead>
<tr>
<th>OCT Feature</th>
<th>No of eyes (%)</th>
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<tbody>
<tr>
<td>Pigment epithelial detachment (PED)</td>
<td>59 (50.4%)</td>
</tr>
<tr>
<td>Bumpy, irregularity of RPE</td>
<td>33 (28.2%)</td>
</tr>
<tr>
<td>Subretinal fibrin at the site of leak</td>
<td>73 (62.4%)</td>
</tr>
<tr>
<td>Sagging/dipping of posterior neurosensory layer</td>
<td>42 (35.9%)</td>
</tr>
<tr>
<td>No OCT abnormality at the site of leak</td>
<td>7 (5.9%)</td>
</tr>
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TABLE 1
Fig 1: OCT showing focal RPE thickening

Fig 2: OCT showing PED at the site of leak

Fig 3: OCT showing fibrin at site of leak

Fig 4: OCT showing sagging of the neurosensory retina at site of leak with fibrin.

Fig 5: FAF showing hypofluorescence (arrow) corresponding to the site of leak on FFA

Fig 6: FAF finding corresponding to the site of leak. Eyes (%)
On the FAF photograph hypoautofluorescence was seen in 105 eyes (89.7%) corresponding to the site of leakage on FFA (Fig.5,6). Granular and confluent areas of hypoautofluorescence was seen in these eyes. The area of detachment revealed hypoautofluorescence on FAF images in 73 eyes (62.4%).

96 of the 100 eyes with OCT features corresponding to the site of leakage also showed hypoautofluorescence on FAF images. All the 92 eyes with RPE abnormalities showed a change in autofluorescence on FAF images. 4 eyes did not have any change in autofluorescence on FAF images but showed evidence of fibrin leading to the site of leak with or without the corresponding site of the neurosensory layer showing sagging/ dipping on the OCT. Out of the rest 7 eyes which had normal OCT, 3 eyes also showed hypoautofluorescence on FAF imaging at the site of leakage. The remaining 4 eyes (3.4% of the whole series) showed no change in the OCT or in FAF images corresponding to the site of leak. Thus 113 out of the 117 (96.6%) eyes in this series demonstrated either an abnormality in the OCT or on FAF imaging corresponding to the site of leakage.

DISCUSSION

The primary pathology of acute CSCR is thought to begin with disruption of choroidal circulation. The RPE then decompensates and allows exudation from the choroidal vasculature to pass into the subretinal space.1,2,3,4 These hypotheses are based on FFA and indocyanine green angiography findings, and precise morphologic correlations have not been observed. The development of OCT has provided a better understanding of the mechanism in CSCR, especially the abnormalities in RPE layer.5,6 Reports on 3D OCT images have revealed certain specific RPE abnormalities corresponding to the leakage points on FA.7 These RPE abnormalities were within areas of choroidal vascular hyperpermeability.10

In our study 50% eyes had PED’s, 28.8% eyes had irregularity of the RPE, 62.9% eyes revealed increased reflectivity corresponding to subretinal fibrin at the site, 36 % eyes showed sagging/ dipping of the posterior surface of the neurosensory layer corresponding to the site of leakage on FFA. Similar findings have been reported by other authors. Fujimoto et al11 had reported that among 23 leakage sites in 21 eyes, FD OCT showed RPE abnormalities - 61% with PED and 35% with a protruding or irregular RPE layer. Fibrinous exudates in the subretinal space and sagging/dipping of the posterior layer of the neurosensory retina above the leakage sites were seen in 52% and 43% respectively. Hirami et al10 reported that of the 20 eyes studied, a leaking point was located within PEDs in 25% and was consistent with the bulge of RPE in 45%. 91% of eyes with PED showed PED within the areas of choroidal vascular hyperpermeability on ICG. 89% of eyes with a bulge of RPE showed the bulge within areas of choroidal vascular hyperpermeability on ICG. Hussain et al12 reported that 60% of eyes in their series showed a characteristic dipping pattern of neurosensory retina with intervening hyper-reflective echoes suggestive of fibrin over the leakage site. All these eyes had ink-blot leak on FFA. Shukla et al13 had also reported that presence of subretinal reflective fibrin on the OCT could alter the angiographic pattern of leakage often presenting with atypical leakage patterns.

Even though gross RPE abnormalities corresponding to the site of leakage as discussed above may be seen , the initial point of leakage on FA is smaller than a PED or RPE protrusion as seen on OCT. Therefore, there might be a defect in the RPE layer that allows passage of fluid from the sub-RPE to the subretinal area. The newer generation SD/FD OCT is superior to conventional OCT in picking up these defects. Fujimoto et al11 observed RPE abnormalities in 95% of eyes with acute CSC and clearly visualized a minute defect of the RPE within the PED, which seemed to correspond precisely to the leakage point on FA in 24% eyes. In our study an RPE defect within the PED and intraretinal fluid was observed only in 1 eye (0.9%). Gupta et al14 also had reported that 54.5% of eyes in their series showed PED’s with a disruption/breach in the RPE on transverse C-scan and on OCT fit C-scan and called them microrips of PED all of which showed spontaneous closure with resolution of subretinal fluid.

The absence of RPE at the leakage point is supported by recent findings of fundus autofluorescence. In acute CSC, focal areas of hypoautofluorescence corresponding to the site of the focal RPE leak were observed, and it is speculated that the origin of the hypoautofluorescence may be blowout of the RPE at or near the junction of the attached and detached RPE.15 However, not all eyes with CSC had hypoautofluorescence at the leakage site.16 In acute CSC, decreased AF is presumably due to a blockage caused by oedema, whereas in chronic-recurrent forms, irregular and increased AF is observed, possibly reflecting reactive RPE changes secondary to RPE defects and neurosensory detachment. FAF might be an interesting non-invasive tool for monitoring RPE changes in CSCR and for performing differential diagnosis.17

In our study hypoautofluorescence at the site of angiographic leakage was seen in 90% eyes and hypoautofluorescence corresponding to the subretinal fluid in 62%. Eandi et al15 had reported that all nine eyes in their series demonstrated hypo-autofluorescence corresponding precisely to the site of the focal RPE leak seen on FA. Framme et al17 had observed that in 36 patients with acute CSCR (<6 wks) a significantly decreased AF at the leakage
point was seen in 72% and decreased AF in the area of neurosensory detachment was seen in 77%. In chronic-recurrent CSC as determined by a decrease in VA for longer than 6 weeks and mottled hyperfluorescent appearance in angiography, decreased or mottled AF was observed at the leakage point itself in 76%, whereas significantly increased AF was seen in the area of residual neurosensory retinal detachment in 85%. Dinc et al18 had reported that hypoautofluorescence was found in 80% and 88% of eyes in the acute and chronic CSCR groups respectively, corresponding to the leakage points depicted by fluorescein angiography. Hypoautofluorescence corresponding to the areas of subretinal fluid accumulation was seen in 92% and 82% of the acute and chronic CSCR groups respectively. In 12% eyes with chronic CSR, hyperautofluorescent changes were noted at the previous leakage points. Ayata et al19 reported that focally decreased AF at the leakage site was seen in 77% eyes in SW-AF and 100% eyes in NIR-AF imaging in eyes with acute CSC.

