October 2017 Volume XXXV No 3

insight

Scientific Journal of

MEDICAL & VISION RESEARCH FOUNDATIONS

SANKARA NETHRALAYA

www.sankaranethralaya.org

Editor: Parthopratim Dutta Majumder

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Scientific Journal of **Medical & Vision Research Foundations**

Year: 2017

Issue: Vol. XXXV | No. 3 | October 2017 | Pages 1–34

Typeset at: Nova Techset (P) Ltd., Chennai, India

Printed at: Gnanodaya Press, Chennai, India

Sankara Nethralaya – The Temple of the Eye.

It was in 1976 when addressing a group of doctors, His Holiness Sri Jayendra Saraswathi, the Sankaracharya of the Kanchi Kamakoti Peetam spoke of the need to create a hospital with a missionary spirit. His words marked the beginning of a long journey to do God's own work. On the command of His Holiness, **Dr. Sengamedu Srinivasa Badrinath**, along with a group of philanthropists founded a charitable not-for-profit eye hospital.

Sankara Nethralaya today has grown into a super specialty institution for ophthalmic care and receives patients from all over the country and abroad. It has gained international excellence and is acclaimed for its quality care and compassion. The Sankara Nethralaya family today has over 1400 individuals with one vision – to propagate the Nethralaya philosophy; the place of our work is an Alaya and Work will be our worship, which we shall do with sincerity, dedication and utmost love with a missionary spirit.

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What is new in paediatric ophthalmology?

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Paediatric ophthalmology – The oldest subspecialty in ophthalmology and an area which continues to intrigue me even after 20 years of practice! The practice of ophthalmology in children is replete with challenges, starting from difficult examination to the special needs of the immature visual system. The fact that these young patients have an entire lifetime to live with the consequences of our intervention makes it imperative that we offer them the best care we can. While it is true, that research is often skewed towards the older population, recognition of special needs of our young patients, has directed more and more trials to address the diseases in paediatric population. Here are the highlights of what is new in the management of some common paediatric eye conditions.

Amblyopia is one of the most commonly encountered conditions in paediatric ophthalmology practice. For more than 100 years, amblyopia, or the 'Lazy eye' as it is often referred to, was believed to be a result of cortical suppression of a blurred or disconjugated image. The new emerging picture of amblyopia is that of a monocular consequence of a primary binocular obstacle to normal visual development. The focus is now on exploring binocular therapy in the form of dichoptic stimulation for amblyopia treatment in children. Simple games have been developed which use a red green display with chromatically matched goggles. Birch et al.[1] compared the dichoptic iPad gameplay with sham iPad gameplay in amblyopia treatment and found that the group given dichoptic iPad games had significant visual gain 3 months post-treatment. Although the randomized controlled trial conducted by PEDIG[2] favoured patching, it did not establish that iPad therapy was substantially worse than patching. While we wait for more concrete results, we hope the future of amblyopia treatment will be 'No Patch, Only Play'!

Strabismus is another disease entity we commonly encounter in our practice. The ultimate goal of treatment as a strabismologist is not just cosmesis, or good distance and near vision, but good stereopsis. With advances in our knowledge of aetiologies and newer techniques of surgery, the outcomes are bound to improve. The technique of Minimally Invasive Strabismus Surgery (MISS) is being employed to perform all types of strabismus surgeries, namely rectus muscle recessions, resections, plications, reoperations, retroequatorial myopexias, transpositions, oblique muscle recessions, and adjustable sutures, even in the presence of restricted motility.[3] The technique first described by Gobin for access to rectus muscles has been adapted for other procedures. Muscles are not accessed through one large opening, but using several keyhole openings placed where needed for the surgical steps, ensuring they are far away from the limbus. The benefits include lesser corneal complications, avoiding disruption of the limbus, increased patient comfort, faster recovery, easier to perform resurgery. Mini-plication is a new rectus muscle tightening procedure for the correction of small- angle strabismus that can be performed under topical anaesthesia. The results have been encouraging for correction of diplopia in patients with small angle deviations up to 20 prism diopters.[4] Mini-tenotomy is a similar procedure for weakening of recti. Although it has a long learning curve, MISS is a technique which must be explored by every strabismus surgeon. The transposition surgeries for incomitant strabismus are undergoing a paradigm shift. Transposition surgeries commonly performed today are superior rectus transposition (SRT), modified Nishida's procedure, and Knapp's procedure. SRT with or with medial rectus recessions is very useful in esotropic Duane's and sixth nerve paresis. Modified Nishida procedure involves direct suturing of the recti muscles to the sclera without any tenotomy or splitting of muscle, thus very useful in the treatment of sixth nerve palsy and also missing medial rectus muscles. Medial transposition of split lateral rectus has also been tried with success in cases of third nerve palsy.[5]

Childhood cataract accounts for 7.4–15.3% of childhood blindness.[6] Most who have operated adult and paediatric cataracts would agree that these patients cannot be treated as young adults. Challenges and controversies abound right from deciding whether or not to operate to need for intraoperative skill, to postoperative management of complications. The technological advances have made cataract surgery in children faster and safer. The availability of 23–25 gauge instruments, namely scissors, capsule forceps, and vitrectors, ensures safer surgery and well-maintained anterior chamber. Higher vitrectomy cut rates of up to 4000/min and better fluidics in newer machines have made vitrectomy safer in the youngest patients. At the end of the procedure, the wounds can be left sutureless without fear of leak. The Fugo's plasma blade, which has been approved by the US Food and Drug Administration, can be

used for capsulotomy, especially in cases of the persistent foetal vasculature and posttraumatic fibrotic capsules. The newer advanced microscopes include a rhexis assistant which is an intraoperative projection of rings of custom sizes which can be used as guides for anterior and posterior capsulorhexis. The future certainly lies in the use of femtosecond and Zepto surgery for anterior and posterior rhexis. Lin et al.[7] have described a novel technique, in which children <2 years of age received lensectomy with an eccentric, smaller capsulorhexis leaving LECs intact. The residual cells regenerated a lens structure with refractive power and accommodative ability. This must be studied in more eyes before it can be an adopted widely.

Genetics is an ever-evolving branch in medical science, it being no different in paediatric ophthalmology. The most researched disorder and its genetic association in recent times has been myopia. An Australian study[8] came up with the conclusion that multiple genes on chromosome 5q are responsible for determinants of axial length which ultimately determines myopia, while another recent study[9] found the gene locus for myopia being 15q14. Genetics has also helped us group together similar diseases which were diverse in their presentation, but were later found to have similar genetic associations. Duane's syndrome, Duane radial ray syndrome, CFEOM, Moebius syndrome, and congenital ptosis have now come under the common umbrella of diseases termed as Congenital Cranial Dysinnervation Disorders (CCDDs), the genes associated being SALL4[10], KIF21A, etc.[11] As the genetic profiling of different diseases is completed, our understanding of the disease will change over time. Also, novel avenues of therapy can be targeted at the involved gene for treatment, like sub-retinal injection of adeno-associated virus carrying wild-type RPE65 complimentary DNA for RPE65-associated LCA.

The field of paediatric ophthalmology is indeed evolving and newer horizons are being explored. Recognition of muscle pulleys and the neurological basis of CCDDs have improved our understanding of strabismus aetiologies. Newer imaging modalities such as high-resolution and dynamic MRI aid better planning of strabismus surgery. Management of nystagmus has come a long way with the availability of video nystagmography, and various drugs and surgical procedures. We are fortunate to witness this era of innovation in paediatric ophthalmology. Even as we say this, it is important that we, as ophthalmologists, constantly update what we have learnt during our training and ensure we are aware of various diagnostic and therapeutic options at our disposal.

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How to cite this article Guha S. What is new in paediatric ophthalmology? *Sci J Med & Vis Res Foun* 2017;**XXXV**:1–2.

Implants in glaucoma: a minor review

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Introduction

Glaucoma filtering procedures have been the mainstay of surgical glaucoma management for the past century. Ever since Cairns described his technique of trabeculectomy in 1968, it has been modified and augmented by surgeons over the years, but the basic principle has remained the same. However, the vision-threatening complications associated with trabeculectomy[1] make it a less suitable option in many cases. Hence has come the era of implants – glaucoma drainage devices (GDDs) and Stents in surgical glaucoma management which we shall briefly review in this article.

Glaucoma drainage devices

They occupy an important place in the treatment of complicated and refractory glaucomas, both as a primary surgical modality and as a secondary procedure.[2–8]

In 1906, a horse hair thread[9] was placed through a corneal paracentesis in an attempt to drain hypopyon externally. The same technique was used subsequently to treat two patients with painful absolute glaucoma.

In 1969, Molteno explained the pathophysiology of bleb resistance and postulated the idea of draining fluid away from the limbus, to increase the success rate and designed a tube.[10] All of the currently available GDDs are based on these fundamental principles.[11–16]

GDDs work by creating an alternate pathway for aqueous outflow from anterior chamber (AC) through a tube of implant toward subconjunctival space.[11–16]

Indications

- Failed trabeculectomy.
- Uveitic glaucoma.
- Neovascular glaucoma.
- Sturge–Weber's syndrome.
- Penetrating keratoplasty with glaucoma.
- Retinal detachment surgery with glaucoma.
- Iridocorneal endothelial syndrome.
- Refractory infantile glaucoma.

Relative contraindications

- Vitreous in AC.
- Eyes with severe scleral and or sclerolimbal thinning.
- Extensive fibrosis of conjunctiva.

Types of implants

GDD with no resistance	GDD with resistance
Single-plate or double-plate Molteno	Ahmed glaucoma Valve (AGV)
Single-plate or double-plate Baerveldt	Krupin slit valve

Mechanism of action: Following implantation, a fibrous capsule forms around the end-plate and a tube drains aqueous from the AC to the space between the endplate and surrounding non-adherent fibrous capsule. This fibrous capsule around the end-plate creates resistance to flow. Aqueous passes through the capsule by the process of passive diffusion and is absorbed by surrounding capillaries and lymphatics.

Non-valved implants

Molteno implant: The **Molteno implant** (Figure 1a) consists of a silicone tube (outer diameter 0.6 mm and inner diameter 0.3 mm) that opens onto the upper surface of a circular, acrylic plate 13 mm in diameter. The surface area of the single-plate model is 134 mm. The edge of the plate has a thickened rim 0.7 mm high that is perforated to permit suturing to the sclera, thus preventing plate migration. The **double-plate Molteno (**Figure 1b) – a second end-plate is attached to the right or left of the original end plate, thus doubling its surface area.[10,11]

The **Baerveldt implant** (Figure 2) is made up of silicone episcleral plate with different surface areas – 200 mm^2, 250 mm^2, 350 mm^2, 500 mm^2, the 350 mm^2 being the most preferred size. The proximal portion of the plate has a flange with two large fixation holes to allow the growth of fibrous tissues and this aids in scleral attachment.[15]

The **Aurolab aqueous drainage implant (AADI)** (Figure 3) is based on the principles of Baerveldt implant and serves as a low-cost alternative GDD in cases of refractory glaucoma.[16]

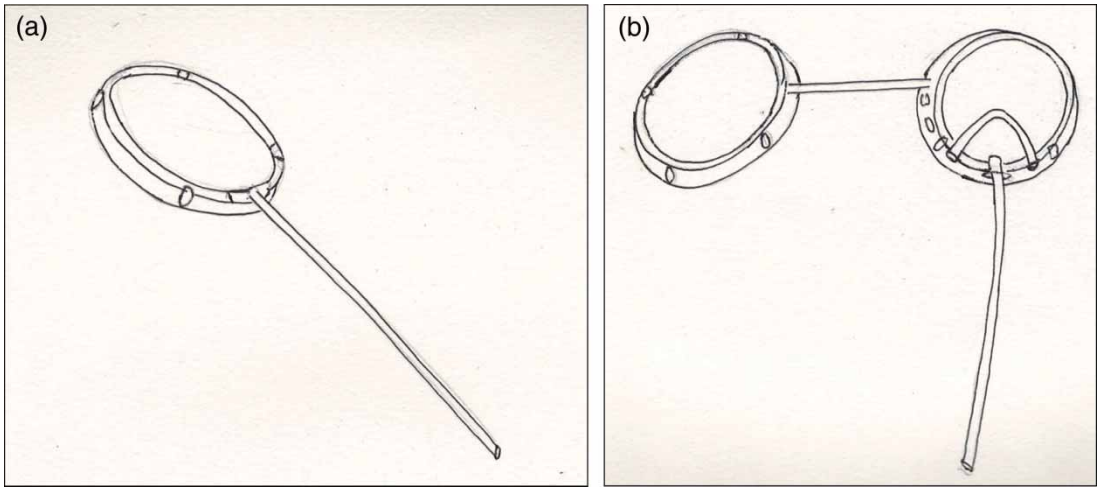

Figure 1. (a) Single plate Molteno implant; (b) Double plate Molteno implant.

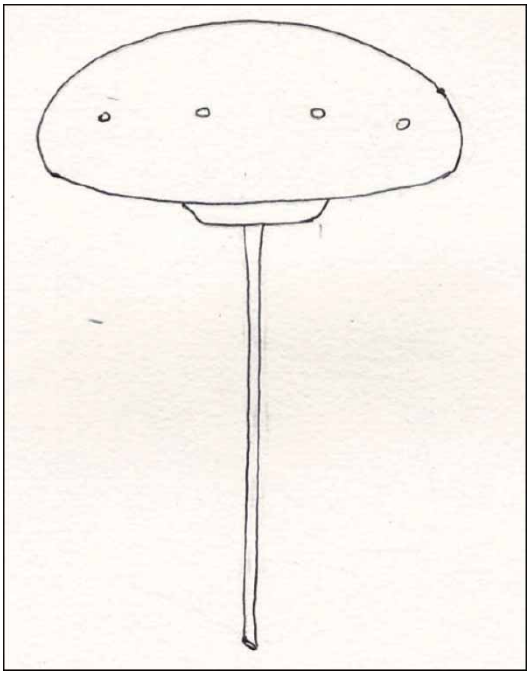

Figure 2. Baerveldt implant.

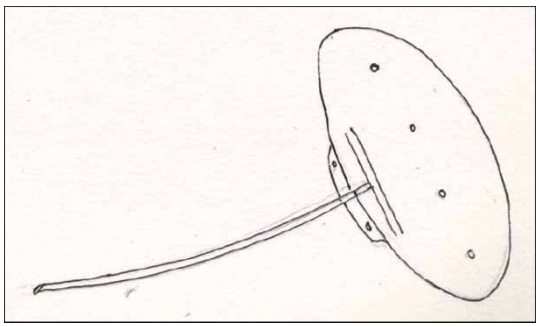

Figure 3. The Aurolab Aqueous Drainage Implant – AADI.

Valved implants

Ahmed Glaucoma Valve (AGV): It provides a more complex mechanism to control aqueous humous outflow. It was developed by Mateen Ahmed and was approved by the FDA in 1993.

The AGV consists of 3 parts (Figure 4a):

1 Plate: silicone, polypropylene or porous polyethylene (depending on the model).

2 Drainage tube in silicone.

3 Thin silicon elastomer membranes which act as a valve.

The valve mechanism of AGV consists of thin silicone elastomer membranes, which are pretensioned and designed to open when intra-ocular pressure is 8 mmHg or more. Hence in hypotony, the flow through the valve ceases and further complications can be avoided.

Site of implantation: The supero-temporal quadrant is the preferred site for the implantation of GDD followed by the infero-temporal quadrant. Supero-temporal site provides the easiest access for the surgeon to implant the plate and is least likely to produce extra-ocular motility disturbances. Supero-nasal quadrant is the least preferred site due to high chances of motility issues. The tube of the AGV is most commonly placed in the AC. However, it may also be placed in the sulcus in a pseudophakic eye and in post corneal transplantation (Figure 4b) cases to prevent corneal decompensation or in the pars plana in case of a vitrectomized eye.

Intraoperative complications

- Perforation and exposure of uveal tissue during fixation of the implant.