Spaide et al9 had reported that autofluorescence imaging of the posterior pole showed several interrelated findings that were predictive of visual acuity. He classified hypoautofluorescence, a finding indicative of RPE atrophy into 2 types- granular and confluent types. Confluent hypoautofluorescence showed a slightly stronger correlation with visual acuity than did granular hypoautofluorescence, implying that any loss of the RPE centrally is an important factor correlated with visual acuity. However, hyperautofluorescence, which has been suggested to be the result of accumulations of unphagocyted photoreceptor outer segments did not seem to affect visual acuity. Imamura et al20 studied the pattern and frequency of FAF abnormalities and their correlations with corrected visual acuity and found that confluent hypoautofluorescence of the macula, granular hypoautofluorescence of the macula, and increasing age all were independent predictors of decreased visual acuity.

113 out of the 117 (97%) eyes in this series demonstrated either an abnormality in the OCT or on FAF imaging corresponding to the site of leakage. However as the remaining 4 eyes (3%) of the eyes did not have any OCT or FAF characteristic corresponding to the site of leak on FFA, these noninvasive tests could miss the site of leak in some eyes. CSCR being a relatively benign disease with a fairly good spontaneous resolution missing the site of leakage in these small percentage of eyes will not do any harm to these patients. Hence these noninvasive modalities may help in detecting the site of leak in majority of the eyes and can therefore replace FFA. Another consideration to be kept in mind is the fact that FFA can detect the pinpoint site of leak origin while the above noninvasive tests may not be able to pinpoint the site of leak atleast in some cases. Though traditionally it is these point of leakages that is treated with laser photocoagulation, it is now believed that treating areas of choroidal hyperpermeability on ICG (which are larger areas) is a better way to treat these pathologies. Also many of these eyes may have similar OCT characteristics in areas other than the site of active leak like the presence of PED or irregularity of the RPE etc. FAF images may be more consistent in this situation but again in FAF interpretation the images analysed were modified manually by increasing the contrast in our study which could alter sometimes the actual autofluorescence obtained and affect the resolution. FAF images with the Hiedelberg system may have better resolution than the Zeiss filters and may overcome some of the above problems mentioned. Correlating the leaking points to the OCT and FAF images again may not be a point to point representation in the manual method that we have adopted. Such point to point representation and correlation is possible with the Hiedelberg system where all these images namely FFA, OCT and FAF images can be compared and analysed simultaneously in the same screen. In spite of these possible drawbacks this study has demonstrated a correlation between OCT and FAF findings with FFA leakage in eyes with CSCR and explored the feasibility of replacing an invasive procedure like FFA with noninvasive investigations like OCT and FAF.

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ABSTRACT

Purpose
To identify, categorize and analyze the Optical coherence tomography patterns of Diabetic Macular Edema

Methods
In this observational study, 43 eyes of 25 patients with Diabetic Macular Edema (DME) were evaluated. DME is defined as the retinal thickening due to fluid leakage and pooling in the macular area. Macular oedema due to other ocular illness was excluded. All patients underwent visual acuity estimation by Snellen's visual acuity chart, dilated slit lamp Biomicroscopic examination, Fundus Fluorescein Angiography (FFA) and Optical Coherence Tomography (OCT) by the same examiner. OCT patterns were categorized under 7 headings. Central foveal thickness was also measured by OCT and macular oedema classified into mild (201µm-300µm), moderate (301µm-400µm) and severe (≥400µm).

Results
Of the total 25 patients in the age group 35-75 years (Mean age 54.08), males predominated in this study (Males-75%, Females-25%). OCT examination revealed that, 30% had Cystoid macular edema and 26% had Sponge-like retinal thickness. Mixed cystoid and spongeiform pattern was observed in 28%, Epiretinal membrane (ERM) in 9%, Plaque of hard exudates in 7%, Serous retinal detachment in 9%, and Vitreo-macular traction in 5%. 32% were with mild macular edema, 21% moderate and 35% severe forms.

Conclusion
Cystoid macular oedema was the predominant form of DME according to this study. Both eyes of a same patient can present with different DME patterns. Subfoveal serous detachment was always seen along with cystoid macular oedema. Spongeiform thickening and sub-foveal serous detachment show better responsiveness to laser treatment.

INTRODUCTION
Diabetic Macular Oedema (DME), a microvascular complication which is caused by the breakdown of the blood-retinal barrier, promotes neuroglial dysfunction and concomitant visual disturbance.1 It is the commonest cause of visual loss in patients with non-proliferative diabetic retinopathy and a common cause of visual loss in proliferative diabetic retinopathy.

Diabetic macular oedema is diagnosed stereoscopically as retinal thickening in the macula using slit lamp biomicroscopy. The ETDRS defined DME as retinal thickening or presence of hard exudates within 1 DD of the centre of the macula. To characterize the severity of macular oedema, and for treatment guidelines the term Clinically Significant Macular Oedema (CSME) is used. Macular oedema is clinically significant, if one of the following conditions is present: 1. Retinal thickening at or within 500µ of the centre of the macula. 2. Hard exudates at or within 500µ of the centre of the macula if associated with thickening of retina. 3. A zone or zones of retinal thickening one disc area or larger, any part of which is within one disc diameter of the centre of the macula.2

Diabetic macular oedema tends to be a chronic disease. Although spontaneous recovery is not uncommon, 24% of eyes with CSME and 33% of eyes with centre involving CSME will have a moderate visual loss (15 or more letters on the ETDRS chart) within 3 years if untreated.3

The incidence of DME over a 10-year period was 20.1% among patients diagnosed before age 30 years (younger onset) and 39.3% among patients diagnosed after 30 years.4 As the severity of overall retinopathy increases, the proportion of eyes with macular edema also increases. 3% in eyes with mild non-proliferative diabetic retinopathy (NPDR), 38% with moderate-severe NPDR and 71% with proliferative diabetic retinopathy (PDR) develop DME.5

Optical coherence tomography (OCT) is a fast and non-invasive tool for examining the retina in cross sectional images that correlate reasonably with the retinal histology. It is not only helpful in detecting DME early, but has the added advantage of being able to reveal not only the presence of cystoid macular oedema, but subfoveal serous retinal detachment, vitreo-macular traction or an Epiretinal membrane which cannot be detected in FFA. Moreover, the macular thickness map gives us a very accurate idea of central retinal thickness and can quantify the degree of improvement or worsening following therapy.
AIM OF STUDY
The aim of the study was to identify, categorize, and analyze the OCT patterns of Diabetic Macular Oedema.

MATERIAL AND METHODS
This was an observational study done between October 2010 and March 2011 in patients who attended the retina clinic of Govt. Medical College, Thrissur. 43 eyes of 25 patients with Diabetic Macular Oedema were evaluated. The study group included both insulin dependent and non-insulin dependent proliferative diabetic retinopathy and non-proliferative diabetic retinopathy between the ages of 35-75 years. The study population had varied glycemic levels and HbA1c evaluation was not done.

None of these patients in our study had undergone previous focal laser or pan-retinal photocoagulation. Other exclusion criteria were dense cataract, macular oedema owing to other ocular illness and advanced diabetic retinopathy.

Some of the patients had associated other systemic illness like hypertension, nephropathy and hyperlipedemia and were on treatment.

All these patients underwent visual acuity estimation by Snellen’s visual acuity chart, and dilated slit lamp biomicroscopic examination. Fundus photographs were taken and FFA and spectral OCT done for them on the same day, by the same examiner. OCT was done in all eyes, a line scan program was chosen and the image processed and analyzed. Based on the OCT findings, we classified DME into 7 groups. 1. Macular thickening with Cystoid features 2. Macular thickening with Spongy oedema 3. Macular thickening with Mixed Spongy and Cystoid features 4. Macular thickening with Epiretinal membrane 5. Macular thickening with Plaque of hard exudates 6. Macular thickening with Serous Retinal detachment and 7. Macular thickening with Vitreo-Macular traction.

Central macular thickness was measured in line scan in all possible cases and thickness mapping done. Macular oedema was categorized into mild (with a thickness of 201-300µ), moderate (301-400µ) and severe (≥400µ).

RESULTS
Of the 25 patients we analyzed, there were 3 patients in the age group 30-39 years (12%), 2 (8%) in 40-49 yrs age group, 11 (44%) in 50-59 years age group, 8(32%) in 60-69 age groups and 1(4%) in 70-79 age group. Males predominated with M: F of 2.6:1. 67.3% had NPDR and 32.7%PDR. Mean diabetic age was 14.08 years.

Biomicroscopically all these patients had Diabetic macular edema, 11% with DME associated with cystoids macular oedema (CME), and 2% had DME with vitreo-macular traction (VMT). No patients had Epiretinal membrane (ERM) or Serous Macular Detachment with SubRetinal Fluid (SRF) clinically.

In OCT, eyes with spongy edema showed diffuse thickening of macula. It mostly involves the outer retinal layers, while the internal layers maintain their normal reflectivity. Cross sectional scans show swelling of the retina giving it a spongy appearance with increase retinal thickness.
Eyes with CME showed large cystic spaces in the foveolar and parafoveal region. It involves various depth of retina and has intervening septa in between.

Serous macular detachment is seen as a hypo reflective area between neurosensory retina and RPE.

Vitreo-macular traction was seen as hyper-reflective band in the vitreous, which was adherent to the fovea, either centrally or paracentrally causing traction and pulling up the macula.

ERM was identified as a hyper reflective thickening at the level of ILM, causing distortion and flattening of the foveal surface.

<table>
<thead>
<tr>
<th>Type of DME</th>
<th>Biomicroscopy</th>
<th>OCT</th>
</tr>
</thead>
<tbody>
<tr>
<td>CME</td>
<td>11%</td>
<td>30%</td>
</tr>
<tr>
<td>SRF</td>
<td>nil</td>
<td>9%</td>
</tr>
<tr>
<td>VMT</td>
<td>2%</td>
<td>5%</td>
</tr>
<tr>
<td>ERM</td>
<td>nil</td>
<td>9%</td>
</tr>
</tbody>
</table>

**Table 1: OCT types in DME**

<table>
<thead>
<tr>
<th>Type of DME in OCT</th>
<th>% manifestation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spongy</td>
<td>26</td>
</tr>
<tr>
<td>Cystoid</td>
<td>30</td>
</tr>
<tr>
<td>Mixed</td>
<td>28</td>
</tr>
<tr>
<td>ERM</td>
<td>9</td>
</tr>
<tr>
<td>SRF</td>
<td>9</td>
</tr>
<tr>
<td>VMT</td>
<td>5</td>
</tr>
<tr>
<td>Plaques of hard exudates</td>
<td>7</td>
</tr>
</tbody>
</table>

**Table 2: Comparison of DME characteristics identified by biomicroscopy and OCT**
Hard exudates are seen as hyper reflective intraretinal plaque which cast a black shadow due to blocking of the light.

According to OCT picture, 26% of our study group had macular thickening with Spongy oedema, 30% with Cystoid changes, 28% with Mixture of spongy and cystoid oedema, 9% with ERM, 9% with Serous retinal detachment with Subretinal fluid ,5% with Vitreo-macular traction and 7% with Plaques of hard exudates

Measurement of macular thickness revealed, 33% of eyes with mild macular oedema, 21% moderate oedema and 35% with severe diabetic macular oedema.

DISCUSSION

Optical Coherence Tomography is a fast and non invasive tool for examining the retina in cross sectional images that correlates reasonably with the retinal histology. Till recently slit lamp biomicroscopy and FFA were the tools for the diagnosis and management of DME. It is true that they are highly sensitive for the qualitative detection of DME. OCT enables us to detect and understand the accurate subclinical retinal changes associated with DME that may not be detectable even in FFA. Yang et al have suggested that OCT may be more sensitive than clinical examination in assessing DME and is a better tool for documenting changes in macular thickening. In his series, OCT identified spongy retinal thickness was seen in 58% of eyes.6 Otani et al found spongy retinal thickness in 88%, CME in 47%, SRF in 15% of eyes with CSME. Kim et al found spongy retinal swelling in 97%, CME in 55%, SRF in 7%, VMT in 13% of eyes with DME.7 Ozdek et al had reported spongy swelling in 66%, CME in 16%, SRF in 10% of eyes with DME. In our series, cystoid macular was the common form of presentation.8 Our study revealed that 26% had macular thickening with spongy edema, 30% with cystoid changes, 28% with mixture of spongy and cystoid edema, 9% ERM, 9% with serous retinal detachment, 5% with vitreo-macular traction and 12% with plaques of hard exudates. The higher incidence of spongy form of macular edema in our series could be due the fact that the section of diabetic population presenting to our retina clinic is with longer diabetic age and thus their diabetic macular edema a long standing one. CME type represents a chronologically later stage of DME. Further this was a smaller group and thus the fact require confirmation by further study and follow up involving larger number of diabetic macular edema population.

In our study, 30% of the eyes had CME on OCT, compared to 11% detected by biomicroscopy. Ozdek et al also found that 40% of CME detected on OCT were not detected by biomicroscopy and 63% were not detected even by FFA. Thus OCT tends to be a better diagnostic tool in detecting CME than biomicroscopy or FFA.

In our study, 9% of eyes had SRF with subfoveal retinal detachment, which could not be detected by biomicroscopy. Most series have found SRF in 8-12% of eyes with DME.

According to our study, 5% had VMT as per OCT and 2% in biomicroscopy. VMT has been reported by various authors.
between 10-60% of eyes with DME.

Another important finding of our study was both eyes of a same patient can present with different DME patterns

Spongiform thickening and subfoveal serous detachment which may be chronologically earlier than the CME type are the ones which show better responsiveness to conventional laser treatment. The newer modalities like intravitreal triamcinalone, posterior subtenon’s injection of triamcinalone, intravitreal anti-vascular endothelial growth factor are the other options to the laser resistant cases of DME. VMT and ERM require surgical intervention.

CONCLUSION
Diabetic macular edema is a major cause of visual disability in diabetic patients. DME may be more easily and accurately diagnosed in an early stage with OCT compared to clinical methods and other diagnostic modalities. Being non-invasive, its acceptance as a follow up imaging modality to monitor the course of DME and response to therapy is high. It helps to selectively identify cases like VMT and ERM which needs surgical intervention.