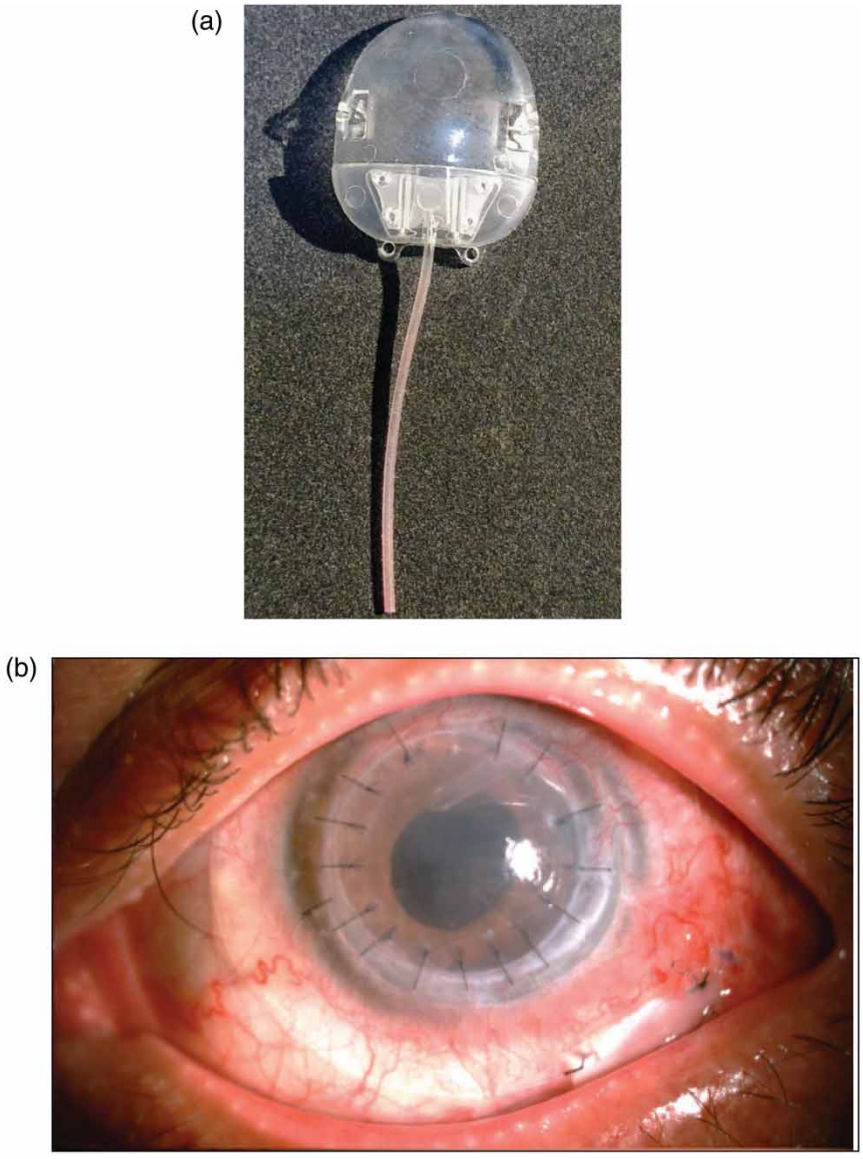

Figure 4. (a) Ahmed glaucoma valve; (b) AGV in a post PK eye.

- Ciliary body hemorrhage.
- Vitreous loss.
- Leakage around the tube.
- Hyphema.
- Vitreous hemorrhage.
- Suprachoroidal hemorrhage or expulsion.

Post-operative complications

- Hypotony: More common in non-valved implants. Excess flow can result in a flat AC, prolonged hypotony and choroidal detachment.
- Tube exposure/migration/extrusion: Cases with end-plate exposure may require conjunctival autograft or pericardial patch graft sutured to the Tenon capsule.
- Tube obstruction: Can happen with blood, fibrin, vitreous, iris plug or kinking.[13]
- Tube retraction: Retraction of the tube from the AC may be managed by placing an extender sleeve with a larger inner diameter over the existing tube.
- Bleb encapsulation: Managed with aqueous suppressants.
- Corneal endothelial touch.
- Corneal graft failure.
- Ocular motility disturbance.
- Endophthalmitis.
- Vision loss.

Modifications to prevent hypotony with non-valved implants

- **Stent – Internal tube occlusion**: Aqueous drainage through a non-valved device can be regulated in the early post-operative period by passing a 4-0 or 5-0 prolene or nylon suture through the lumen of the implant. Once the fibrous capsule is formed around the implant, the stent suture is removed at slit lamp.
- **Ligature – External tube occlusion**: The flow of aqueous through a non-valved device is restricted by placing a suture ligature around the external aspect of the tube.
- **Two-stage procedure**: In the first stage, only the plate is anchored to the globe and the tube is left in the subconjunctival space without entering the eye. Four to six weeks later, once the capsule has been formed around the implant, the conjunctiva is opened and a tube is inserted into the chamber to establish the flow.

Post-operative events

Hypotensive phase: It is particularly common in nonvalved implant from immediate post op period up to 3–4 weeks. However, in few cases it can be seen in valved implants too.

Hypertensive phase: It is more commonly seen with the AGV. It is defined as IOP more than 21 mmHg from 3–6 weeks to 6 months post surgery that was not a result of tube obstruction, retraction or malfunction. Topical anti-glaucoma medications are needed to control the intraocular pressure (IOP) in this stage.

Let us look at a few important studies involving GDDs.

Ahmed Baerveldt comparison study

This study compared surgical outcome of Ahmed and Baerveldt implants in a total of 276 patients. Mean IOP was lower after Baerveldt placement after 1 year of follow-up (15.4 mmHg Ahmed group vs. 13.2 mmHg Baerveldt group, $p = 0.007$), and use of adjunctive glaucoma medications was similar with both aqueous shunts (1.8 medications Ahmed group vs. 1.5 medications Baerveldt group, $p = 0.07$).[14]

Although the cumulative probability of failure at 1 year was not significantly different, patients, who received a Baerveldt implant, experienced significantly more early post-op complications during the first 3 months after surgery (43% Ahmed group vs. 58% Baerveldt group, $p = 0.016$). No significant difference in the rate of late post-op complications was observed between the two aqueous shunts (29% Ahmed group vs. 37% Baerveldt group, $p = 0.16$). The lower IOP observed with the Baerveldt implant can be explained by the larger end plate; however, the greater efficacy of the Baerveldt implant in reducing IOP occurs at the expense of a higher rate of surgical complications.

Tube versus trabeculectomy (TVT) study

This is one of the landmark trials which compared surgical outcomes of trabeculectomy vs Baerveldt implant. Tube shunt surgery had a higher success rate compared to trabeculectomy with MMC at 5 years of follow-up in the TVT Study.[15,17] Both procedures were associated with similar IOP reduction and use of supplemental medical therapy at 5 years. The cumulative probability of failure at 5 years of follow-up was 29.8% in the tube group and 46.9% in the trabeculectomy group. The rate of reoperation for glaucoma was 9% in the tube group and 29% in the trabeculectomy group.

Stents in glaucoma

Trabeculectomy and GDDs remain the gold standard for surgical glaucoma management; however, during the past few years, the interest and use of less invasive glaucoma surgeries has gradually evolved[18] 'Micro Incision Glaucoma Surgeries-MIGS' encompasses a group of procedures which are characterized by minimum external dissection, short operating times, good safety profile and rapid recovery. Tubular stents, composed of different materials, which lower IOP, without the associated risks form an important component of MIGS.[19]

In the following section, we shall be covering a few of these stents.

iStent

The iStent (Glaukos Corporation, Laguna Hills, CA, USA) is a 1 × 0.3 mm heparin-coated titanium implant, which is placed ab interno, through the trabecular meshwork into the Schlemm's canal. This bypasses the juxtacanalicular trabecular meshwork and the inner wall of the Schlemm's canal (which is the major component of aqueous outflow resistance) and allows drainage of aqueous into the collector channels. It is an L-shaped device, with a pointed tip which penetrates the trabecular meshwork. It has a 1-mm long trough which is placed in the Schemm's canal and a 'snorkel' that faces the AC to drain aqueous from it (Figure 5a). It is implanted with the use of a disposable inserter via a gonioscopic guided approach through a paracentesis opening. The iStent has been US FDA approved since 2012 for use in combination with cataract surgery in patients with ocular hypertension and in mild-to-moderate glaucoma on anti-glaucoma medications.[20]

The results of the iStent study group, which is the largest RCT to date (240 patients), compared

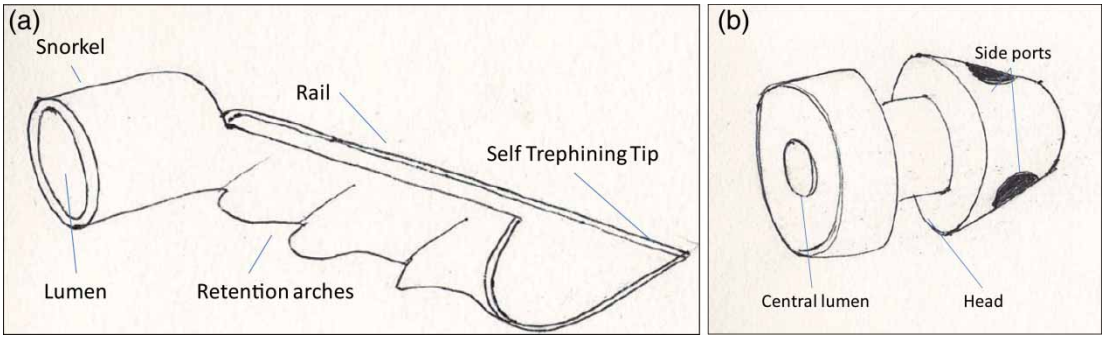

Figure 5. (a) The Glaukos iStent; (b) The iStent inject.

IOP drop and medication reduction between patients who underwent cataract surgery alone vs cataract surgery combined with a single iStent. The study group showed an 8% drop in IOP and 87% medication reduction compared to 5.5% drop in IOP and 73% medication reduction in the control group at the end of 1 year, with the medication reduction being statistically significant ($p = 0.005$).[20]

With an idea that multiple iStents appear to provide better IOP control, Glaukos has manufactured a second-generation iStent called iStent Inject (Figure 5b) which is designed to be used both left- and right-handed and comes preloaded with 2 stents. Studies show a 48% reduction in IOP with the iStent Inject at the end of 12 months.[21]

Common complications are minimal hyphema from Schlemm's canal, transient IOP spike, corneal edema and stent malposition.

Hydrus Microstent

The Hydrus Microstent (Ivantis Inc., Irvine, CA, USA) is an 8 mm long nickel-titanium device which resembles an intracanalicular scaffold. It has three windows along its length and is open on the posterior surface. It dilates approximately one quadrant of the Schlemm's canal which allows the aqueous to bypass the trabecular meshwork and gain access to multiple collector channels. It is inserted via an ab interno gonioscopic approach.[22]

The device is available only for investigational use in the USA. Studies, however, have shown a significant drop in IOP (almost 50%) and medication reduction when combined with cataract surgery.[23]

Xen GEL implant

The XEN GEL Implant (Allergan NYSE:AGN, Dublin, Ireland) is a 6-mm cylinder of collagen derived gelatine, crosslinked with glutaraldehyde, which is biocompatible and non-biodegradable. It comes pre-loaded in an injector and is implanted ab interno through a clear corneal incision under gonioscopic visualization. It creates a drainage pathway between the AC and subconjunctival space (Figure 6). Conjunctival dissection is not required. Currently, 45 nm lumen-sized tube is recommended for clinical use.

Galal et al.[24] have reported significant drop in mean IOP, from 16 ± 4 mm of Hg to 12 ± 3 mm of Hg postoperatively, after 1 year of follow-up in 13 POAG patients who underwent XEN 45 gel implant surgery with Mitomycin C. There was significant drop in the number of glaucoma medications from preoperative 1.9 ± 1 to postoperative 0.3 ± 0.49.

EX-PRESS glaucoma filtration device

The EXPRESS glaucoma filtration device (Alcon Laboratories Inc., Fort Worth, TX, USA) is a stainless steel implant which is <3 mm in length. It is implanted ab externo under the scleral flap with an injector. It creates a communication between the AC and subconjunctival space. It does not require peripheral iridectomy. Studies have shown almost similar IOP lowering ability of ExPRESS shunt compared to conventional augmented Trabeculectomy.[25]

The higher cost of this implant is a matter of consideration when deciding for primary Ex-PRESS implant insertion.

Cypass microstent: suprachoroidal space

The Cypass stent (Alcon Surgical, division of Novartis Pharma, USA) is a fenestrated polyamide tube 6.35 mm in length, with a 300 um lumen. It is designed to be implanted ab interno with the help of a guide wire and inserted between the ciliary body and the sclera (Figure 7).

The Compass trial is a multicenter randomized controlled trial on 505 mild-to-moderate POAG patients who underwent phacoemulsification with IOL and Cypass stent implantation and only phacoemulsification with IOL implantation.

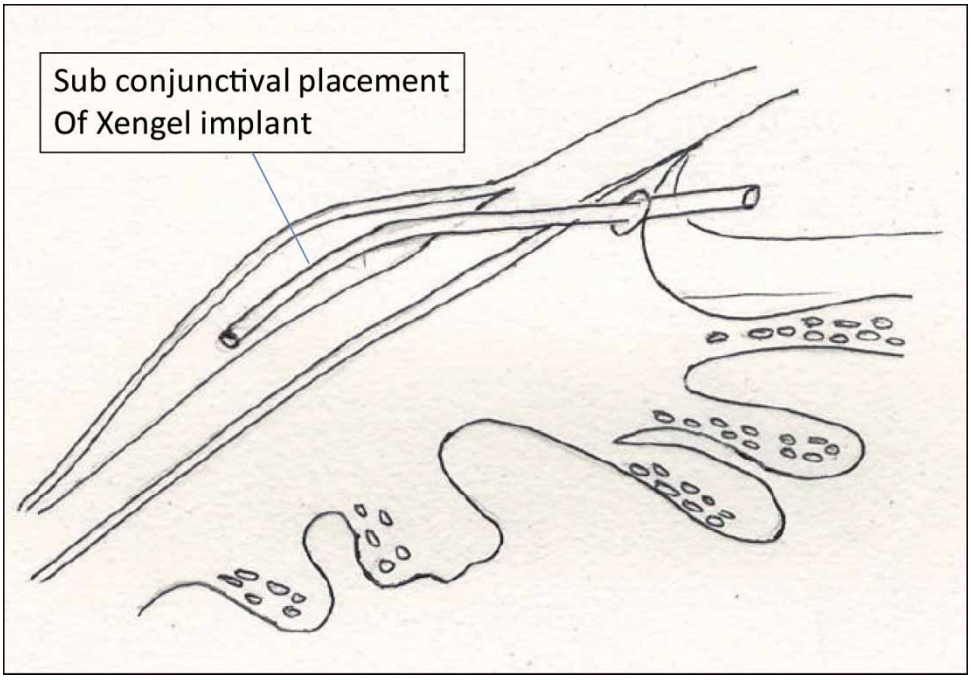

Figure 6. The XenGel implant.

At 2 years of follow-up Cypass group has shown significant better IOP control and reduction in the number of glaucoma medications compared to only phacoemulsification group.[26] The CyCLE (Cypass clinical experience) study has also shown similar efficacy of the device.[27]

InnFocus Microshunt

The InnFocus Microshunt (InnFocus Inc., Miami, FL, USA) is a synthetic tube that diverts aqueous from the AC to the subconjunctival space. The MicroShunt is made of a highly biocompatible material, SIBS. It is inserted ab externo under a sclera flap.

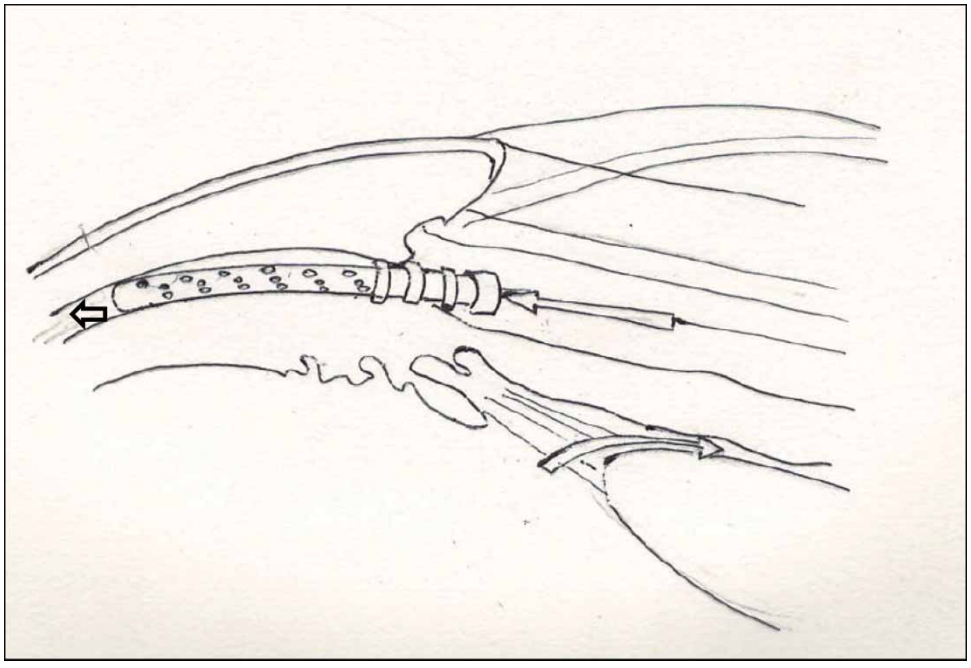

Figure 7. CYPASS implant.

A study by Batlle et al. on 23 eyes, with Innfocus microstent implantation with or without cataract surgery, has shown that over 80% patients had IOP <14 mm of Hg at 3 years with significant decrease in the number of anti-glaucoma medications.[28]

Conclusion

GDDs have sufficient evidence to prove their efficacy and safety profile as discussed above, but the same does not hold true for MIGS. Large multicenter randomized control trials to assess IOP lowering ability, operative ease and safety profile will further add insight into their use. Their high cost and lack of data in Indian eyes remain a major stumbling block for their use in surgical glaucoma management in our part of the world. However, with time and technological advances, it is to be seen if MIGS becomes the next trabeculectomy in glaucoma.

Acknowledgement: The authors would like to acknowledge the contribution of Dr. Debmalya Das, Associate Consultant, VR services, SN Kolkata towards the sketches and diagrams in the article.

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How to cite this article Ravi K., Srivastava P., Movdawalla M., Sen S. and Kedia P. Implants in glaucoma: a minor review, *Sci J Med & Vis Res Foun* 2017;**XXXV**:3–9.

Central serous chorioretinopathy (CSCR)

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Von Graefe first coined the term 'central recurrent retinitis' in 1866 for recurrent serous macular detachment.[1] In 1967, Gass explained the pathogenesis and clinical features and named it central serous choroidopathy (CSC).[2] CSCR typically affects middle-aged men and is characterized by serous neurosensory detachment (NSD) of retina at the posterior pole. Most cases are idiopathic and regress spontaneously within 4 months with good visual recovery.[3] However, a few suffer from persistent or recurrent serous macular detachment leading to progressive visual loss. Advances in indocyanine green angiography (ICGA) and optical coherence tomography (OCT) have led to greater understanding of CSCR. Modifications of photodynamic therapy (PDT) have changed CSCR management. Newer treatments in the form of anti-vascular endothelial growth factor (anti-VEGF) and mineralocorticoid receptor (MR) antagonists appear to be promising, but needs more scientific evidence before incorporating them into regular clinical practice.

Definition

CSCR is a disease characterized by localized NSD with or without focal pigment epithelial detachments (PEDs) and altered retinal pigment epithelium (RPE).[4] There are two forms, i.e. acute and chronic. The acute form usually resolves within 4 months, leaving mostly color discrimination defects in few patients. The chronic form is characterized by widespread tracks of RPE atrophy, showing reduced fundus autofluorescence (FAF).[5] Chronic form of the disease can also have irregular RPE detachments and long-standing intraretinal cystoid cavities.[6]

Pathogenesis

The pathophysiology of CSCR is thought to involve multiple etiologies and mechanisms that lead to widespread choroidal circulatory abnormalities and subsequent RPE disturbances. The hyperpermeability of choroid can be caused due to stasis, ischemia or inflammation, which is evident with the staining of the inner choroid in mid-phase ICGA.[7] These hyperpermeable choroidal vessels, hypothesized to cause increased tissue hydrostatic pressure, overcome the barrier function of the RPE and lead to the formation of PEDs.[8] Further increase in hydrostatic pressure in the choroid causes breach in the RPE and allows entry of fluid in the subretinal space.[9] Recently, aldosterone/MR pathway has been postulated in the pathogenesis of CSCR where intravitreal aldosterone had provoked vasodilation, thickening and leakage of choroidal vessels with accumulation of subretinal fluid (SRF) in pre-clinical animal models.[10]

Risk factors

Type-A personality, anti-psychotic medication and psychological stress are independent risk factors for CSCR and depression is associated with increased risk of recurrence.[11,12] Hypertension, obstructive sleep apnea and patients under systemic and local corticosteroid therapy are at higher risk of developing CSCR. It has been described after the administration of inhalational, intranasal, topical and periocular steroids.[13] Steroid-induced CSCR is an idiosyncratic response with less male predilection with more bilaterality.[14] CSCR has also been reported following kidney, bone marrow and heart transplantations.[15] Diseases, producing increased endogenous cortisol, such as Cushing's disease and pregnancy, especially the third trimester, increase the risk of having CSCR. Aqueous sample in patients with CSCR showed lower level of platelet-derived growth factor (PDGF), implicating PDGF in the pathogenesis of CSCR.[17] Gastroesophageal reflux and Helicobacter pylori infection have been separately reported to be associated with CSCR and treatment of the above conditions had shown to hasten the rate of SRF resolution.[18,19] Though the use of phosphodiesterase-5 inhibitors (sildenafil, tadalafil) had shown to cause CSCR, their discontinuation gives conflicting evidence regarding resolution of the disease in different studies.[20] So far, cases of familial CSCR had been reported in the literature, but no clear transmission pattern or genotype has been found to be associated with the disease.[21]

Clinical features

CSCR has become the most common vision-threatening disease after age-related macular degeneration (AMD), diabetic retinopathy and retinal vein occlusion. Men are mostly affected and a recent epidemiological data confer a higher mean age of affected patients, ranging between 39 and 51 years. The older patients show more bilaterality, female prevalence and increased risk of developing choroidal neovascularization (CNV). The incidence of CSCR has been noted to be more among Asians and Caucasians, but the disease behaves more aggressively among African-Americans.[22]

In acute presentation, patients usually complain of blurred vision, relative central scotoma, metamorphopsia, dyschromatopsia, hypermetropization and micropsia due to the SRF in the macular area (Figure 1). The hallmark of acute CSCR is a well-demarcated round- or oval-shaped area of NSD over the posterior pole with or without associated serous PEDs. In those patients, loss of foveal reflex provides a good hint toward diagnosis. Phagocytosed photoreceptor outer segments from the outer retina can sometimes appear as tiny yellow dots at the inner surface of RPE[23] (Figure 2). SRF in acute CSCR is usually transparent, but occasionally can become turbid due to subretinal fibrin deposition (Figure 3). This fibrin sometimes gets organized to cause subretinal fibrosis, leading to permanent drop in vision. Patients with CSCR can also present with inferior bullous exudative retinal detachment.[24] In chronic cases, there are areas of RPE changes and typical RPE atrophic tract in the inferior fundus due to the gravitational effect of long-standing SRF.[25] They can also present with chronic CME and secondary CNV.

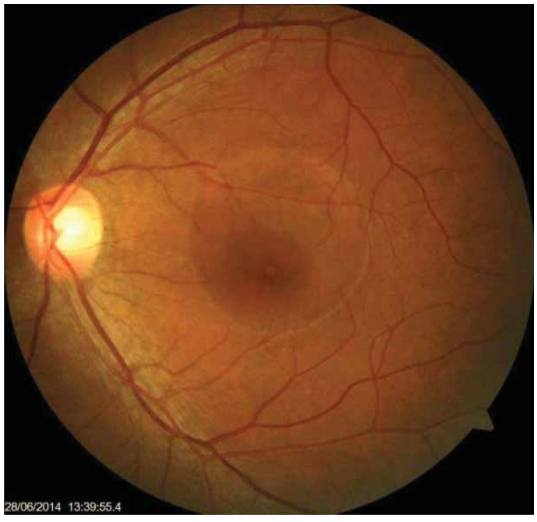

Figure 1. Color fundus of a young male patient with typical CSCR (central serous chorioretinopathy) presenting with circular neurosensory detachment at the posterior pole.

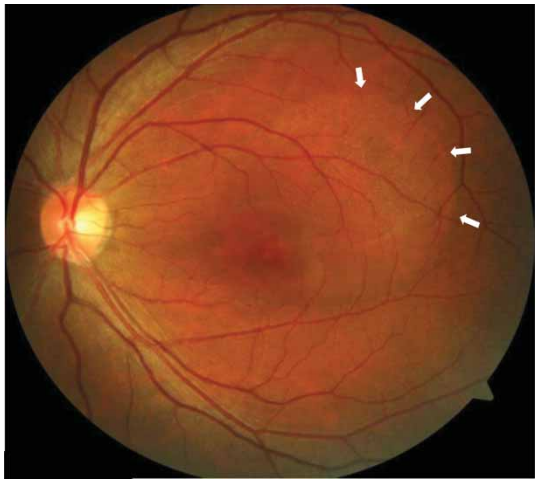

Figure 2. Color fundus image of CSCR showing neurosensory detachment of the parafoveal region, with yellow dots on the posterior surface of the detached retina (white arrow).

Differential diagnosis

Age-related macular degeneration

CSCR, CNV and PCV can be put together in a spectrum of 'pachychoroid' condition.[26] AMD is the most important differential diagnosis in CSCR patients aged 50 years or more. Secondary CNV, mostly type 2, can develop in patients with chronic CSCR during follow-up or after laser photocoagulation.

Polypoidal choroidal vasculopathy

Because of SRD, RPE alteration and choroidal hypermeability in ICG, sometimes it becomes difficult to distinguish PCV from chronic CSCR. The points, which favor the diagnosis of polyps, are subretinal hemorrhage, branching vascular network and leaking polyps in ICGA. OCT typically shows serosanguineous, notched or tall peaked PED and higher optical density of SRF.[27]

Optic disc PIT

Optic disc pits are focal excavations located in the temporal aspect of optic nerve head, creates a communication between the vitreous cavity, the subretinal space and to some extent the subarachnoid space. They produce chronic or recurrent SRD following schisis of inner retina in around half of the cases with variable intraretinal cystoid edema. Careful peripapillary examination and absence of leakage in FA remain diagnostic of optic disc pits.

Inflammatory diseases

Vogt–Koyanagi–Harada (VKH), a bilateral granulomatous panuveitic condition, often presents with multiple SRD mimicking CSCR. Apart from its systemic, neurological and dermatological signs, the presence of vitritis, increased choroidal thickening in ultrasound and pinpoint multifocal leaks on FA readily distinguish it from CSCR. Differentiation for this condition is of utmost importance as unlike CSCR systemic steroids are the mainstay of treatment here.

Autoimmune and vascular disorders

Autoimmune diseases, such as systemic lupus erythematous, polyarteritis nodosa and scleroderma, due to the disease processor during systemic steroid therapy can have NSD, complicating the outcome. Non-autoimmune conditions such as malignant hypertension, toxemia of pregnancy and disseminated intravascular coagulopathy can

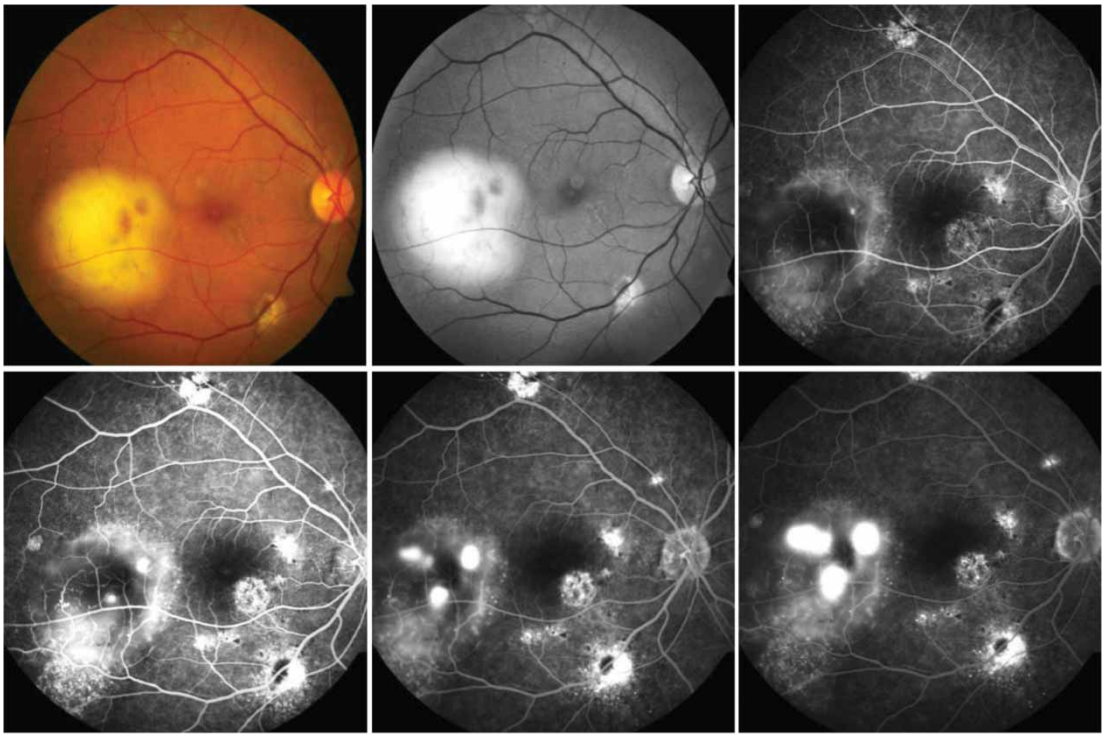

Figure 3. This patient of chronic CSCR shows organized subretinal fibrin with subretinal fluid temporal to macula. Fluorescein angiography (FA) shows areas of retinal pigment epithelial (RPE) atrophy with multiple pinpoint leaks adjoining the fibrin.

also present with a secondary NSD due to choroidal arterial occlusion.

Intraocular tumors

Various types of choroidal tumors including choroidal hemangioma, choroidal melanoma, choroidal osteoma and choroidal metastasis can cause exudative SRD mimicking CSCR. It is important to differentiate a malignant and potentially lethal condition from CSCR. Ultrasonography is useful in detecting and differentiating the nature of the tumor. In hemangiomas, ICGA shows classical 'wash-out' phenomenon in late phases of angiography and EDI-OCT shows increased caliber of large choroidal vessels in the tumor along with normal choriocapillaris.[28]

Ancillary testing

Optical coherence tomography

SD-OCT and recently enhanced-depth imaging and swept-source technologies have made the understanding of CSCR better by allowing better full-depth visualization of the neurosensory retina, RPE, choroid and choroidal vessels. Elongation of photoreceptor outer segments in the area of a macular SRD is a frequent OCT finding in CSCR. Erosion of photoreceptor outer segments at the site of leakage points toward a mechanical abrasion from an active flow through the RPE break. In active CSCR cases, a combination of SD-OCT with FA identified an RPE elevation or a PED at the leakage sites in most of the cases[29] (Figure 4). Localization of PEDs in the areas of dilated, large choroidal vessels and thickened choroid on SD-OCT with vascular hyperpermeability on ICGA suggest the role of choroidal flow deregulation in the pathogenesis of PED.[30] In chronic CSR, there can be a hyper-reflective content over Bruch membrane, creating a 'double layer sign' in OCT. The thinning of outer nuclear layer, cystoid macular degeneration (CMD) and disruption of the ellipsoid zone in OCT are associated with poorer visual outcome.

Fundus autofluorescence

FAF mostly originates from the RPE lipofuscin and reflects RPE health. Focal areas of hypoautofluorescence corresponding to the leakage points in acute CSCR support the hypothesis of a focal RPE defect[31] (Figure 5). The SRD in acute CSCR also shows hypoautofluorescence due to masking effect of SRF and as the disease progresses to become persistent or chronic, there is increasing hyperautofluorescence due to the accumulation of non-shed fluorophores. The pattern of this change in FAF has been shown to correlate with visual acuity.[32] FAF can be pathognomonic in chronic CSCR by multiple hypoautofluorescent 'gravitational tracks' with thin border of surrounding hyperautofluorescence (Figure 6).

Fluorescein angiography

In acute CSCR, classically there are two leakage patterns in fluorescein angiography (FA). First one,

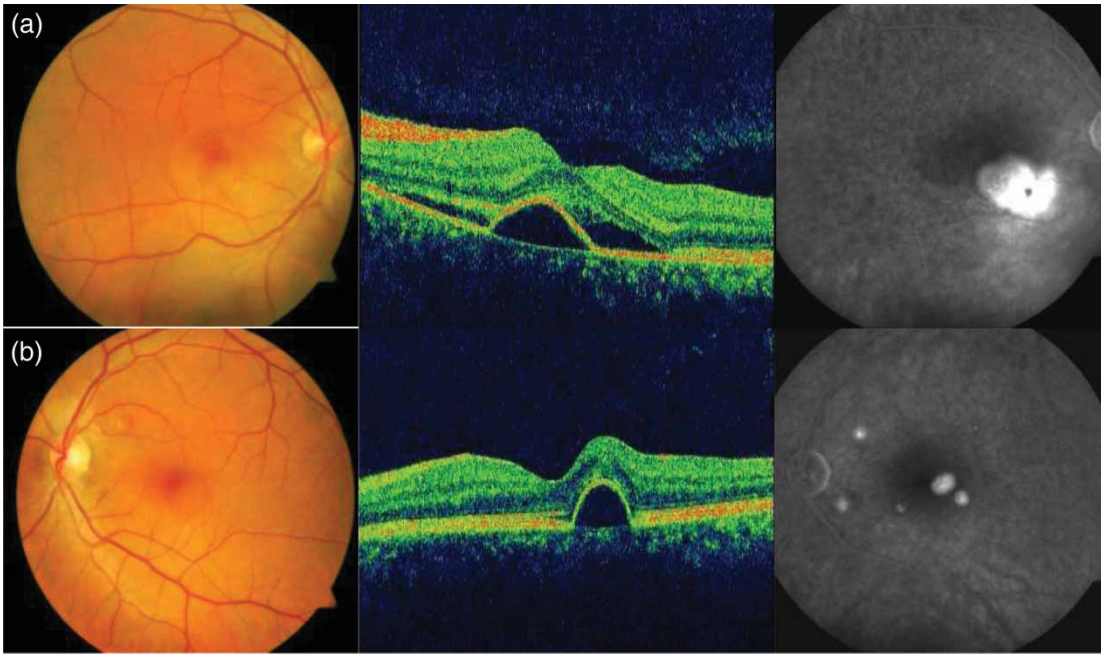

Figure 4. This 45-year-old male presented with a right eye relative scotoma for 3 weeks. (a) Clinical examination showed subretinal fluid (SRF) together with a pigment epithelium detachment (PED) in the macula. Optical coherence tomography (OCT) shows PED with SRF. FA shows initial pooling of dye in the PED followed by leakage; (b) Left eye shows PED in fundus photo and OCT, with pooling of dye in FA.