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Dr. Mallika Harikrishnan, MS, DO, currently working as Asst. Professor in Dept. of Ophthalmology
Govt. Medical College, Thrissur
We report results of a case series of preterm, extremely low-birth-weight infants, with zone-1, stage 3+ AP-ROP, treated successfully with intravitreal ranibizumab injection (LUCENTIS®; Novartis Inc.). 20 consecutive eyes of 10 preterm babies, 5 boys and 5 girls, with gestational age range from 27 to 32 weeks and gestation weight in range of 940 gms to 1200 gms, presented with zone-1, stage-3 ROP. They were treated with intravitreal ranibizumab injections at a dose of 0.3mg (60% of the normal adult dose) under sterile conditions given through the nasal pars plana of each eye. Improvement was seen in all eyes within 48 hours after the procedure and within next 1 week following the injections, the AP-ROP disappeared. After a period of 4-12 weeks, laser photocoagulation was performed in the peripheral retina. No adverse effects related to intravitreal ranibizumab were observed in any eye.

Aggressive posterior retinopathy of prematurity successfully treated with intravitreal ranibizumab and laser photocoagulation

Introduction:
Retinopathy of prematurity (ROP) is a disease that affects immature vasculature in the eyes of premature babies and is inversely related to gestation and birth weight. ROP has been divided into five stages. Stage-1 and 2 customarily get better on their own. However some eyes go on to Stage 3 ROP, which exists when disturbing new blood vessels grow out from the ridge in the retina toward the center of the eye, since premature baby has not had the time while in the womb to allow the blood vessels within the retina to grow all the way from the optic nerve in the back of the eye to the front of the eye. Aggressive posterior ROP (AP-ROP), sometimes referred to as Rush disease, is a rapidly progressive form of ROP. It is observed most commonly in zone I, but may also occur in posterior zone II. If untreated, it usually progresses to stage 5 ROP. The characteristic features of AP-ROP are its posterior location, prominence of plus disease.

ROP is emerging as a major cause of blindness, in developing countries. The incidence of ROP varies between 16-48% and 27-35% in infant weighing less than 1000g and 1500g respectively at birth.2 The incidence of ROP in neonatal intensive care units (NICUs) or referral to tertiary care hospital in India ranged from approximately 21 to 40%.3 The reasons for this high prevalence rate can be higher rate of premature birth, lack of resources resulting in compromised neonatal care, leading to higher rates of severe ROP not only in extremely premature infants but also in larger, more mature infants.

Vascular endothelial growth factor (VEGF) is an important oxygen-regulated factor and its overexpression plays an important role in pathogenesis of ROP.4 The development of ROP is largely dependent on VEGF. When an infant is born prematurely the relatively hyperoxic environment the baby is introduced to shuts down the production of VEGF. Retinal maturation is delayed. Subsequently, at a time when intraocular VEGF levels would normally be declining late in the third trimester of pregnancy, abnormally high levels of VEGF are seen due to large areas of avascular retina and associated tissue hypoxia. The availability of FDA-approved drugs for anti-VEGF treatment renders it possible to treat such eyes off-label. The rationale for this treatment approach is that VEGF promotes retinal vascularization. Available drugs include pegaptanib sodium (Macugen®) for partial blockage of VEGF-A, or drugs such as ranibizumab (LUCENTIS®; Novartis inc.) and bevacizumab (Avastin®), which cause complete blockage of VEGF-A.

The studies, Pan-VEGF Blockade for the Treatment of Retinopathy of Prematurity (BLOCK-ROP) and Bevacizumab Eliminates the Angiogenic Threat of Retinopathy of Prematurity (BEAT-ROP), have shown promise to potentially change the standard for treatment of ROP.

The purpose of BEAT-ROP was to determine whether injections into the vitreous of an anti-VEGF will reduce the incidence of blindness by suppressing the neovascular phase of ROP, compared to a control group receiving conventional laser therapy and to determine the safety and efficacy of intravitreal bevacizumab in the treatment of ROP. The result from BEAT-ROP showed significant efficacy of Intravitreal bevacizumab in treatment for zone-1 disease (P=0.003), in infants with stage 3+ retinopathy in comparisons with conventional laser therapy.5 Also, bevacizumab allowed continued vessel growth into the peripheral retina, whereas conventional laser therapy permanently destroyed vessels in the peripheral retina. Although question of right dose and safety of using intravitreal bevacizumab in this population still remains, nevertheless results of the BEAT-ROP trial
suggest that intravitreal bevacizumab monotherapy to be far safer and more effective than conventional laser for treating retinopathy of prematurity. These finding suggest that bevacizumab and other anti-VEGF drugs may signify a real advancement in treating this disease.

We report results of a case series of preterm, extremely low-birth-weight infant, with zone-1, stage 3+ AP-ROP, treated successfully with intravitreous ranibizumab injection (LUCENTIS®; Novartis Inc.), an anti-angiogenic, monoclonal antibody fragment, with strong binding to VEGF-A. A detailed search and analysis of content in medical databases like Medline, EMBASE, etc., failed to reveal mention of any case study, illustrating treatment of ROP, with Intravitreal ranibizumab. Few recent studies have shown that bevacizumab may be an effective alternative in the management of ROP.6 Since ranibizumab is derived from the same parent murine antibody as bevacizumab but is much smaller than the parent molecule and has better tissue penetration, encouraged us for experimenting Intravitreal ranibizumab in combination with laser, for treatment of ROP.

**Report of a Case:**

20 consecutive eyes of 10 preterm babies 5 boys and 5 girls, with gestational age range from 27 to 32 weeks and gestation weight in range of 940 gms to 1200 gms, presented with zone-1, stage-3 ROP with corneal haze, media hazy, and iris neovascularisation. The International Classification of Retinopathy of Prematurity Revisited1 was referred to define Zone I and to subdivide the severity of stage 3 into mild, moderate, or severe depending on the extent of extraretinal fibrovascular tissue infiltrating the vitreous. Subsequently infants with bilateral moderate or severe stage 3 ROP were included in study, excluding infants with any congenital systemic or ocular abnormality. Diagnosis of AP-ROP plus disease was confirmed. The tunica vasculosa lentis and hyaloid arteries were persistent.

All the eyes were treated with bilateral intravitreal injections of ranibizumab (0.3 mg/0.02 ml) followed by peripheral retinal laser photocoagulation. 10 babies (6 boys and 4 girls), received intravitreal injections of ranibizumab. After a period of 4-12 weeks, laser photocoagulation was performed in the peripheral retina with the following settings: spot size 300 microns, duration of 20 milliseconds with 100 milliseconds repeat interval.

Considering the severity of disease and based on recent use of anti-VEGF as intravitreal injections for ocular diseases caused by neovascularisation especially wet age-related macular degeneration (US Food and Drug Administration approved for ranibizumab in June 2006), off label use of ranibizumab intravitreal injection was considered. It was decided that intravitreal ranibizumab injections at a dose of 0.3mg (60%of the normal adult dose) under sterile conditions would be given through the nasal pars plana of each eye. The injections were administered using continuous cardiorespiratory monitor. A speculum for premature infants was placed between the lids. A drop of povidone–iodine (5%) ophthalmic solution was placed into the conjunctival sac for 1 minute (pre and post injection) with the excess removed by a sterile cotton tip applicator from the temporal lid margin. Toothed forceps was used to steady the eye as dose of ranibizumab (0.02mL [0.3mg]) was injected behind the lens, repeating same procedure for the other eye. Post procedure, ophthalmic antibiotic drop Moxifloxacin (Vigamox®) was prescribed for both eyes to begin immediately for the next 10 days at an interval of 4 hrs. Indirect ophthalmoscopy was utilized to look for any injury to the lens, to determine the presence of adequate blood flow through the central retinal artery, and to identify any retinal tears or vitreous hemorrhage immediately after the injection.