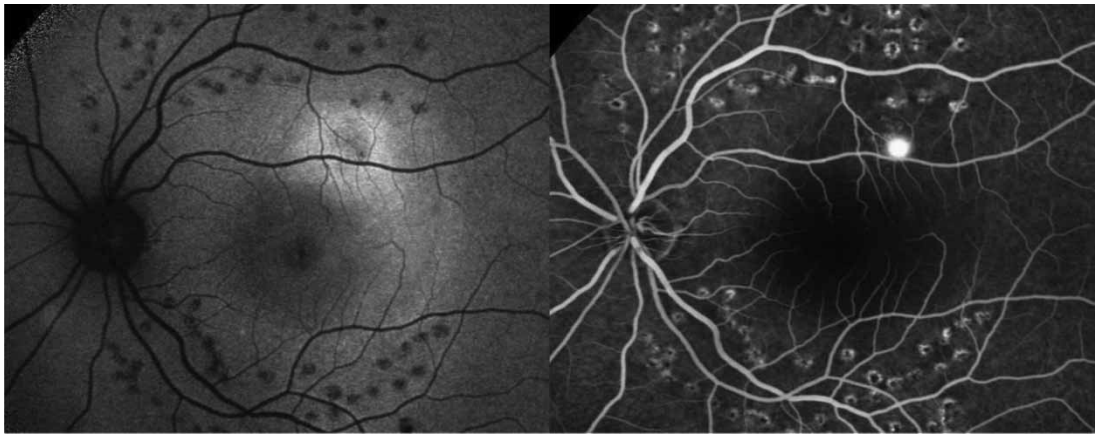

Figure 5. This 38-year-old man was treated with ill-defined laser for submacular fluid before he presented to us with active pinpoint leak in FA of the left eye. Fundus autofluorescence (FAF) showed increased autofluorescence suggestive of long standing subretinal fluid with point hypoautofluorescence (black arrow) corresponding with the leak in FA.

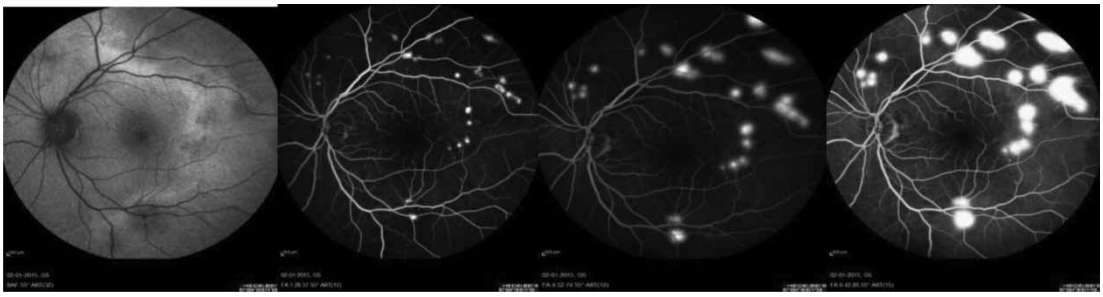

Figure 6. Left eye of a post renal transplant patient on long-term systemic steroids showing multifocal pinpoint leaks in CSCR. Note the hypoautofluorescence patches (white arrow) with surrounding hyperautofluorescence (black arrow) suggestive of chronic disease.

the leakage starts as a pinpoint in early phase and then concentrically enlarges in the late phase to appear like an inkblot (Figure 7). Secondly, the leak increases from a pinpoint gradually to ascend and expand like a mushroom cloud or umbrella in the late phase to cause a smoke stack, which is seen in 10–15% of patients (Figure 8).[33] It is caused by an increased protein concentration in the SRF. Chronic CSCR shows diffuse RPE window defect and patchy hyperfluorescence due to RPE atrophy. FA is also useful in differentiating CSC from other diagnoses, such as CNV and VKH, and helps in diagnosing unnoticed extramacular leak in affected or fellow eye.

Indocyanine green angiography

ICGA helps to demonstrate the choroidal vascular changes, which contributes in the disease process and can act as a guide to treatment with PDT. ICGA in CSCR shows delay in choroidal filling in the early phase with hypofluorescent areas resulting from non-perfusion of choriocapillaris. This leads to choroidal venous dilatation and choroidal hyperpermeability with the zone of hyperfluorescence in the mid-phase (Figure 9). In the late phase, there is either washout or persistent hyperfluorescence.

Multifocal electroretinography

Multifocal electroretinography (mfERG) can indicate more widespread retinal dysfunction in CSCR than appreciated on clinical examination. First-order kernel mfERG amplitudes are reduced in the center and the second-order responses predominantly gets reduced for the peripheral retina.[34]

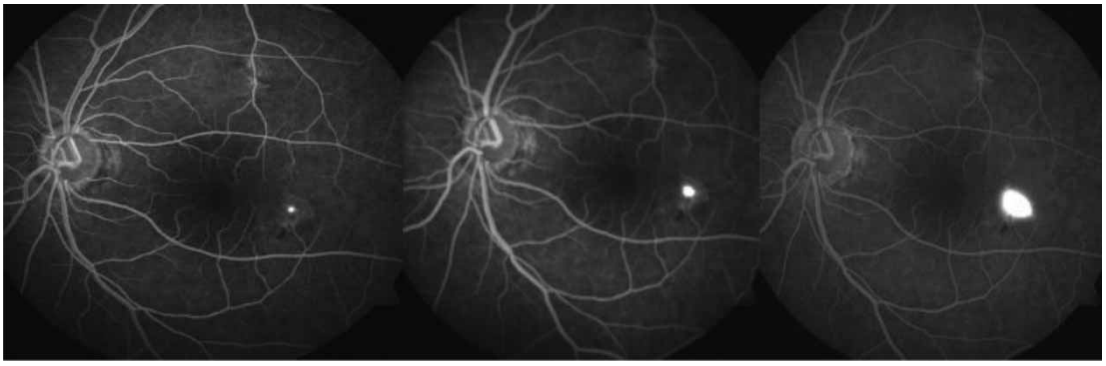

Figure 7. Ink blot leak: the hyperfluorescence starts as a pinpoint and then enlarges circumferentially to produce intense hyperfluorescence in late phase similar to the appearance of drop of ink into a piece of paper.

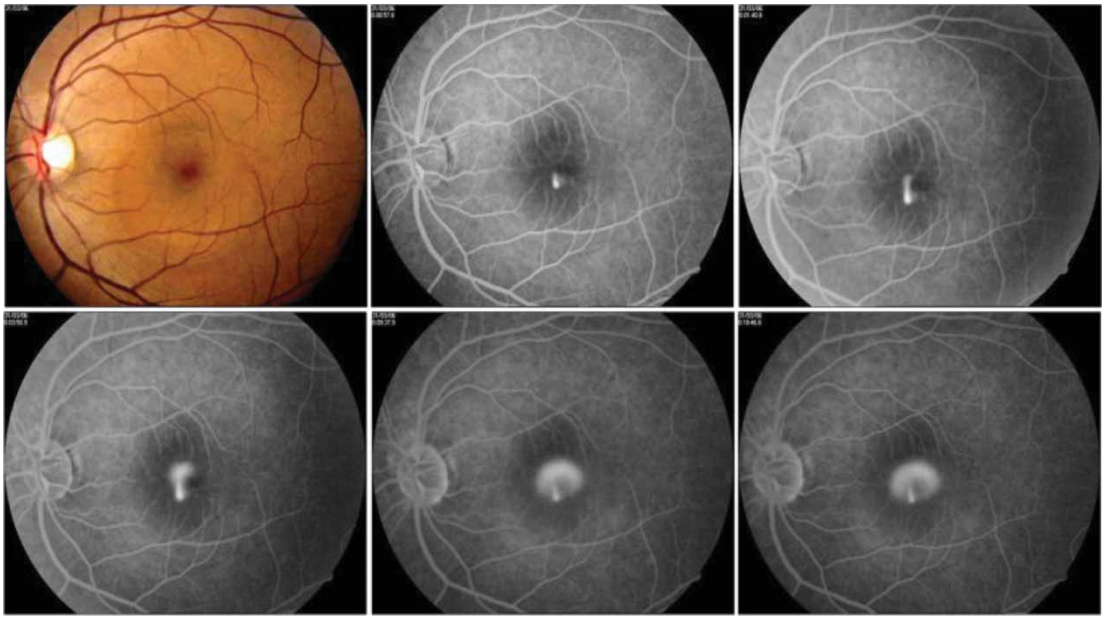

Figure 8. Smoke stack appearance: the hyperfluorescent spot starts as a pinpoint and then diffuses upward and laterally to give a mushroom cloud or umbrella-like appearance.

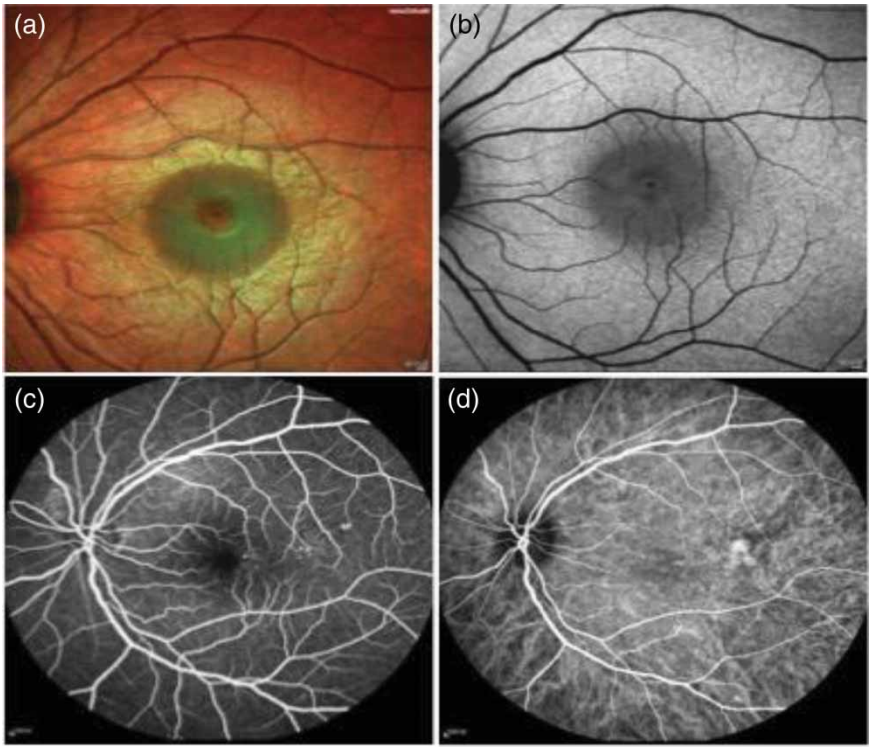

Figure 9. Confocal scanning laser ophthalmoscope brings more imaging mode to scan simultaneously and provides more information on macular changes in central serous chorioretinopathy patients. (a) Multicolor imaging showing dome shaped neurosensory detachment (NSD); (b) Hypoautofluorescence of NSD; (c) Point leak in fluorescein angiography; (d) Dilated choroidal vessels in Indocyanine green angiography.

Natural history

Most acute CSCR patients show spontaneous visual recovery within 4 months. However, some progresses to chronic or recurrent disease, which lead to areas of RPE atrophy and pigmentation in the macular area with subsequent visual loss. Up to 50% patients of CSCR develop recurrence within the first year of presentation.[35] A small proportion of patients develop irreversible visual loss due to gross RPE atrophy, subretinal fibrosis, CNV or CMD. Adaptive optics have shown reduced cone density in eyes with resolved CSCR with 20/20 visual acuity compared with controls.[36] This explains the reason for having residual symptoms such as metamorphopsia, scotoma, reduced contrast and color sensitivity even in well-recovered patients after acute CSCR.

Treatment

Observation with or without removal of risk factors

Acute CSCR is a self-limiting disease, with re-attachment of the NSD occurring within 4 months in most cases. Because of this favorable natural history, observation has been considered as an appropriate first-line approach. As high levels of endogenous or exogenous corticosteroids have been implicated in the etiology of CSCR, discontinuation of steroid in any form is advocated. In CSCR, recurrences occur in ~20–50% of patients by 1 year and chronic NSD often leads to permanent loss of visual functions. Lifestyle modification, treatment of sleep apnea and psychosocial therapies has also helped in treating patients prone to have CSCR.[37] So, though observation is the standard initial management in most cases of CSCR, active treatment should be initiated when symptoms persist for more than 3 months.[38] Treatment most of the time speeds up visual recovery, but no treatment could maximize the final visual gain. Early treatment is recommended in cases where rapid recovery of vision is required for vocational reasons, and also where untreated CSCR had previously resulted in a poor visual outcome in the fellow eye.[39]

Argon laser photocoagulation and micropulsed diode laser

Laser photocoagulation, when applied to the RPE leakage points, causes direct thermal sealing effect on the focal RPE defects and favors stimulation of surrounding RPE cells. This hastens the resolution of NSD, but rarely alters the final visual outcome and rate of recurrences. This may be because zonal hyperperfusion and hyperpermeability of the choriocapillaris, the presumptive pathophysiology in CSCR, are not amenable to laser therapy. This treatment method can have adverse effects

such as permanent scotoma, enlargement of RPE scar, secondary laser-induced CNV, and, rarely, inadvertent foveal burn.[40] Thermal laser photocoagulation is now indicated in the management of CSCR with discrete, solitary or multiple extrafoveal leaking points with persistent NSDs (Figure 10). Subfoveal or juxtafoveal leak and bullous exudative RD are better managed by safety-enhanced PDT rather than argon laser.

There has been a revival of interest in using micropulse diode laser, instead of the conventional argon laser photocoagulation, to treat CSCR. The 810 nm micropulsed diode emissions enable subthreshold therapy to the RPE and choroid without a visible burn, reducing the risk of structural and functional retinal damage. In a series of 30 patients, the diode group had faster visual recovery and better final contrast sensitivity than the argon laser group without any persistent scotoma.[41] Nevertheless, the efficacy and safety of micropulse diode laser in chronic CSCR is not proved and randomized controlled trials (RCT) are necessary to fully substantiate the treatment efficacy.

Photodynamic therapy

There are reports showing favorable visual outcomes of ICG-guided PDT to treat CSCR.[42] The analyzed mechanism of action of PDT causing narrowing of choriocapillaris, choroidal hypoperfusion and choroidal vascular remodeling supports its treatment in CSCR, which is primarily a pachychoroid disorder and success of PDT also depends upon the degree of hypermeability on ICGA[43] (Figure 11). The potential hazards of conventional PDT in AMD, i.e. RPE atrophy, choriocapillaris ischemia, and secondary CNV has made clinicians modify PDT to get the safest form to reduce iatrogenic risks.[44]

Conventional PDT

In nAMD following standard PDT, there is normalization in calibers of the congested choroidal vasculature with a decrease in the extravascular leakage in few pilot studies.[45] In a recent meta-analysis all the 10 studies that met a standard STROBE criteria showed significant improvement in BCVA after conventional PDT treatment in CSCR.[46] However, the possible post-treatment visual loss and potential choroidal ischemia have restricted clinicians from the widespread application of standard dose PDT in CSCR patients.[47]

Safety-enhanced PDT with reduced verteporfin dosage

To reduce the potential iatrogenic hazards of PDT, lot of studies in CSCR have tried to modify the dosage of verteporfin or reduce the fluence of laser to minimize its side-effects.[48] The aim of these studies was to lower the risk of retinochoroidal complications of PDT without compromising its ability to remodel the vascular bed.[49] In one study, verteporfin at a dose of 3 mg/m^2 instead of 6 mg/m^2 showed a complete resolution of the fluid in 79.5% and 94.9% of the half-dose PDT-treated eyes at 1 and 12 months, respectively. Another study reporting the long-term results of half-dose PDT in chronic CSCR showed a dry macula at 12 months for all 27 eyes.[51] Eyes without PED, duration of CSCR for <6 months

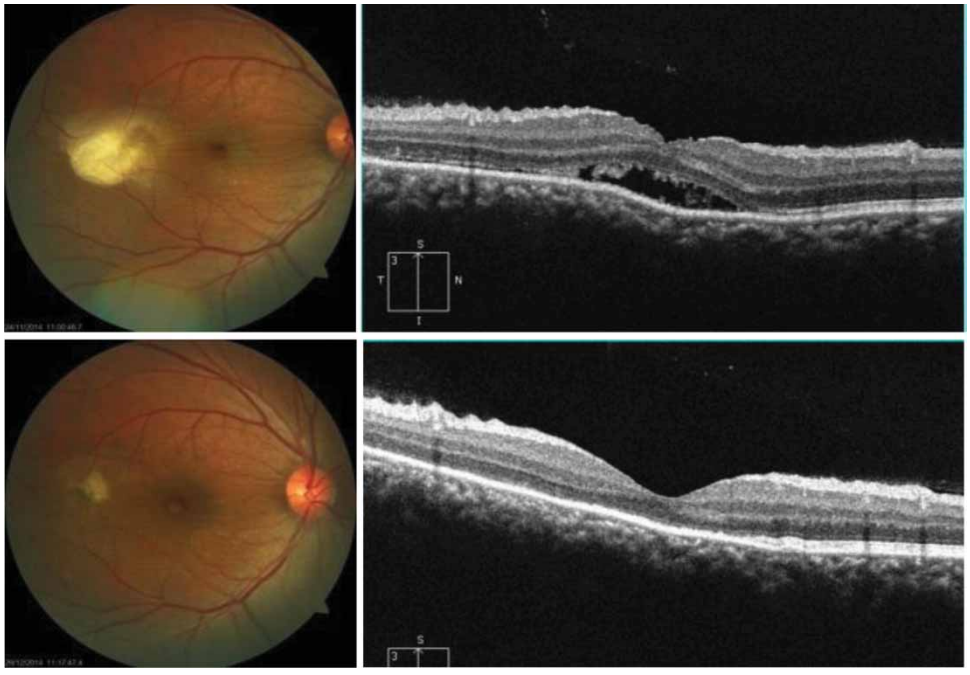

Figure 10. Resolved subretinal fibrin and subretinal fluid in long standing CSCR following focal laser to extrafoveal leak.

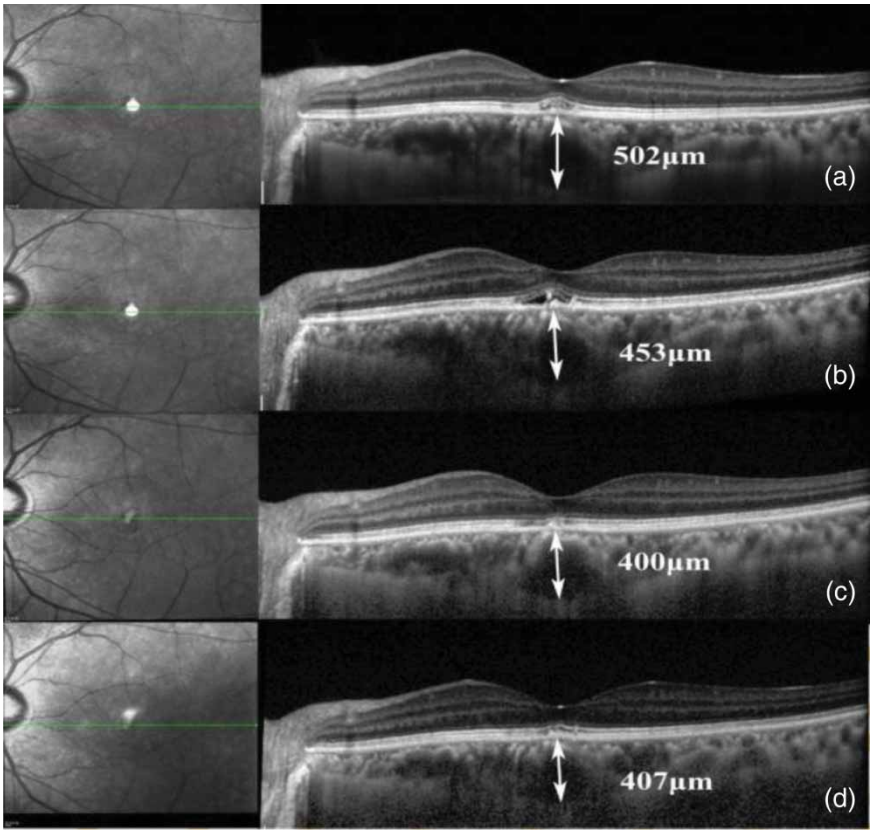

Figure 11. This 42-year-old male presented with a left eye persistent central scotoma for more than four months. (a) OCT shows mild NSD of the macula (b), (c) and (d) shows resolved SRF after photodynamic treatment (PDT) and restoration of choroidal thickness.

and age younger than 45 years were having better visual improvement. Though studies have tried lower doses of verteporfin, it is the 3 mg group, which had the best results in terms of BCVA and CFT reduction at 6-month follow-up period.[50,51] The role and limitations of PDT in CSCR should be emphasized clearly to the patients, as most of them will have good visual potential and there has been sporadic observations of transient impairment of multifocal ERG and development of juxtafoveal CNV with half-dose PDT.[52]

Safety-enhanced PDT with reduced laser fluence

There are reports of improved efficacy and safety profiles of low fluence PDT in the management of chronic CSCR. The RCT by Bae at al compared half-fluence PDT with intravitreal ranibizumab to treat 16 eyes of chronic CSCR patients and found complete resolution of SRF at 6 months in 75% of the half-fluence group compared with 25% in the ranibizumab group.[53] The PDT group also showed significant CFT reduction at the end of 9 months. In other two studies, half-fluence group had better BCVA compared with the standard group at the end of 12 months.[54,55]

A recent retrospective study with 56 patients of chronic CSCR treated with half-dose and half-fluence PDT divided in equal numbers showed a complete resolution of SRF in 19 (61.3%) and 26 (83.9%) half-fluence-treated eyes at 1 and 12 months. The corresponding values were 25 (86.2%) and 29 (100%) in the half-dose-treated eyes without any statistical difference in BCVA in between the groups with overall 15 and 5 recurrences in the groups, respectively.[56] The meta-analysis by Erikitola et al. also concluded that 100% of the studies in reduced fluence group and 42.9% studies of half dose group had recurrence within 1-year-follow-up period.[57] So, there can be a cost-effective, superior role of half-dose PDT compared with half-fluence in keeping the macula dry for longer period.

Anti-vascular growth factor injections

Although CSCR is not associated with increased anti-vascular growth factor (VEGF) aqueous or plasma levels, anti-VEGF therapy was proposed in CSCR to reduce the choroidal hyperpermeability.[58] A limited number of interventional case series had reported beneficial effects of bevacizumab and aflibercept in terms of visual acuity improvement and SRF reduction without significant complications.[59,60] Out of 2 RCTs, the first one showed no difference in terms of visual acuity gain, central

foveal thickness reduction or duration of SRD between bevacizumab and control groups.[61] The second study, by Arevalo et al., showed superior effect of half-fluence PDT to ranibizumab.[62] In a recent meta-analysis, there was no significant improvement with intravitreal bevacizumab, compared with observation, PDT or laser photocoagulation in terms of final visual acuity and central macular thickness.[63]

Transpupillary thermotherapy

Transpupillary thermotherapy (TTT) is a long-pulse, low-energy, 810-nm near infrared laser, which causes choroidal vascular thrombosis. There are studies in which TTT had proved to be safe in focal juxtafoveal leakage instead of thermal laser.[64] Wei and colleagues were the first to report complete resolution of SRF 4 weeks after TTT in a case of chronic CSC with no observed visual improvement.[65] In a large, non-randomized, prospective cohort study, with unmatched control of 15 eyes, 96% of the 25 TTT-treated experienced complete resolution of NSD and leakage on FA at 3 months. Vision improved significantly in 92% of cases compared with 33% in the control group.[66] Still, well-controlled, properly matched RCTs are warranted to find the precise role and efficacy of TTT in the management of CSCR.

Anticorticosteroid therapy

Patients with CSCR commonly have endogenous hypercortisolism, resulting in trials of medications targeting cortisol pathways.[67] There are anecdotal reports with ketoconazole, mifepristone (RU486), finasteride, rifampin in the treatment of CSCR without any long-term acceptable benefit.[68–71] MR activation in choroid vessels had been shown to be involved in the pathogenesis of CSCR.[72] In a recent study, 17 eyes of 13 patients with chronic CSCR had improvement of Log MAR visual acuity from 0.42 at baseline to 0.29 and decreased CFT from 339.5 microns to 270 microns at 6 months when treated with 25 and 50 mg of oral eplerenone per day.[73] Though these studies throw a lot of hope for medical management of CSCR, there is a need for extensive research with longer follow-up before they can be accepted in primary oral therapy of CSCR.

Conclusion

The pathophysiology of CSCR remains multifactorial. The role of choroidal vascular hyperpermeability related to the deranged mineralocorticoid pathway sounds appealing, but needs more scientific support. Though the natural history of CSCR has been thought to be favorable, frequent reports of significant anatomical and functional loss even from a mild course of the disease and the risk of frequent recurrences require early effective treatment. Newer imaging techniques and treatment options have opened new horizons in the management of the disease. In the treatment 'Safety-enhanced' PDT using lower doses, oral corticosteroid antagonists, intravitreal anti-VEGF therapy and micropulsed diode laser do merit further research.

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How to cite this article Das S. and Das D. Central serous chorioretinopathy (CSCR), *Sci J Med & Vis Res Foun* 2017; **XXXV**:10–20.

Synopsis of TFOS (tear film and ocular surface society) DEWS (dry eye work shop) II Report, 2017

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The TFOS DEWS II report was published on 21 July 2017, revising the definition of dry eye disease (DED) and giving new insight into the way the disease is diagnosed and treated. The initial focus on DED began with the publication of the report of the National Eye Institute/Industry Workshop on Clinical Trials in Dry Eye in 1995. It was the first formal attempt to define and classify DED, in addition to reviewing its management, treatment, and the design of clinical trials. Now, 10 years later, progress continues with the publication of this 2017 Report of the TFOS (tear film and ocular surface society) International Dry Eye Workshop II (TFOS DEWS II). This workshop, a 2-year effort for 12 subcommittees made up of 150 experts from 23 countries, has led to the creation and publication of this substantial report (almost 400 pages).

The report has been divided into the following subheadings:

1 Definition and classification.
2 Sex, gender and hormones.
3 Epidemiology.
4 Tear film.
5 Pain and sensation.
6 Pathophysiology.
7 Iatrogenic dry eye.
8 Diagnostic methodology.
9 Management and therapy.
10 Clinical trial design.
11 Public awareness and education.
12 Industry liaison.

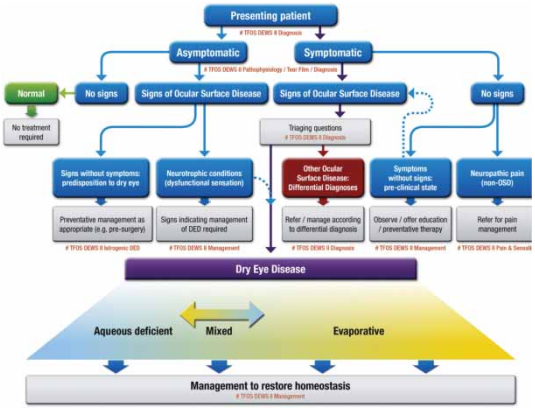
Definition and Classification

The revised definition of DED acknowledged the significant role of inflammation and hyperosmolarity within the DED pathway.

'Dry eye is a multifactorial disease of the tears and ocular surface that results in symptoms of discomfort, visual disturbance, and tear film instability with potential damage to the ocular surface. It is accompanied by increased osmolarity of the tear film and inflammation of the ocular surface'.

The loss of tear film homeostasis can arise from a multitude of factors that encompass eyelid and blink abnormalities, in addition to ocular surface or tear component deficiencies. These changes can induce focal or global tear film instability and tear hyperosmolarity in response to excessive evaporation from the ocular surface, and are regarded as significant entry points that contribute to the pathogenesis and perpetuation of a cycle of events, or 'Vicious Circle', in DED. Mounting evidence of the potential role of neurosensory abnormalities in the understanding and management of DED. Neuropathic pain occurs due to overt damage within the somatosensory nervous system, distinguishing it from DED. Nociceptive pain occurs in response to local tissue damage.

Classification



Sex, gender and hormones

Female sex is an established risk factor for DED-related autoimmune diseases such as Sjogren syndrome. Female sex is also among the most widely studied and consistently identified risk factors for DED throughout the world.Sex, gender and hormones exert significant influence on the ocular surface and adnexa, and play a significant role in the pathogenesis of aqueous-deficient and evaporative DED. However, further studies are required to clarify the precise nature, extent, and mechanisms of these sex, endocrine and gender effects on the eye in health and disease. A better understanding how these factors influence the pathophysiology of DED may result in improved, more tailored and appropriate options for the treatment of DED.

Epidemiology

The epidemiology of DED continues to be a challenge due to the lack of a standardized worldwide definition. This has resulted in epidemiologic studies using different diagnostic criteria based on symptoms and signs and self-reported diagnoses. A meta-analysis of published prevalence data estimated the impact of age and sex. Global mapping of prevalence was undertaken. The prevalence of DED ranged from 5 to 50%. The prevalence of signs was higher and more variable than symptoms. The meta-analysis confirmed that prevalence increases with age; however, signs showed a greater increase per decade than symptoms. Women have a higher prevalence of DED than men, although differences become significant only with age. Risk factors were categorized as modifiable/non-modifiable, and as consistent, probable or inconclusive. Asian ethnicity was a mostly consistent risk factor. While disease definitions vary between studies, the prevalence of disease increases with age and females are more frequently affected, with the exception of MGD where sex effects are more equivocal. Limited studies have been carried out in youth and there remains a need for studies in populations below 40 years of age. Prevalence appears to be higher in Asian than in Caucasian populations, although studies have not been conducted in major geographic regions. There are limited studies of both disease incidence and the natural history of treated and untreated disease, both of which remain future needs for this field.

Tear film

Clinically, DED is characterized by loss of tear volume, more rapid breakup of the tear film and increased evaporation of tears from the ocular surface. The tear film is composed of many substances including lipids, proteins, mucins and electrolytes. All of these contribute to the integrity of the tear film, but exactly how they interact is still an area of active research. Tear film osmolarity increases in DED. Changes to other components such as proteins and mucins can be used as biomarkers for DED.

DED implies major changes to the tear film structure and function, which are associated with this disease. Historically, the tear film has been viewed as a 3-layer 'sandwich' composed of distinct lipid, aqueous and mucin layers. Evidence continues to support the more contemporary two-phase model of the tear film, with a lipid layer overlying a mucoaqueous phase. While it may be that the whole tear film (lipids, mucins, proteins and salts) prevents tear film evaporation and collapse, additional studies are needed to confirm or deny this concept. While tear proteins are reported to change in DED, no definitive set of proteins or changes in protein levels have been validated to aid in diagnosis. There is a need to further characterize the biochemistry of the tear film to identify new markers that can be used to diagnose, and perhaps predict and treat, DED. There is also a need for ways to dynamically measure tear film osmolarity and markers of inflammation over the whole ocular surface.

Pain and sensation

Nociceptive pain occurs in response to actual or threatened damage to tissues, while neuropathic pain occurs due to a lesion within the somatosensory nervous system. Cold thermoreceptors continuously discharge nerve impulses at the normal ocular surface temperature, responding to warming or cooling and to osmolarity increases, likely contributing to reflex control of basal tear production and blinking.

The most prominent nerve disturbance is with the cold thermoreceptors, suggesting that dryness-induced nerve damage dominates over inflammation, again emphasizing a need to focus on possible treatment strategies involving cold thermoreceptors.

Long-term inflammation and nerve injury alter gene expression of ion channels and receptors at terminals and cell bodies of trigeminal ganglion and brainstem neurons, changing their excitability, connectivity and impulse firing. Perpetuation of molecular, structural and functional disturbances in ocular sensory pathways ultimately leads to dysesthesias and neuropathic pain referred to the eye surface. Pain can be assessed with a variety of questionnaires, while the status of corneal nerves is evaluated with esthesiometry and within vivo confocal microscopy.

Pathophysiology

Its central mechanism is evaporative water loss leading to hyperosmolar tissue damage which, either directly or by inducing inflammation, causes a loss of both epithelial and goblet cells. The consequent decrease in surface wettability leads to early tear film breakup and amplifies hyperosmolarity via a Vicious Circle. Pain in dry eye is caused by tear hyperosmolarity, loss of lubrication, inflammatory mediators and neurosensory factors, while visual symptoms arise from tear and ocular surface irregularity. Increased friction targets damage to the lids and ocular surface, resulting in characteristic punctate epithelial keratitis, superior limbic keratoconjunctivitis, filamentary keratitis, lid parallel conjunctival folds, and lid wiper epitheliopathy. Hybrid DED, with features of both aqueous deficiency and increased evaporation, is common and efforts should be made to determine the relative contribution of each form to the total picture.

Inflammation of the ocular surface can cause inhibition of lacrimal secretion and loss of

epithelial barrier function at the ocular surface. Tear film breakup, leading to localized hyperosmolarity, can result in ocular surface damage either directly or through the cascade of inflammation that it initiates. Improved understanding of the role of subclinical inflammation in the early stages of DED also warrants further study.