Improvement was seen within 48 hours after the procedure and within next 1 week following the injections, the AP-ROP disappeared. Extraretinal fibrovascular proliferation superior and inferior to the typical indentation toward the macula disappeared and retinal vessels continued their anterior
growth into the previously avascular retina. Ophthalmic examinations revealed central and steady fixation without strabismus, round pupils could be fully dilated, clear corneas and lenses, and minimal or no myopia or anisometropia. No ocular complications, incidence of endophthalmitis or systemic thrombotic events, hypertension or gastrointestinal hemorrhages related to the intravitreal ranibizumab injections were observed.

Discussion

AP-ROP is an aggressive variant of ROP that has unique characteristics and can proliferate rapidly. Laser photoagulation, is still considered as initial treatment but thermal injury to the long posterior ciliary arteries in the horizontal meridian may result in anterior segment ischemia, the most devastating complication of ablative laser for threshold ROP.7 The choice of ranibizumab in our case study was deliberate, in order to try and minimize the possibility of systemic complications. The molecular weight of ranibizumab is 48 kd (provide greater retinal penetration) with intravitreal half-life of ~9 days and binding affinity of 0.14 nM. 8The shorter half life of ~ 15 hours versus 20 days for bevacizumab, leads to reduced risk of systemic complications.9 In addition to this, Fc fragment of antibodies binds immune molecules such as complement factors but ranibizumab does not comprise Fc fragment10, leading to a reduced risk of complement-mediated toxicity and eye inflammation. Similarly, the decision to give bilateral intravitreal injections was deliberate to avoid creating a case series of amblyopic eyes due to the unilateral visual deprivation caused by the inflammatory response, cataract, hemorrhage, or other complications after laser therapy.

Intravitreal injection of anti-VEGF agents in neonates offers potential advantages over laser treatment, as shown by BEAT-ROP study.5 These benefits include eliminating the direct effects of laser, which may include visual field loss secondary to retinal atrophy and myopia related to scleral weakening. Additionally, anti-VEGF therapy may offer a safer treatment option than blind external application of cryotherapy or laser photoagulation in infants with rigid pupils or media too opaque for adequate visualization of the retina. Intravitreal injection can also cause regression of the proliferative component of ROP, leading to absorption of hemorrhage and improved visualization for subsequent laser therapy, if needed.

In our case series AP-ROP was successfully treated by ranibizumab and revealed the effectiveness of intravitreal injection of ranibizumab for treatment of severe stage 3 ROP in zone I. Appropriate controlled studies with long-term follow-up are warranted to determine the potential safety and benefit Anti-VEGF agent ranibizumab, which may potentially play an increasing role as primary and/or adjunctive therapy in the future as additional studies become available.

Reference:

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Brief Report

Non Infectious Toxic Keratitis Following Bee Sting Injury –A Case Report

Introduction:
In our day to day clinical practice we come across various infective keratitis cases. Their management and prognosis depends on the clinical presentation. In this case report we present a different clinical scenario with an unusual presentation of keratitis.

Case report:
A 47-year-old gentleman, presented to ophthalmic casualty on 8th March 2011 with complains of defective vision, pain, redness & swelling in and around right eye - since 1 day. He gave a history of an insect (? bee) hitting the right eye on the previous day while driving his two wheeler. Following which he had severe foreign body sensation. He consulted a nearby ophthalmologist, who removed a foreign body from his right eye and topical antibiotics were prescribed. There after the patient was asymptomatic. Next day he got up to notice significant diminution of vision in RE associated with severe pain, redness and swelling around the eye. For this complaint he consulted another ophthalmologist who referred him to our institute for further management.

On presentation patient was on topical antibiotics. There was no history suggestive of scraping / any other procedure.
UCVA RE – CF at 1 meter NIP NIG LE – 6/18 with refraction +1.75 DS 6/6 N.V + 2.50 J2.

Right eye presented with lid edema with normal margin & lashes, Conjunctival chemosis and congestion
Corneal examination showed lack of lustre and transparency with corneal edema, dense radiating Descement’s folds and Striate Keratitis. With a circular 3x2.5 cm corneoscleral infiltrate at 3’o clock position.

Fig.1 :RE : Lid edema with normal lid margin & lashes.
Fig.2: RE conjunctival chemosis and congestion.
Fig.3 : Striate keratopathy with corneo-scleral infiltrate

On staining with fluorescein corneal Punctate Staining with epithelial defect over corneoscleral infiltrate was noted. Corneal sensation was intact.
AC- Details hazily seen, Pupil – RRR to D&C Reflex and lens appeared clear.
LE – Anterior segment examination was WNL.
Fundus examination: RE hazy view due to corneal edema, red glow seen. LE – WNL.

PROVISIONAL DIAGNOSIS:
On the basis of injury by insect & foreign body removal (probably sting) and typical clinical appearance a working diagnosis was made
RE: Toxic keratitis with corneoscleral infiltrate S/P Bee sting injury
LE - simple hypermetropia, presbyopia

Address for correspondence- aneetajabbar@yahoo.com Cornea and Anterior segment services,
Little Flower Hospital, Angamali.
Investigations: Routine blood investigations were WNL. Corneal scraping revealed no organism & negative culture report.

Patient was started on oral antihistaminics and analgesics. Topical prednicolone Acetate eye drops were started under antibiotic cover.

Supportive treatment in the form of topical cycloplegic and lubricant were given.

After 3 days pain & swelling subsided but defective vision persisted without any progression. Lid edema has considerably reduced, conjunctiva showed mild congestion without chemosis. Corneal edema was decreased. Corneoscleral infiltrate was resolving with surrounding early scarring & epithelial defect reduced in size. At this time oral medication were stopped and patient was discharged on topical steroids, antibiotics & cycloplegic.

After 1 week follow up:

UCVA in RE has improved to 6/60.

O/E: RE Lids and conjunctiva were normal. Corneal edema has significantly reduced with scarring of corneoscleral infiltrate. Specular reflection showed presence of corneal guttae.

At this stage topical steroids were tapered and cycloplegic was stopped

After 3 weeks follow up:

VA RE – 6/24

Refraction: +1.5 ds/+ 0.25 dc x 140- 6/6

O/E: Lids & Conjunctiva clear. Clear cornea with a marginal scar

Fig 4 Complete resolution of stromal edema with resolved corneo scleral infiltrate

DISCUSSION:

Corneal bee sting - an uncommon environmental eye injury. Though corneal or conjunctival bee sting represent localized form of reaction generalized reaction such as anaphylaxis may occur.

Complications due to ocular bee sting may involve the cornea, conjunctiva, anterior chamber, lens, optic nerve, or extraocular muscles.1,2,3 Complication associated with bee sting injury may be due to the penetrating, immunological or toxic effect of the stinger and its injected venom. Stinger, a modified ovipositor with a venom sac attached at the proximal end.