Iatrogenic dry eye

Dry eye can be caused by a variety of iatrogenic interventions. The increasing number of patients looking for eye care or cosmetic procedures involving the eyes, together with a better understanding of the pathophysiological mechanisms of DED, have led to the need for a specific report about iatrogenic dry eye within the TFOS DEWS II. Topical medications can cause DED due to their allergic, toxic and immuno-inflammatory effects on the ocular surface. Preservatives, such as benzalkonium chloride, may further aggravate DED. A variety of systemic drugs can also induce DED secondary to multiple mechanisms. Moreover, the use of contact lens induces or is associated with DED. However, one of the most emblematic situations is DED caused by surgical procedures such as corneal refractive surgery as in laser-assisted in situ keratomileusis (LASIK) and keratoplasty due to the mechanisms intrinsic to the procedure (i.e. corneal nerve cutting) or even by the use of postoperative topical drugs. Cataract surgery, lid surgeries, botulinum toxin application and cosmetic procedures are also considered risk factors to iatrogenic DED, which can cause patient dissatisfaction, visual disturbance and poor surgical outcomes. Future recommendations for research include conducting further epidemiologic studies to better define risk factors, creating less toxic medications and preservatives, devising less invasive ophthalmic procedures, and developing strategies for the detection of early DED prior to surgical interventions.

Diagnostic methodology

The sensitivity and specificity of tests for the diagnosis of DED are dependent on the inclusion criteria for DED. If DED is suspected, screening with questionnaire such as the 5-item dry eye questionnaire (DEQ-5) or the ocular surface disease index (OSDI), will help to decide for further evaluation with tear break-up time (non-invasive methods preferred), tear film osmolarity determination, and ocular surface staining (that includes the cornea, conjunctiva and lid margin) with fluorescein and lissamine green. Identification of a disruption in tear film homeostasis with these tests allows a diagnosis of dry eye to be made. Other tests, such as meibography, lipid layer interferometry, evaporation and tear volume measurements can help clarify where the individual with DED falls on the evaporative and aqueous deficient DED subtype classification spectrum and promote the selection of appropriate therapeutic interventions.

Management and therapy

Restoration of tear film homeostasis is the ultimate goal in the management of DED, and this involves breaking the vicious circle of the disease. Management of DED is often difficult and challenging. In summary, the management of DED remains something of an art, not easily lending itself to a rigid, evidence-based algorithm that accommodates all patients with dry eye symptoms and signs. All eye care providers who treat DED must exercise their clinical expertise to judge the significance of each of the varied pathogenic processes (aqueous deficiency, MGD, inflammation, etc.) that may manifest similar subjective complaints and similar signs of disrupted ocular surface homeostasis.

Available options to treat DED have increased dramatically. The last decade has seen new developments in topical lubricants (particularly lipid-containing drops), autologous serum options, and punctual plug designs. There have been many new developments to help with lid hygiene, as well as the availability of new treatments for demodex infestation, devices to manage MGD, and rigid gas permeable scleral lenses. In addition to the various options to manage the inflammatory processes associated with DED that have come to market, the impact of dietary modifications (particularly the value of essential fatty acid supplements) is better understood and the potential value of various complementary medicines has come under discussion. While the prescribing of lubricants remains the mainstay of early treatment for DED, of particular value would be studies comparing the efficacy of products with and without lipids in evaporative and in aqueous-deficient DED. Studies to determine the impact of various formulations on tear film osmolarity and the duration of treatment required for changes to occur are also worthy of consideration, particularly for lubricants expected to influence tear film stability.

Clinical trials

In order to improve the quality of clinical trials going forward, to optimize resources, and increase the opportunity for novel therapeutics for patients with DED, the TFOS DEWS II Clinical Trials subcommittee has the following recommendations. First, that studies be conducted consistent with good clinical practice (GCP). This involves using GMP-quality clinical trial material. While this may be a daunting task, clinical trialists should consult colleagues and drug development experts who are familiar with this system of controls. This includes appropriate protections for the study subjects. GCP also requires compliance with

appropriate regulatory requirements in the jurisdiction of study conduct, and may require additional regulatory filings if the investigational shipment is prepared and shipped from another state or country Design, treatments, and sample size need to align with the investigational treatment, the objectives of the study, and the phase of development.

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How to cite this article Barbhaya R.D. and Bhargava M. Synopsis of TFOS (tear film and ocular surface society) DEWS (dry eye work shop) II Report, 2017, *Sci J Med & Vis Res Foun* 2017;**XXXV**:21–24.

Resolution of cystoid macular edema by topical dorzolamide in a case of central serous chorioretinopathy: a case report

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Introduction

Central serous chorioretinopathy (CSCR) is a posterior segment disease characterized by localized serous detachments of the neurosensory retina often associated with focal detachments of an altered retinal pigment epithelium (RPE) and having multifactorial etiology and complex pathogenesis.[1] In chronic CSCR, intraretinal cysts and cystoid macular edema (CME) can form. Both topical and systemic carbonic anhydrase inhibitors (CAIs) have been tried as means of treatment of both CSCR[2] and CME[3]caused by CSCR. We present a case of chronic CSCR in which the resolution of CME occurred with topical CAI.

Case report

A 45-year-old male presented with painless, gradual and progressive diminution of vision of both eyes for 2 years. On examination, his best corrected distant visual acuity (BCVA) was 20/120 in the right eye and 20/200 in the left eye. Posterior segment examination revealed extensive areas of RPE atrophy in both eyes with the presence of subretinal fluid (SRF) in the right eye. Fundus fluorescein angiography corroborated the clinical findings showing extensive areas of RPE atrophy in both eyes with ink blot leak in the right eye. Optical coherence tomography (OCT) of the right eye showed sub- and juxtafoveal RPE atrophy with SRF and the left eye revealed juxtafoveal cystic changes with RPE atrophy (Figure 1). Focal laser to the area of leak in the right eye was done and the patient was asked to review after 2 months. On his next visit, BCVA of right eye increased to 20/80 with no change in the left eye BCVA. OCT showed resolution of SRF in the right eye and an increase in CME and schisis in the left eye (Figure 2). The patient was started on topical dorzolamide (2%) thrice a day in his left eye. On his next visit, after 1 month, OCT showed complete resolution of CME though the visual acuity remained the same (Figure 3).

Discussion

One of the characteristics of acute CSCR is that despite SRF, the morphology of retinal layers generally remains unchanged. However, in chronic cases, intraretinal cysts and CME may develop. These may disappear or fluctuate slowly over time, suggesting fluid passage through a compromised RPE which contributes to their formation.[1] However, in our case, CME resolution occurred within a month, suggesting a possible role of dorzolamide in its resolution.

Investigations of the ability of CAIs to enhance SRF absorption based on animal models have shown acidification of the subretinal space, a decrease in standing potential, and an increase in retinal adhesiveness. This acidification of the subretinal space is responsible for the increased fluid resorption from the retina through the RPE to the choroid resulting from modulation of carbonic anhydrase IV in RPE. RPE loses normal polarity in the presence of macular edema, and treatment with CAIs re-establishes normal polarization in RPE.[4–6] Another possible explanation for the effect of CAIs on inflammation-related macular edema is its ability to inhibit γ glutamyl trans

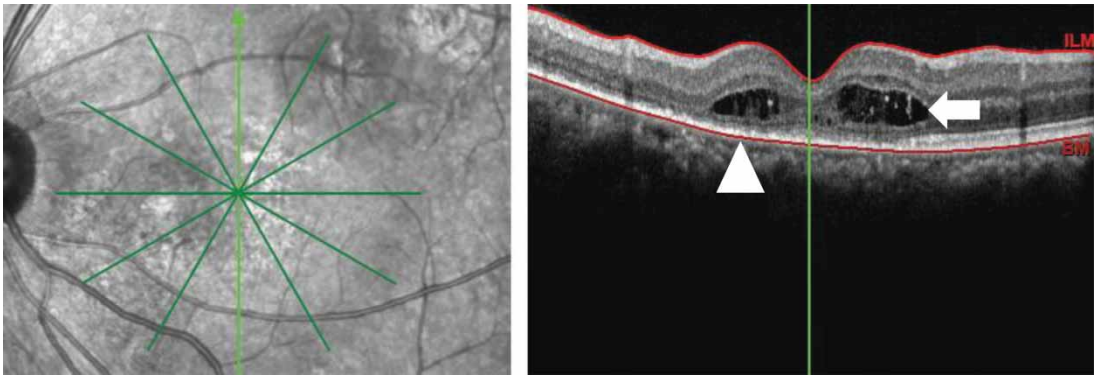

Figure 1. OCT through the fovea of the left eye showed Juxtafoveal cystic changes (white arrow) with areas of RPE atrophy (white arrow head).

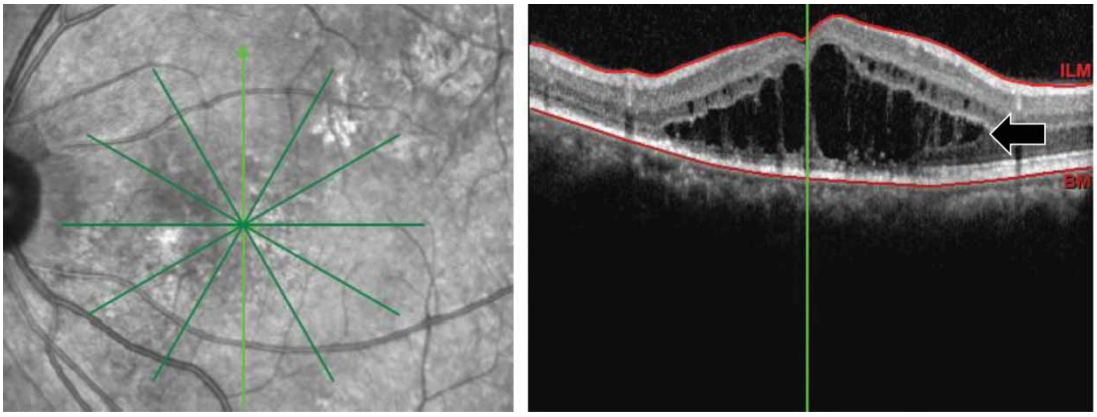

Figure 2. OCT showed increased cystic and schitic changes (black arrow with white border).

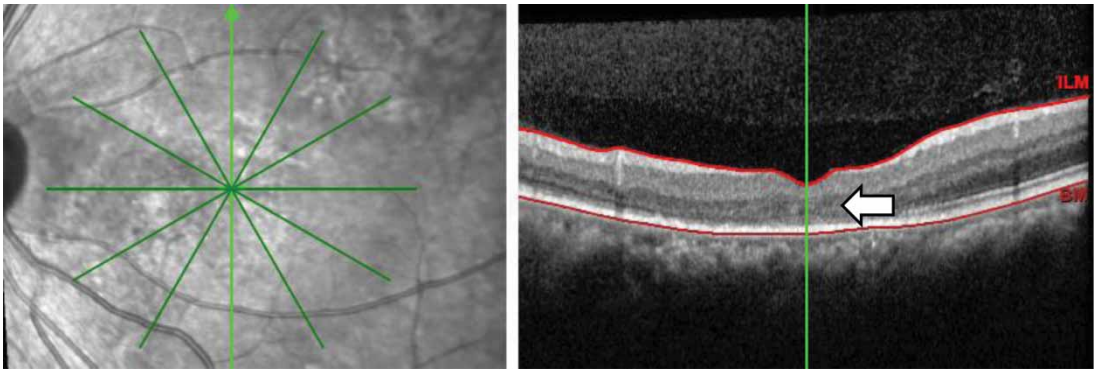

Figure 3. OCT showed resolution of cystoid macular edema (white arrow with black border) with foveal thinning and RPE atrophy.

peptidase activity in ocular tissues. This facilitates cellular adhesion, neutrophil chemotaxis and degradation through elevation of leukotriene D4 concentration.[7] A similar cellular mechanism may influence CSCR-related CME and may contribute to its resolution, as was seen in our case.[2] Reports have also shown that CAI has no effect on the final BCVA[2] as was also seen in our case.

One major drawback of our case is that it is a single case report with no long-term follow-up. Also, to date, only one case series, showing the efficacy of systemic CAI in CSCR, has been published.[2] Although small case reports on topical CAIs in CSCR have been reported, few have highlighted the effect of CAIs on CME[3] due to CSCR. Our case report adds to the knowledge that topical CAIs are as effective as oral CAIs in the resolution of CME due to CSCR, but has no effect on the final visual acuity.

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How to cite this article Das D. and Nigam E. Resolution of cystoid macular edema by topical dorzolamide in a case of central serous chorioretinopathy: a case report, *Sci J Med & Vis Res Foun* 2017;**XXXV**:25–26.

Metallic orbital foreign body: to dive or not to dive?

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Introduction

Intraorbital foreign body (IOrFB) is a rare occurrence[1] and usually is a result of a high-velocity penetrating injury such as gunshot injuries or industrial accidents.[2] However, sometimes it may occur even with trivial trauma with a vague history.[3] Decision to remove an orbital foreign body is based on several factors, weighing the risks vs benefits. We present a case of a young male who presented with an iron foreign body within the lateral rectus muscle that was successfully removed.

Case report

A 39-year-old gentleman, driver by profession, presented to our hospital with a history of injury to the right eye 6 days ago. He sustained the injury while he was beating an iron wire with a hammer, trying to fashion it into a ring. On presentation, the vision in right eye was counting fingers at 1 m. Ocular motility was full, free and painless. Pupil was reacting to light briskly and there was no relative afferent pupillary defect. Slit lamp examination revealed a localized area of conjunctival congestion over the insertion of lateral rectus muscle. There was a self-sealed corneal tear inferonasally and a subsequent tear in the peripheral iris inferiorly (Figure 1). Although the lens was cataractous, no frank breach was noted in the anterior capsule and there was no cortical matter in the anterior chamber. Intraocular pressure was 16 mmHg on applanation tonometry. Dilated retinal evaluation revealed normal attached retina and optic nerve head with localized vitreous hemorrhage inferiorly. Vision in left eye was 6/6, N6 and ocular examination was normal.

On ultrasonography (USG) B scan of the right eye, a high reflective echo was noted in the inferotemporal quadrant, raising the suspicion of an intraocular foreign body. Thus, computed tomography (CT) scan of the orbits was performed. It revealed a hyperdense mass in the lateral rectus muscle and the mid-posterior orbit level, suggestive of an iron foreign body within the orbit. There was no evidence of any intraocular foreign body (Figure 2).

The patient subsequently underwent removal of the foreign body through an inferotemporal forniceal incision. Intraoperatively, a flat iron particle (4 × 5 mm) was found to be embedded into the substance of the lateral rectus ~10–15 mm from the insertion of the lateral rectus (Figure 3). At the final visit 2 months postoperatively, the patient was doing well with no functional deficit. He was advised cataract surgery in the right eye for visual rehabilitation for which he is yet to report back.

Discussion

Most commonly, metallic foreign bodies enter the orbit with high-velocity injuries. BB pellets or metal fragments in cases of BB injuries are light weight with a velocity of 250–750 feet/s. They, thus, are lodged within the confines of the orbit without causing much collateral damage.[4] Bullets, on the other hand, in cases of gunshot injuries are heavier with a higher velocity and are more likely to enter the sinuses or brain through the orbit, causing excessive damage.[4,5] In case of work-related injuries, this assessment is difficult to make as the history is often unclear. Hence, every such accident should be dealt with a high suspicion of ocular and orbital foreign body and fully examined and investigated for assessing the extent of damage caused. In our patient, we found that the iron particle entered through the cornea, tore the peripheral iris and grazed through the inferotemporal retina and scleral and got lodged in the lateral rectus. It is noted in experimental studies that the scleral wounds heal by fibrosis within 7 days of injury.[6] This is probably the reason that there was no scleral wound seen as the patient presented 6 days after the injury.

It is universally accepted that organic foreign bodies in the orbit are to be removed as early as possible, due to the danger of causing infection, inflammation and resultant functional and visual deficits. However, guidelines for the management of metallic and inorganic foreign bodies are still controversial.[2] In this scenario, it is appropriate to consider the size, location and present and possible future complications caused by the foreign body. Also, it is essential to contemplate upon the iatrogenic complications that might occur on its removal.[1] Removal of posteriorly located IOrFB may be fraught with postoperative optic neuropathy, strabismus and ocular motility deficits.[7] Anteriorly placed IOrFBs, on the other hand, are comparatively easier to remove; hence, in the presence of anteriorly located inorganic IOrFBs, the decision of removal can be taken after discussing with the patient.[1,7]

Another aspect to be considered while deciding on IOrFB removal is the content of FB. Most metallic FBs are known to be inert, except Iron, Copper and Lead. There was a conflict of decision in terms of the iron FB removal in our case. Iron IOrFBs carry a theoretical risk of siderosis,

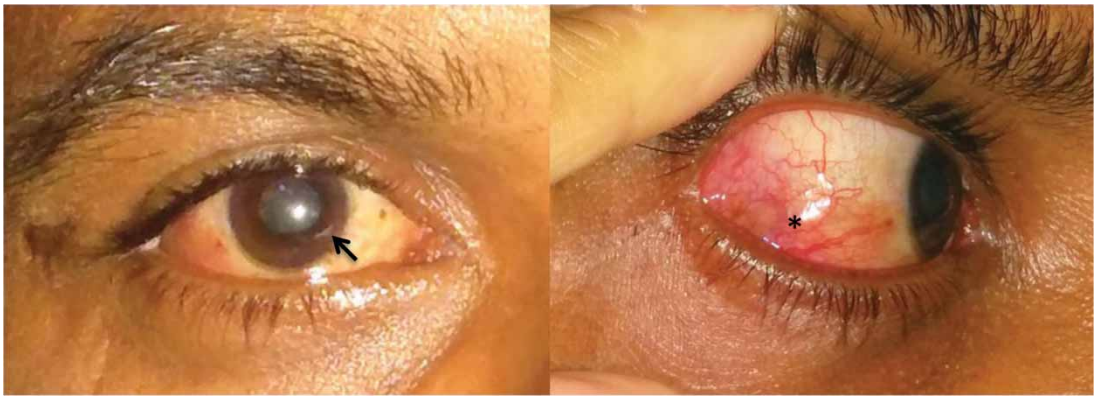

Figure 1. External photograph showing a sealed corneal wound (black arrow) and localized conjunctival congestion over the lateral rectus muscle (asterisk).