**Bee venom is a complex toxin consisting of:**

- Biologic amines - histamine, dopamine
- Nonenzymatic polypeptides toxins-melittin, apamin, mast-cell degranulating peptide, and minimine.
- High molecular weight enzymes - phospholipase A, phospholipase B, and hyaluronidase

Various clinical presentations which has been reported in literature includes: Periorbital edema, External ophthalmoplegia, Conjunctival chemosis & injection, Corneal edema, Striate keratopathy with radiating DM folds, Hyphaema, Iritis, iris depigmentation causing heterochromia iridis & Sector iridoplegia, Cataractous changes in lens & Subluxated lens, Optic nerve involvement in the form of papillitis, atrophy or papilloedema

MANAGEMENT:

**ACUTE MEASURES INCLUDE:**

- Suppression of inflammation – topical steroids
- Prevention of secondary infection – topical antibiotics
- Oral antihistaminics – to counteract biogenic activity
- Severe iritis with hypopyon in AC – paracentesis
- Surgical removal of sting
  Surgical removal of the retained bee stinger is still a matter of debate in the literature:
  Gilboa et al addressed two patients with corneal bee sting in whom the stingers remained protruding into the anterior chamber for 21 years and 28 years with no ocular manifestations.
  Arcieri et al described a 12-year-old boy with a retained corneal stinger protruding into the anterior chamber resulted in a severe corneal inflammation.

**LONG TERM MANAGEMENT:**

- Refractive correction - astigmatism induced by corneal scar
- Penetrating keratoplasty:
  corneal scar in visual axis
  corneal decompensation
- Lens extraction: cataractous or subluxated lens

Summary:

Corneal bee stings with or without retained stinger, are rare causes of keratitis. Though, it may present with violent clinical features, proper diagnosis and prompt management
carries wonderful prognosis.

**References:**


Sonali Nagpure et al. - Non Infectious Toxic Keratitis following bee sting injury

Sonali Nagpure is working at Little Flower Hospital, Angamail.
Bilateral PION - A Case Report.

Introduction

Visual loss following general surgical procedures have been widely reported, the etiology being ischemic optic neuropathy. Both anterior and posterior ischemic optic neuropathy is described, the former being more common. This condition generally occurs in the perioperative period. We report a case of bilateral visual loss in a young male patient which occurred one month after a major cardiac surgical procedure. A brain MRI of the patient showed diffusion sequence restriction which confirmed the diagnosis of ischemic optic neuropathy.

CASE

A 48 year old male patient was referred to us with bilateral total loss of vision of 3 days duration. He apparently noticed decreased vision in the right eye on waking up in the morning which progressed to complete blindness by afternoon. The left eye was normal at that time but the next day similar loss of vision in the second eye. There was no history of transient visual obscuration or visual field defects prior. He gave a history of DM of 5 years duration on treatment. He underwent cardiac bypass grafting with mitral valve repair surgery 1 month back. He was on Warfarin and antidiabetics. He was treated at a local hospital with intravenous methylprednisolone 1 gram iv for 3 days.

On examination the visual acuity was perception of light both eyes. The pupils were 5mm dilated and fixed. Rest of the anterior segment was within normal limits. Fundus examination was also normal. On the basis of clinical examination we made a provisional diagnosis of bilateral posterior ischemic optic neuropathy, possibly post surgical. The differential diagnosis we had in mind were bilateral ophthalmic artery occlusion, bilateral optic neuritis and bilateral occipital lobe infarction.

The patient underwent a FFA which showed normal A-V transit time. There was mild leakage from both the optic discs in the late frames. A brain MRI showed restricted diffusion from both the optic nerves suggestive of ischemia. MRA brain was normal. VEP showed inconsistent wave forms suggestive of bilateral optic pathway dysfunction. Routine blood and urine investigations were normal. With an ESR of 40mm / hour, ANA and CRP were non reactive. ECHO cardiogram showed left ventricular dysfunction.

Based on these findings we made a diagnosis of bilateral posterior ischemic optic neuropathy, possibly post surgical. In the followup period the vision improved to counting finger 1 metre and there was pallor of both optic discs.

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DISCUSSION

The incidence of post operative visual loss varies between 0.1 – 1.0 %, the cause being ischemia to the optic nerves. This manifests as anterior or posterior ischemic optic neuropathy. The term posterior ischemic optic neuropathy was first coined by Hayreh SS in 1981(15). This denotes ischemia to the retrobulbar optic nerve not supplied by the PCA. This is classified as arteritic (A-PION), non arteritic (NA-PION) and surgical (peri or post operative)3,4. The etiology of surgical PION is multifactorial. The main factors include severe and prolonged arterial hypotension, and hemodilution. This type of PION tends to cause bilateral massive vision loss and even complete blindness. The diagnosis of PION is one of exclusion based on normal fundus findings and no other ocular, orbital or neurological cause to explain the visual loss. Unlike NA-PION where spontaneous recovery and beneficial effect of systemic steroids is proven, the vision loss in surgical PION tends to be permanent and steroids are not effective5.

Patients generally experience vision loss as soon as they recover in the perioperative period. This patient had a rather delayed presentation. Hayreh SS has noted in one of his studies that a time lag of 2-3 weeks may occur2. More over a brain MRI showed diffusion sequence restriction which classically occurs in ischemic stroke. We came across one similar case report describing diffusion restriction6.

REFERENCES


Dr Sandhya. A completed her DNB from Sankara Eye Centre, Coimbatore and Fellowship in Medical retina from Amrita Institute of Medical Sciences, Kochi. She is presently working for Comtrust Eye Hospital, Ottapalam.
Comparison Of Screening Procedures In Hydroxychloroquine Toxicity.

Arch Ophthamol. 2012;130(4):461-469
Michael F. Marmor, MD

The study compared different screening procedures for hydroxychloroquine sulfate (Plaquenil) toxicity at different stages of damage. This article describes 10 referred patients with hydroxychloroquine retinopathy seen in 1 year and examined using the same battery of modern tests like 10-2 automated fields, multifocal electroretinography, spectral domain optical coherence tomography (SD-OCT) and fundus autofluorescence. All 10 patients used hydroxychloroquine for more than 6 years, and those with severe toxicity had been overdosed. Fundus examination findings were normal except for the patients with severe toxicity. All the patients showed parafoveal field loss, but this was sometimes subtle. Multifocal Electroretinography demonstrated parafoveal weakness in the milder cases. The SD-OCT cross sections showed parafoveal loss of the inner segment-outter segment and cone outer segment tip lines at early stages of toxicity, progressing to parafoveal thinning of the outer nuclear layer and eventually to retinal pigment epithelium damage. There was a ring of autofluorescence in most patients. The results demonstrated that fields, mf ERG, FAF and SD-OCT can all detect damage at a relatively early stage of hydroxychloroquine toxicity, but it is not predictable as to which test will be most definitive any given individual. This makes a strong argument for using more than one modality routinely. The choice will depend on availability, the quality of the records and the experience of the examiner.

Duration of anti-tubercular therapy in uveitis associated with latent Tuberculosis: A case-control study.
Marcus Ang, A Hedayatfar, W Wong, S P Chee.

The study aim to evaluate the effect of the duration of anti-tubercular treatment on the recurrence of uveitis associated with latent tuberculosis. It was a retrospective review involving 182 patients and all of them had uveitis suggesting a tubercular cause with positive Tubercular skin test and excluded other causes of infectious and non infectious uveitis. All patients had a minimum follow up of 6 months. Clinical characteristics, treatment type, treatment duration and clinical response were recorded. The main outcome measure was the effect of ATT duration on the recurrence of inflammation. Patients who completed > 9 months ATT were less likely to develop recurrence compared with those not treated with ATT (p=0.027), however the difference between >9 months and 6 to 9 months treatment were not statistically significant. They found that being female was an independent risk factor for recurrence of inflammation for which they could not find any explanation. The authors concluded that patients with uveitis associated with latent TB treated with ATT of > 9 months duration were less likely to suffer from recurrences compared with those who received corticosteroids without ATT. Thus the authors recommended that patients with uveitis associated with latent TB with no known etiology other than LTBI to account for their uveitis, should be treated with ATT of at least 9 months. The authors admit the relative short duration of follow up as a limitation of their study. Ideally a randomized control study is required to confirm this hypothesis.