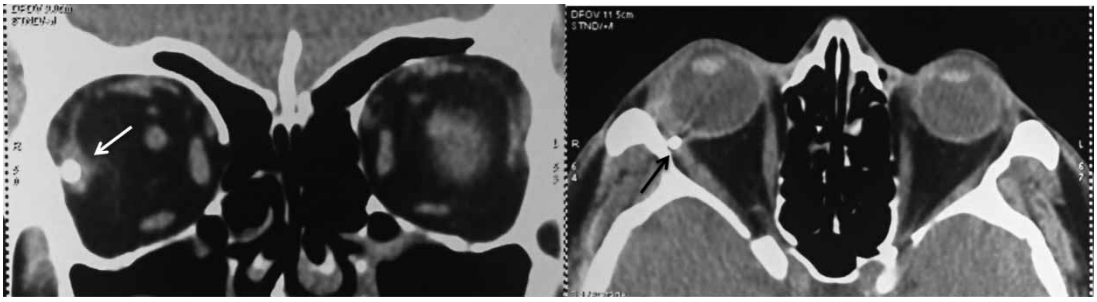

Figure 2. CT scan of the orbits revealing a hyperdense foreign body in the substance of the lateral rectus muscle posteriorly (white arrow), in close proximity to the macula (black arrow).

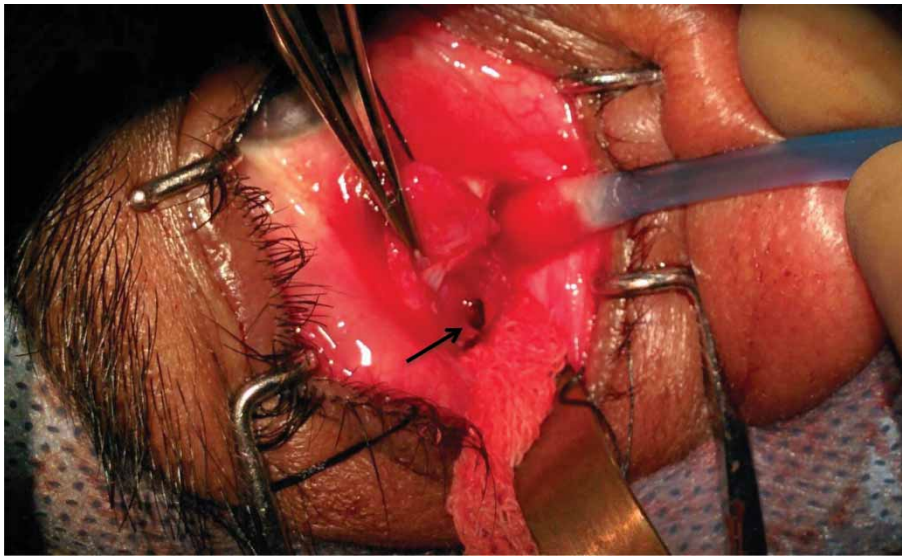

Figure 3. Photograph taken under the operating microscope showing a iron particle in the lateral rectus posteriorly (black arrow).

resulting in vision loss. Experimental models in rats have shown significant iron absorption in the sclera, choroid, retina, ciliary body and corneal epithelium of rats in which iron foreign bodies were placed in the orbit close to the sclera. The severity of penetration and damage was found to be directly proportional to the area of contact with the sclera and the amount of iron.[8,9] A similar risk existed in our patient as the iron IOrFB was lodged in the lateral rectus at the level

posterior to the equator (as evident in Figure 2), in close proximity to the macula. The patient presented within a week of injury which negated the chance of any fibrosis. There was exact localization on CT scan and presence in the rectus muscle instead of orbital soft tissue. We thus predicted easy surgical removal of the IOrFB. All these factors collectively led to our decision of removing the iron IOrFB.

Copper is another metal that warrants removal due to its tendency to incite intense inflammation.[10] Intraocular iron and copper are known to diminish Electroretinography (ERG) responses. In cases of a conflict, serial ERGs can be performed to monitor and detect early retinal damage and subsequently plan management.[11] Lead, present in gunpowder, is the offending foreign body in gunshot injuries. Ho et al.[1] observed no ocular complications in 95% of patients with gunshot injuries over a follow-up period of 6 months to 6 years (median 2 years). However, few studies have demonstrated increased serum levels of lead in patients with retained lead pellets. Thus, the risk of systemic absorption and toxicity cannot be denied.[12] Signs of systemic lead intoxication include colicky abdominal pains, stomatitis with a blue line around the gums, polyneuritis with wrist drop and encephalopathy. Ocular complications include papilledema, retinal hemorrhages, vascular sheathing, pupillary dilation, optic neuritis and extraocular muscle palsies may be noted. It is worthwhile to mention that due to the developments in the ammunition manufacturing, current gun pellets include an antimony coating rendering the lead insoluble.[13] Modern day lead pellets can thus be safely left untouched in the orbit and in high-risk cases, serum lead levels can be monitored.

There is no room for complacency in cases of penetrating ocular and orbital injuries. A high suspicion must be kept for a foreign body and thorough examinations and investigations must be carried out on those lines. Removal of a metallic foreign body in cases where there is no functional or visual deficit due to it is a controversial subject and decision needs to be taken on a case-to-case basis.

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How to cite this article Mehta N.S. Metallic orbital foreign body: to dive or not to dive?, *Sci J Med & Vis Res Foun* 2017;**XXXV**:27–29.

Sixth nerve palsy: a rare presentation of parapharyngeal abscess caused by *Mycobacterium abscessus*

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Abstract

We report a case of a 31-year-old female who came with complaints of sudden onset binocular horizontal diplopia, with the inability to move right eye laterally since 1 week. She had no history of (h/o) trauma or any other systemic features, but she had a past h/o pulmonary tuberculosis (TB) 20 years ago, for which she had received treatment for 1 year. A thorough clinical examination revealed the clinical features of right lateral rectus palsy. Magnetic resonance imaging findings were suggestive of abscess in right prevertebral soft tissue and pachymeningeal involvement along the clivus. She underwent intraoral aspiration of abscess, which was culture positive for *Mycobacterium abscessus*. The patient was treated with a combination of specific antitubercular antibiotics and short course of oral steroids.

Introduction

Sixth nerve palsy is the most common ocular motor nerve palsy.[1] Most common causes are viral illness in children and microvascular diseases in older adults, with good rate of recovery in these cases.[2,3] It is comparatively uncommon in young adults and causes can be idiopathic, vasculopathic, tumors and multiple sclerosis.[4] CNS space-occupying lesions are the most common etiology, followed by multiple sclerosis, in young adults. Whatever be the cause, the recovery of sixth nerve palsy in young adults is poor.[5] Abducens nerve palsy secondary to clivus involvement is very rare and clival metastasis in various cancers has been reported as a cause in the literature,[6–8] but infective pathology has not been reported yet. We report a case of abducens nerve palsy secondary to parapharyngeal abscess involving the clivus.

Case report

A 31-year-old female presented to the strabismus clinic of tertiary eye care center in eastern India with complaints of sudden onset binocular constant horizontal diplopia associated with the inability to move right eye outward. She had no history of (h/o) trauma, headache or any other systemic illness. She had past history of pulmonary tuberculosis (TB) 20 years back for which she had received anti-tubercular treatment for 1 year.

On ophthalmological examination, the best-corrected visual acuity in each eye was 20/20 for distance and N6 for near. Bilaterally, pupils were round, regular and equally reacting to light. Anterior segment examination was normal in both eyes. Intraocular pressure by Goldman applanation tonometry was 14 mmHg in both eyes. Squint examination revealed right esotropia with 25 prism dioptre primary deviation and 50 prism dioptre secondary deviation in primary gaze. Extra-ocular movement examination revealed right eye limitation of abduction (−3), rest extraocular movements were normal (Figure 1). Forced duction test did not reveal any restriction. Dilated fundus examination in both eyes revealed no abnormality. Examination of other cranial nerves was essentially normal. General physical examination did not reveal any other abnormality.

Provisional diagnosis of right-sided abducens nerve palsy was made and in view of young age and no other systemic illness, magnetic resonance imaging (MRI) of brain with orbit with contrast enhancement was advised.

MRI revealed marrow edema with diffuse enhancement in basiocciput as well as anterior arch and lateral masses of atlas vertebra. A hyperintense lesion (of size 1.3 cm × 1.9 cm × 4.1 cm) with rim enhancement was noted in the right prevertebral soft tissue, in STIR (short tau inversion recovery) and T2 images, which was bulging into nasopharyngeal airway. Thick pachymeningeal involvement with enhancement was seen along the clivus. Based on MRI images, t parapharyngeal abscess was suspected and the patient was referred to ENT surgeon and neurology department for abscess aspiration and culture. Patient underwent right parapharyngeal abscess aspiration. Gram stain of the abscess material revealed pus cells with no organism, fungal culture after 14 days of incubation revealed no growth. Automated Fluorescent rapid Acid-Fast Bacillus culture testing revealed the growth of *M. abscessus*, which is a rapid grower Mycobacterium other than tuberculosis (MOTT). Also *Mycobacterium tuberculosis* interferon gamma release assay came out to be positive. Patient was started on antitubercular treatment along with short course of oral steroids by the ENT surgeon.

Discussion

The parapharyngeal space lies in the posterior pharyngeal wall, between the middle and deep layers of the deep cervical fascia, extending from

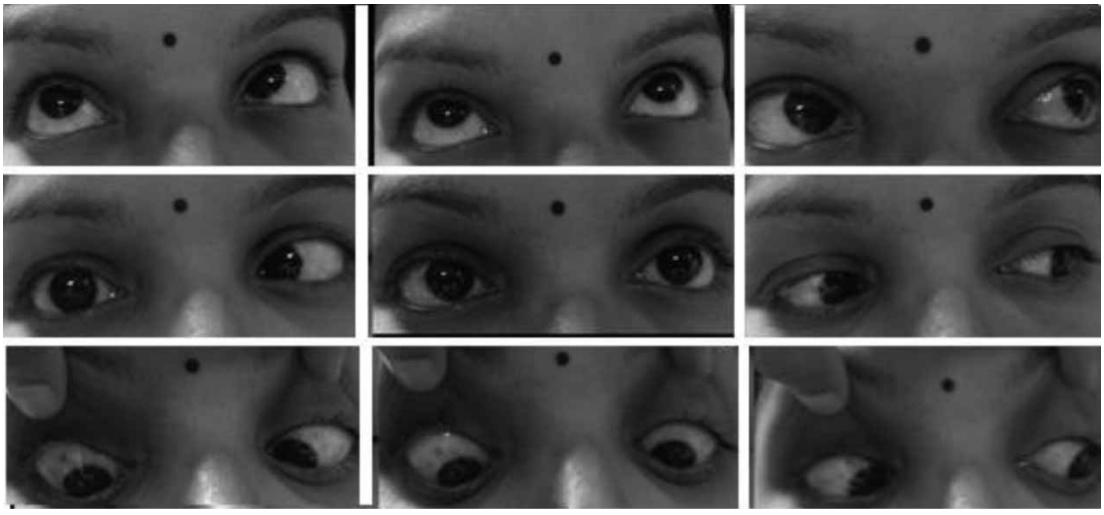

Figure 1. Clinical photographs of patient showing right esotropia in primary position with abduction limitation of right eye.

the base of the skull to the mediastinum.[9] Parapharyngeal abscess in adults is mostly pyogenic and usually secondary to pharyngeal or esophageal perforation or sepsis in the throat or sinuses. However, chronic retropharyngeal abscesses are rare in immune-competent adults.[10] It occurs mostly in immune-compromised patients[10] and retropharyngeal abscesses caused by MOTT in immune-competent patient are very rare. *Mycobacterium abscessus* complex comprises a group of rapidly growing, multidrug-resistant, non-tuberculous mycobacteria that are responsible for a wide spectrum of skin and soft tissue diseases, central nervous system infections, bacteremia and other ocular infections. Sixth nerve has a long course in the brain, so it is vulnerable to trauma and metastasis.[1] George et al.[5] studied the causes and prognosis of nontraumatic sixth nerve palsies in young adults, and reported that CNS mass lesions is the most common cause followed by multiple sclerosis and only 13% of the patients had complete resolution of the palsy. They did not report any case of infective pathology. Bhaswati et al.[11] reported a case of bilateral cavernous sinus thrombosis with left eye third cranial nerve palsy-associated retropharyngeal abscess, culture of which grew Staphylococcus aureus sensitive to vancomycin. Patient improved dramatically with vancomycin and short course of steroids. Complete recovery in ocular movements was recorded after 3 months in that case.

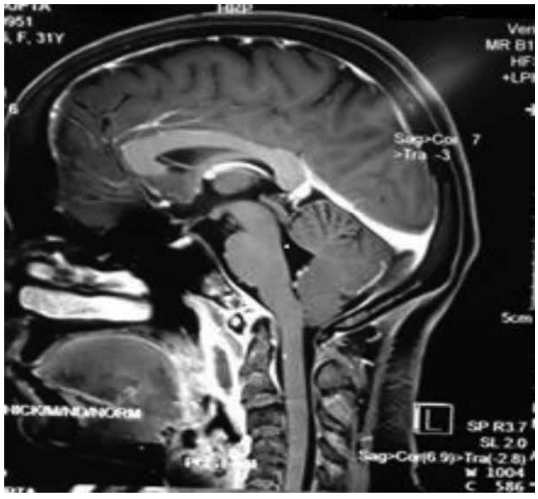

Figure 2. MRI STIR- and T2-weighted sequence hyperintense lesion in right prevertebral soft tissue bulging into nasopharyngeal airway with rim enhancement after contrast along with thick pachymeningeal enhancement along clivus and basiocciput.

We reported a case of young immune-competent 31-year-old female with no h/o other symptoms in relation with parapharyngeal abscess, presented with sixth nerve palsy as first sign. Abscess aspiration revealed a very rare organism from Mycobacteria other than tuberculosis group of organisms, *M. abscessus*. Very few drugs are available for this subgroup of mycobacteria. Our case was treated with anti-tuberculosis medicines and also short course of oral steroids. After a follow-up of 2 months, patient did not improve with her symptoms of diplopia, and was given the option of occluder or Fresnel prism. Our case was unique, because the patient was a young immune-competent female, with no h/o other symptoms in relation with parapharyngeal abscess, and presented with sixth nerve palsy as the first and only sign. Abscess aspiration revealed a very rare organism from Mycobacteria other than tuberculosis group of organisms, *M.*

abscessus, for which only very few drugs are available. Also this case emphasizes the significance of neuro-imaging in young adults with abducens cranial nerve palsy.

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How to cite this article Bhamare B., Sheth S., Mittal S., Biswas T., Sahana S. and Guha S. Sixth nerve palsy: a rare presentation of parapharyngeal abscess caused by *Mycobacterium abscessus*, *Sci J Med & Vis Res Foun* 2017; **XXXV**:30–32.

Role of polymerase chain reaction in current ophthalmology practice

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Introduction

Polymerase chain reaction (PCR) is a technique involving enzymatic amplification of nucleic acid sequences in repeated cycles of denaturation, oligonucleotide annealing and DNA polymerase extension. It selectively amplifies a single or few copies of a piece of DNA, thereby generating millions or more copies of a particular DNA sequence. It is thus also described as 'molecular photocopier' in a test tube. PCR was invented by Kary Mullis of USA in 1983 for which he was awarded the Nobel Prize in chemistry in 1993.[1]

Advantages of PCR are that it can be performed on a very small amount of tissue and almost any tissue or body fluid can be used. The sensitivity of the technique is high. Generally, PCR can detect 10–100 viral genomes which is less than the amount of genomes which is required to form 1 plaque in viral culture.[2]

In ophthalmology practice, the samples for PCR are usually obtained from conjunctival swab, tear fluid, corneal epithelial scrapings, anterior chamber or vitreous aspirate. The choice of collecting sample should be guided by disease suspicion. Fifty microliters of aqueous and 100–300 µl of vitreous aspirate are sufficient for diagnostic purpose. Specimens should be aseptically transferred to a sterile, capped tube (i.e. a 1.5-ml microfuge tube) and quick-frozen on dry ice or in liquid nitrogen.

Indications of PCR[3–8]

a In diagnosing Acanthoemeba keratitis and in the detection and serotyping of viral genomes in patients with adenoviral infections.

b Infectious endophthalmitis: To differentiate bacterial from fungal aetiology.

c Viral retinitis: To detect Herpes simplex virus, Varicella Zoster virus or Cytomegalovirus as causative organism.

d In the diagnosis of Ocular toxoplasmosis and Ocular tuberculosis.

e Masquerade syndrome: To detect IgH gene rearrangements and provide a helpful adjunct for the diagnosis of B-cell lymphoma in the eye.

f HLA typing in non-infectious endophthalmitis.

PCR lab in Sankara Nethralaya Kolkata

PCR was introduced in Sankara Nethralaya, Kolkata in the year 2012. It was set up with the expertise of the L&T Microbiology research centre, Vision Research Foundation in Chennai. Currently, it is the only centre in Eastern India providing state-of-the art PCR diagnostic facilities to both our in-house patients and surrounding referral hospitals. Presently, our lab provides facilities to detect Eubacterial genome, Panfungal genome, P Acnes genome, HSV,VZV,CMV, HIV1 and 2, Toxoplasmosis, Mycobacterium Tuberculosis (MPB64 gene) and for HLA B27 typing. For infectious endophthalmitis panel, the eubacterial genome (targeting the 16s rRNA gene), *Propionibacterium acnes* (targeting the 16s rRNA gene) genome and the panfungal genome (28Srna gene) are tested.

Our experience with PCR in Infectious Endophthalmitis

At Sankara Nethralaya Kolkata, we did a study to analyse the efficacy of PCR in Endophthalmitis isolates. We conducted a retrospective analysis of consecutive post-cataract surgery endophthalmitis patients treated at a Sankara Nethralaya, Kolkata between 2012 and December 2015. The data about clinical features, investigations, treatment, and outcome were obtained from the medical records. All patients had undergone comprehensive ophthalmic examination which included recording of best corrected visual acuity (BCVA) with Snellen's distance visual acuity chart, slit lamp examination and fundus evaluation. In eyes with no view of fundus, ultrasonography was performed to assess the status of posterior segment.

Aqueous tap (0.05 to 0.1 ml) was performed as the first step in the microbiological analysis of endophthalmitis. It was performed in the outpatient department at the first visit of the patient. Vitreous sample was obtained for analysis only in patients who underwent pars plana vitrectomy. To identify the bacterial and fungal isolates, samples were first analysed with Gram's stain, 10% potassium hydroxide (KOH) mount, Giemsa stain and Ziehl–Neelsen stain. Further samples were inoculated into blood agar, chocolate agar, Sabouraud's dextrose agar, thioglycolate medium, brain– eart infusion agar and Lowenstein–Jensen agar. All samples were analysed with PCR to identity Eubacterium, Panfungal, and P Acnes genome.

One hundred and thirty two eyes with post-cataract surgery endophthalmitis were included in the study. Seventy (53%) patients were male and 62 (47%) were female. The mean age of patients was 58.87±12.27 years (range 18–90 years). Gram's stain was positive in 21 (15.9%) and negative in 111 (84.1%) eyes. KOH mount showed fungus in five (3.8%) eyes and was inconclusive in 127 (96.2%) eyes. PCR was positive 118 (89.4%) and negative in 24 (18.2%) eyes. A total of 95 (72%) were eubacterium genome positive and 23 (17.4%) were panfungal genome positive. Overall, 51 (38.6%) eyes were culture positive and remaining 81 (61.4%) were culture negative. Gram negative bacilli (27; 51.9%) were the commonest isolate, followed by Gram positive cocci (10; 19.2%), fungus (9; 17.3%), and Gram positive bacilli (6; 11.53%). *Pseudomonas aeruginosa* (11; 21.1%) was the commonest bacterial isolate, followed by *Staphylococcus epidermidis* (8; 15.4%) and *Bacillus cereus* (6; 11.5%). The isolated fungi were *Aspergillus fumigatus* (3), *Fusarium* (2), *Candida albicans* (1), *Candida lipolytica* (1), *Scedosporium apiospermum* (1), and *Paecilomyces* (1).

In the literature review, it was noted that microbial culture was effective in identifying the causative organism in a limited number of cases. This may mean that newer diagnostic modalities like PCR need to be utilized for timely identification of microorganism and institution of targeted antimicrobial therapy. The present study is the first to report the diagnostic role of eubacterial and panfungal genome PCR in post-cataract surgery endophthalmitis in Eastern Indian scenario. PCR positivity (81.8%) was significantly higher than culture positivity (38.6%) in our study. Hence in patients with symptoms of endophthalmitis but negative culture, a positive PCR helped us in diagnosing it as endophthalmitis. This may mean that rather than conventional staining techniques, it is the PCR which would help us in pinpointing the diagnosis in endophthalmitis with negative culture reports and start early treatment. Since conventional culture techniques are time-consuming and have variable yield, PCR techniques can be a significant tool in the armamentarium of ophthalmologists for the timely management of endophthalmitis.

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How to cite this article Thomas N.R., Saurabh K. and Roy R. Role of polymerase chain reaction in current ophthalmology practice, *Sci J Med & Vis Res Foun* 2017;**XXXV**:33–34.

List fellows trained in Sankara Nethralaya Kolkata

SARAT RAVI	CLINICAL V R FELLOW
SUDIPTA DAS	CLINICAL V R FELLOW
DEEPAK AGRAWAL	CLINICAL V R FELLOW
KUMAR SAURABH	CLINICAL V R FELLOW
DEBAPRIYA DAS	CLINICAL GENERAL OPHTHALMOLOGY FELLOW
KUMAR GUDDU	CLINICAL GENERAL OPHTHALMOLOGY FELLOW
NITISH MAHAJAN	CLINICAL V R FELLOW
SUBHAJIT DAS	CLINICAL V R FELLOW
ARINDAM ROY	CLINICAL V R FELLOW
CHITRALEKHA DE	CLINICAL V R FELLOW
PRATIBHA BAPUSAHEB PATIL	CLINICAL PEDIATRIC OPHTHALMOLOGY FELLOW
EESH NIGAM	CLINICAL V R FELLOW
VIVEK ARORA	CLINICAL V R FELLOW
SANIL VIJAYBHAI SHAH	CLINICAL PEDIATRIC OPHTHALMOLOGY FELLOW
DEBLEENA DEY	CLINICAL GENERAL OPHTHALMOLOGY FELLOW
KAUSHIK SADHUKHAN	ADVANCED CATARACT COMMUNITY OPHTHALMOLOGY FELLOW
TRIPTI JAJODIA	CLINICAL GENERAL OPHTHALMOLOGY FELLOW
ABHINANDAN NARASGOUNDA PATIL	CLINICAL V R FELLOW
PRADEEP KUMAR PANIGRAHI	CLINICAL V R FELLOW
PREETI MEGUNDAPPA HURAKADLI	CLINICAL PEDIATRIC OPHTHALMOLOGY FELLOW
GARIMA HARISHANKAR AGARWAL	CLINICAL GENERAL OPHTHALMOLOGY FELLOW
HIMABINDU ADUSUMILLI	CLINICAL V R FELLOW
VISHAL VIDYADHARRAO KULKARNI	CLINICAL V R FELLOW
SHAMA ALI GOLIYADI	CLINICAL PEDIATRIC OPHTHALMOLOGY FELLOW
SANTOSH KRISHNA	CLINICAL GENERAL OPHTHALMOLOGY FELLOW
PRASHANT SRIVASTAVA	CLINICAL GLAUCOMA FELLOW
ARTI ELHENCE	CLINICAL PEDIATRIC OPHTHALMOLOGY FELLOW
DEBMALYA DAS	CLINICAL V R FELLOW
PRACHEER RANJAN AGARWAL	CLINICAL V R FELLOW
MITTAL SHRUTI RAJENDRA	CLINICAL PEDIATRIC OPHTHALMOLOGY FELLOW
ANINDYA KISHORE MAJUMDER	MEDICAL RETINA AND UVEA FELLOW
ANEESHA LOBO	CLINICAL V R FELLOW
JOHN SARKAR	CLINICAL CORNEA FELLOW
VANDANA AGRAWAL	CLINICAL GENERAL OPHTHALMOLOGY FELLOW
GAURAB MAJUMDAR	ADVANCED CATARACT COMMUNITY OPHTHALMOLOGY FELLOW
SHAH KHYATI PRAVIN	CLINICAL PEDIATRIC OPHTHALMOLOGY FELLOW
AMIT BHURMAL JAIN	CLINICAL V R FELLOW
PRAKASH PARIMAL	CLINICAL PEDIATRIC OPHTHALMOLOGY FELLOW

DHILEESH P CHANDRASEKHARAN	CLINICAL V R FELLOW
BENAZIR ANSARI	MEDICAL RETINA AND UVEA FELLOW
AVIRUPA GHOSE	MEDICAL RETINA AND UVEA FELLOW
ADITI GHOSH DASTIDAR	CLINICAL CORNEA FELLOW
DESHMUKH KAUSTABH PADMAKAR	CLINICAL V R FELLOW
CHINMAYI HIMANSHU VYAS	CLINICAL V R FELLOW
SHETH SIDDHARTH KAMLESH	CLINICAL PEDIATRIC OPHTHALMOLOGY FELLOW
SHARMA PREETI BANSHIDHAR	MEDICAL RETINA AND UVEA FELLOW
ADITYA BANSAL	CLINICAL V R FELLOW
SOMA SARKAR	CLINICAL PEDIATRIC OPHTHALMOLOGY FELLOW
BHAVNA DILIP BHAMARE	CLINICAL GENERAL OPHTHALMOLOGY FELLOW
TANMOY BISWAS	CLINICAL PEDIATRIC OPHTHALMOLOGY FELLOW
PRACHI SUBHEDAR GHOSH	CLINICAL PEDIATRIC OPHTHALMOLOGY FELLOW
MONEESH SAXENA	CLINICAL V R FELLOW
BIJOY KRISHNA CHARABORTY	CLINICAL V R FELLOW
VEER SINGH	CLINICAL V R FELLOW
KALPITA DAS	CLINICAL V R FELLOW
DEBI KUNDU	CLINICAL GENERAL OPHTHALMOLOGY FELLOW
NICEY ROY THOMAS	CLINICAL V R FELLOW
KAUSTAV DATTA	CLINICAL V R FELLOW
RISHI GUPTA	CLINICAL CORNEA FELLOW
RAMA PURUSHOTTAM KALANTRI	CLINICAL PEDIATRIC OPHTHALMOLOGY FELLOW
MADHURA MUKHERJEE	MEDICAL RETINA AND UVEA FELLOW
MAITREYI CHOWDHURY	CLINICAL V R FELLOW
SATISH SHARMA	CLINICAL PEDIATRIC OPHTHALMOLOGY FELLOW
SAKET SHASHIKANT BENURWAR	CLINICAL CORNEA FELLOW
RICHA KAMAL	CLINICAL GENERAL OPHTHALMOLOGY FELLOW
DEEPIKA KUMARI KHEDIA	CLINICAL GENERAL OPHTHALMOLOGY FELLOW
APARAJITA CHATTERJEE	CLINICAL CORNEA FELLOW
RUPA ADHIKARI	SHORT TERM OBSERVER SHIP IN MEDICAL RETINA

Facilities, speciality clinics and state of the art infrastructure in SN Kolkata

S.L.No	Speciality Clinics	Services Provided	Location
1	Contact Lens	Soft Contact lenses Fitting-Spherical, Toric and Multifocal lenses RGP-contact lenses fitting ROSE-K Piggy Back Lenses Scleral Lenses	Rajarhat and Mukundapur
2	Low Vision Clinic	• Low Vision Device Trial • Visual Development therapy • Occupational Therapy • Eye Hand coordination therapy • Certificate for School Children • Special school, Blind School, Integrated School Address • counselling centre address	Rajarhat and Mukundapur
		• Low Vision Trial-Optical and Non-optical both • Tactile procedure training • Environmental Modification • Kitchen Counter Modification • Colour Enhancement Modification • Colour Matching Procedure • Mobile phone Modification • Computer Modification • Head Scanning Technique • Eccentric Fixation Training • Sighted Guide procedure training • Mobility Training • Disability provisional certificate • Rehabilitation Centre Address • IQ Test centre Address • Jaw Computer centre address and Braille centre Address	Rajarhat and Mukundapur
		• D15 Colour Vision Test • FM-100 Hue Test • Contrast Acuity Testing	
3	Orthoptics Clinic	Non-Strabismic Binocular Vision Dysfunction tested and treatment given in the form of Exercises. Prism Trial for Strabismic patients Headache and asthenopic symptoms are assessed and treatment given	Rajarhat and Mukundapur
	Vision Therapy Clinic	Strabismic and Non-strabismic patients are treated. Anti-Suppression tests for amblyopia patients	Rajarhat and Mukundapur
	Adult Amblyopia Clinic	Occlusion Therapy Action Video Games to improve amblyopia	Rajarhat and Mukundapur
	Neuroptometry	Neuro-optometric Vision Therapy Field Expanders for Field Defects Prisms for combating Diplopia Binasal occlusion therapy for brain injury patients	Rajarhat

	Computer Vision Clinic	Comprehensive Orthoptic Tests Dry Eye Evaluation Ergonomic Set up advice Vision Therapy	Rajarhat
	Prosthetic Clinic	Custom made prosthesis Stock Eye	Mukundapur
	Diagnostic Clinic	**Electrodiagnostics** Electroretinogram (ERG) Electro oculogram (EOG) Visual Evoked Potential (VEP) **Retinal Diagnostics** Fundus Fluorescein Angiogram (FFA) Optical Coherence Tomography (OCT) Indocyanine Green (ICG) Fundus Photo **Glaucoma Diagnostics and lasers** Humphrey Visual Field (HVF) Pachymetry Heidelberg Retinal Tomogram (HRT) OCT-RNFL Yag laser iriodtomy Selective Laser Trabeculoplasty **Cornea Diagnostics and Lasers** Topography Oculyser Specular Microscopy AS-OCT Allergeto excimer laser with Femto Second laser **Squint Clinic** Hess Charting Diplopia Charting **Other Diagnostics** Ultrasonography (USG Bscan) Ultrasound Biomicroscopy (UBM)	Mukundapur Rajarhat and Mukundapur Rajarhat and Mukundapur HRT – Rajarhat Mukundapur
	Cataract Clinic	Digital Biometry Reader (DBR) IOL Master Keratometry Specular Microscopy	Rajarhat and Mukundapur

THE JOURNEY OF SANKARA NETHRALAYA KOLKATA

Sankara Nethralaya has always occupied a special place in the hearts of the people of Bengal. Therefore it was no surprise when SN decided to start offering its services at Kolkata to help the multitude of local people who could not make the arduous journey all the way to Chennai despite their best efforts.

The first ever branch of SN in Kolkata was established in 2003 as a joint venture with Rotary and Asia Heart Foundation under the banner "Rotary Narayana Sankara Nethralaya". After its short existence, in July 2007, Sankara Nethralaya started off on its first independent venture in Kolkata with just 5 consultants and a mere 2500 sq.ft. space at an interim location in 10, Raja Subodh Mullick Square (operational till date), while the main building was being constructed at Mukundapur.

20th January 2009 was indeed a proud day for Sankara Nethralaya Kolkata as it was on this day that the magnificent 5-storeyed building "Aditya Birla Sankara Nethralaya" was commissioned with the SN Kolkata team of 12 consultants catering to all the ophthalmic subspecialities like Vitreo-Retina, Cornea, Glaucoma, Paediatric Ophthalmology, Neuro-Ophthalmology and Oculoplasty.

We also acquired the Polymerase Change Reaction Lab in 2012, another landmark of sorts as it was the only one of its kind in this part of the country.

Team SNK grew in strength and so did its operations. In just 7 years, the number of OPD consultations rose from 200 per day to more than 700 per day, and the number of surgeries grew from 2700 per year to more 10000 per year. Due to the rising popularity of SN Kolkata and dirth of space in Mukundapur to expand its services, a new project at Rajarhat was conceived.

Kamalnayan Bajaj Sankara Nethralaya at Rajarhat came into being in February 2017 complete with all the subspecialities including Ocular Oncology, another feather in the cap of SNK.

Fellowship progams had been started at SNK as early as 2009 in General Ophthalmology, Paediatric Ophthalmology and Vitreo-Retinal Services. Later, Medical Retina – Uvea and Cornea fellowships were also introduced. We have 12 fellows who are currently under training and till date, 35 fellows in different specialities have been trained by us. Another milestone for SNK was the recognition by the Diplomate of National Board as a teaching center and we have been granted two primary and two secondary DNB seats starting this academic year.

We are a bright young team and we are proud to share that in just a few years SNK has more the 60 publications to its credit. The presence of SNK in Kolkata has been growing stronger and stronger in both clinical and academic fields. We have come a long way from the time we started with just five consultants in RSM Square a decade back. SNK is a young organisation with immense potential to grow and is now all set to conquer and scale new heights and keep the flag of Sankara Nethralaya flying high.

Dr Sujata Guha
Dr Suchetana Mukherjee