Delayed versus acute onset endophthalmitis after cataract surgery.
A R Shirodkar et al.

The study aim to report a large consecutive case series of patients who developed delayed-onset and acute onset endophthalmitis after cataract surgery. It was a retrospective case series of 118 patients treated for post operative endophthalmitis. All cases were culture proven. 26 delayed onset patients and 92 acute onset patients were included in the study. The presenting visual acuity was <5/200 in 31% of delayed onset and 89% of acute onset patients. Hypopyon was found in 46% of delayed onset patients and 80% of acute onset patients. The most frequent isolate was propionibacterium acne (11/26) in delayed onset and coagulase negative staphylococcus (57/92) in the acute onset cases. Patients with most frequent isolate achieved a visual outcome of >20/100 in 91% of delayed onset patients and 56% of acute onset patients. This study from a large tertiary care centre demonstrated that patients with delayed onset endophthalmitis generally presented with better visual outcomes and less frequent Hypopyon than patients with acute onset endophthalmitis. Recurrence of infection was more commonly observed in patients with delayed onset post operative endophthalmitis. The study was limited by its small sample size, retrospective nature and variability between multiple physicians involved in the treatment of these patients.

Dr Reesha completed DNB from Little Flower Hospital and is currently doing fellowship in Medical Retina from Little Flower hospital, Angamali.

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Clinical Ophthalmology
A SYSTEMATIC APPROACH

The purpose of the seventh edition of clinical ophthalmology, as in previous edition, is to present the basics of clinical ophthalmic practice in a systematic and succinct manner, to be used as a springboard to more in-depth study of individual topics. The authors have tried to be comprehensive in the inclusion of key advances, with considerable updating and revision of the text. The majority of the illustration in the edition are new, and provide a more effective and vivid representation of many conditions. In response to trainees’ requests the present edition also places a greater emphasis on practical management, taking into account numerous published guidelines and other authoritative sources. The book is intended principally for the trainee and practising ophthalmologist, but previous edition have also been widely utilized by other eye care professionals, particularly optometrists.

Ideally suited for rapid reference and efficient, effective recall, this state-of-the-art multimedia resource will keep you up to date with current and evolving practice in the diagnosis and management of ophthalmic disorders, using a visually rich, succinct format that facilitates comprehension for trainees and practitioners. Online and in print, you’ll have access to the latest advances in the field.

- Access the complete contents online at www.expertconsult.com, with a downloadable image gallery.
- Learn from two renowned experts in the field.
- Includes over 2,700 high-quality images, 1,000 of which appear for the first time in this edition.
- Master the latest advances in ophthalmology: radical changes in the management of macular disease, including widespread introduction of VEGF inhibitor therapy; recent developments in the investigation and treatment of retinal vascular disease; new pharmaceutical interventions for a range of conditions, including infectious eye disease and glaucoma; and updated surgical procedures and methods, including oculoplastic, corneal, and glaucoma surgery.
- Guidance on examination, imaging, recognition of systemic conditions associated with ocular disease.

GEOMETRIC, PHYSICAL, AND VISUAL OPTICS

This basic textbook, written primarily for optometry and ophthalmology post graduate students contain an integrated approach to geometric, physical, and introductory visual optics. This book is nontraditional in the integration, sequencing, and conceptual development of the material. The non traditional aspects include an early emphasis on image formation, the use of the vergence-diaoptric power approach from the beginning, the relation of vergence to the geometric properties of wavefronts and the interchangeability of the wavefront representation with the ray presentations.

With nearly 30 years of experience in teaching optics, Dr. Keating offers a new edition of this reader-friendly book to enhance your understanding of geometric, physical, and visual optics.

Written primarily for optometry and ophthalmology post graduate students, the core basic optics, including thin lenses, ametropia corrections, accommodation, introductory astigmatism and refraction techniques, prisms, and prisms in lenses, are conveniently contained in the first 12 chapters. Many students need percolation time to absorb some of the abstracts concepts in optics. Dr. Keating introduces some concepts in early chapters and then returns to a more advanced treatment in later chapter providing you with adequate time to absorb to difficult concepts.

Completely revised and updated, you will benefits from new and rewritten section on: axial magnification • Jackson Cross Cylinder tests • retinoscopy reflex motions • field of view • the optics and indirect ophthalmoscopy • optical aberrations- diffractive lenses • the Doppler shift • lasers and the similarities and differences of Gaussian laser beams versus the propagation of lights from a point source • plus, a new appendix on angles and basic trigonometry.

Although not a matrix optic texts, matrices are used...
extensively in the chapters on spherical system and off-axis aspects of astigmatism. In particular, the matrix treatment of astigmatism serves as a foundation for the recently developed, much improved statistical techniques that deal with refractive corrections and astigmatism in all its aspects.

Through the key concepts of integrating basic geometric, physical, and visual optics, this book:

- Emphasizes conceptual understanding and development of optical intuition
- Uses the vergence-dioptic power-wavefront approach
- Incorporates fun everyday aspects of optics
- Is a helpful review for optometry boards and qualifying examinations

This unique, fundamental, functional textbook is your best source for integrating visual optics with geometric and physical optics.

Dr. C V Andrews after finishing his MS Ophthalmology and further training from the prestigious BJ Medical College, Ahmedabad went on to do M Phil from BITS, Pilani. He is professor of ophthalmology and medical superindentent at Jubilee Mission Medical College, Thrissur and is currently the president of KSOS
## Corneal Dystrophies And Degenerations, Simplified

### Epithelial Dystrophy

<table>
<thead>
<tr>
<th>Eponyms</th>
<th>Inheritance</th>
<th>Onset</th>
<th>Signs</th>
<th>Histology/stains used</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cogan microcystic dystrophy/map-dot finger print</td>
<td>AD-gene</td>
<td>1-2 yrs</td>
<td>Central Intraepithelial cysts sparing limbus Sensations reduced</td>
<td>Intraepithelial cysts Irregular Thickening of basement membrane</td>
<td>Tears substitutes</td>
</tr>
<tr>
<td></td>
<td>12q13 or 17q12</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Meesmann</td>
<td>AD orXLD</td>
<td>1st decade</td>
<td>Grey bands whorled pattern Densely packed microcysts</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Gene-Xp 22.3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Lisch</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Bowman Layer Dystrophy

<table>
<thead>
<tr>
<th>Eponyms</th>
<th>Inheritance</th>
<th>Onset</th>
<th>Signs</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Reis buckler</td>
<td>AD-5q31</td>
<td>1-2nd decade</td>
<td>Central grey white fine polygonal opacities in BM Increase in density with age Sensations reduced</td>
<td>Replacement of bm layer and EBM with fibrous tissue</td>
</tr>
<tr>
<td>Bowman layer 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Thiel behke</td>
<td>AD 10q24</td>
<td>1st decade</td>
<td>Honeycomb pattern opacity</td>
<td>Curly fibers in BM</td>
</tr>
<tr>
<td>Bowman layer 2</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stromal Dystrophy</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
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<td>---</td>
<td>---</td>
</tr>
<tr>
<td>1. Lattice type I</td>
<td>Biber Haab Dimmer</td>
<td>AD 5q31</td>
<td>1st decade</td>
<td>Anterior stromal - Glassy refractile dots. Coalesce into fine lines. Spare periphery Poor epithelial-stromal adhesions, recurrent erosions</td>
</tr>
<tr>
<td>2. Lattice type 2</td>
<td>Meretoja</td>
<td>AD 9q34</td>
<td>3rd decade</td>
<td>Randomly scattered short fine lattice, delicate radially oriented</td>
</tr>
<tr>
<td>3. Lattice type 3&amp;3A</td>
<td>3-AR 3A-AD 5q31</td>
<td>4-7th decade</td>
<td>Thick ropy lines limbus-limbus Rapid progression if trauma+ Mid stromal deposits larger than type I</td>
<td></td>
</tr>
<tr>
<td>5. Granular II</td>
<td>Avellino</td>
<td>AD 5q31</td>
<td>2nd decade</td>
<td>Superficial fine opacities like rings discs stars Central dense</td>
</tr>
<tr>
<td>6. Macular</td>
<td>AR 16q22</td>
<td>1st decade</td>
<td>Central Anterior stromal haze Limbus involved</td>
<td>aggregations of GAG – stains with Prussian blue and colloidal iron</td>
</tr>
<tr>
<td>8. Central cloudy dystrophy</td>
<td>Francois dystrophy</td>
<td>AD</td>
<td>-</td>
<td>Polygonal cloudy grey opacities separated by clear spaces Posterior stroma Leather appearance</td>
</tr>
<tr>
<td>Endothelial dystrophies</td>
<td>Inheritance</td>
<td>Onset</td>
<td>Signs</td>
<td>Treatment</td>
</tr>
<tr>
<td>-------------------------</td>
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<td>-----------</td>
</tr>
<tr>
<td>1. Fuchs dystrophy</td>
<td>AD</td>
<td>Old age</td>
<td>Stage 1 (corneal guttata)</td>
<td>Central corneal edema, Blurred vision, Worse morning, Persistent epithelial edema, Microcyst Bullous keratopathy, Pain discomfort, Exposed nerve ends.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Stage 2</td>
<td>-Topical Nacl 5% drops, -BCL, -PK, -Conj flaps</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Stage 3</td>
<td></td>
</tr>
<tr>
<td>2. Posterior polymorphous</td>
<td>AD</td>
<td>Birth</td>
<td>Subtle vesicular endothelial pattern, Band like lesions, Diffuse opacities</td>
<td>Associations: Alport syndrome, iris membranes, PAS, Ectropion uvea, corectopia, polycoria, glaucoma</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>No treatment</td>
</tr>
<tr>
<td>3. Congenital hereditary endothelial dystrophy CHED 1&amp;2</td>
<td>CHED 1-AD CHED2-AR</td>
<td>Perinatal</td>
<td>Diffuse corneal edema, Blue gray ground glass-total opacification</td>
<td>D/D: Congenital glaucoma, Mucopolysaccharidosis, Birth trauma, Rubella keratitis, Sclerocornea</td>
</tr>
</tbody>
</table>

### Corneal Degenerations

<table>
<thead>
<tr>
<th>Age related degenerations</th>
<th>Age</th>
<th>Location</th>
<th>Layer affected</th>
<th>Material deposited</th>
<th>Pathogenesis</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Arcus senalis</td>
<td>60%-40-60yrs 100%-80yrs</td>
<td>Peripheral cornea</td>
<td>Stroma</td>
<td>Lucid interval +</td>
<td>Cholesterol esters, phospholipids triglycerides</td>
<td>-Proximity of b.v. to peripheral cornea, -High degree of b.v permeability LDL Passes into cornea</td>
</tr>
<tr>
<td>2. Vogt limbal girdle</td>
<td>50% over 40yrs</td>
<td>Peripheral cornea, Interpalpebral</td>
<td>Subepithelial</td>
<td>Calcium deposits +/-</td>
<td>White opacity in the medial &amp; temporal limbal regions, Clear zone separating it from the limbus.</td>
<td>Not required</td>
</tr>
<tr>
<td>3. Corneal farinata</td>
<td>Central</td>
<td>Deep corneal stroma</td>
<td>Flour like deposits</td>
<td></td>
<td></td>
<td>Not required</td>
</tr>
<tr>
<td>4. Crocodile shagreen</td>
<td>Anterior 2/3rds of cornea</td>
<td>-</td>
<td>Grayish white</td>
<td>polygonal stromal opacities</td>
<td>Not required</td>
<td></td>
</tr>
<tr>
<td>Band keratopathy</td>
<td>Central Interpalpebral calcification Band like pattern. Limbus free</td>
<td>Bowmans layer, epithelial basement membrane anterior stroma</td>
<td>Calcium salts</td>
<td>Ocular causes - chronic ant. uvetis, phthy sis bulbi, silicon oil in AC, chronic corneal edema, severe keratitis</td>
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</tr>
<tr>
<td>Spheroidal (corneal elastosis, labrador climatic droplet kpthy Bietti nodular dystrophy)</td>
<td>Bilateral Men UV exposure</td>
<td>Interpalpebral at periphery coalescent lesions in center</td>
<td>Irregular proteinaceous deposits in anterior stroma that replace bowmans</td>
<td>Amber colour granules</td>
<td>Protection from UV rays</td>
<td></td>
</tr>
<tr>
<td>Salzmann nodular</td>
<td>2ndry to chronic keratitistra-choma</td>
<td>Superficial stromal opacities</td>
<td>Epithelial iron deposits</td>
<td>Protection from UV rays</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Terrien marginal degeneration</td>
<td>Bilateral Men 4th decade</td>
<td>Peripheral thinning</td>
<td>Subepithelial &amp; stromal</td>
<td>Fine yellow punctuate stromal opacities Mild vascularisation</td>
<td>Progressive decrease in vision due to astigmatism corrected by Glasses Later stages – corneoscleral graft</td>
<td></td>
</tr>
<tr>
<td>Pellucid marginal degeneration</td>
<td>Bilateral</td>
<td>Peripheral</td>
<td>no vasc, deposits</td>
<td>Arcuate area of thinning in inf peripheral area concentric to limbus separated by clear area. Irregular astigmatism</td>
<td>keratoplasty</td>
<td></td>
</tr>
<tr>
<td>Dellen</td>
<td>Focal thinning in peripheral cornea caused by desiccation (drying). Temporal limbus</td>
<td>thinning of epi, Bowman’s layer &amp; superficial stroma</td>
<td></td>
<td>Rehydrate cornea Bandage C.L/ double patching</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Diagnose this condition?
What two test results will clinch the diagnosis?

Send your answers to gopalspillai@gmail.com
The First Correct answer gets the prize

Last time's winner is Dr Divya Dharmarajan
The answer is "Optic nerve head pit with serous retinal detachment/retinoshisis" (Kranenburg Syndrome).
Treatment strategy is to laser the edge of the pit, vitrectomy and/or gas and positioning.
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 communications, 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: Author(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 copy right 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.

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.