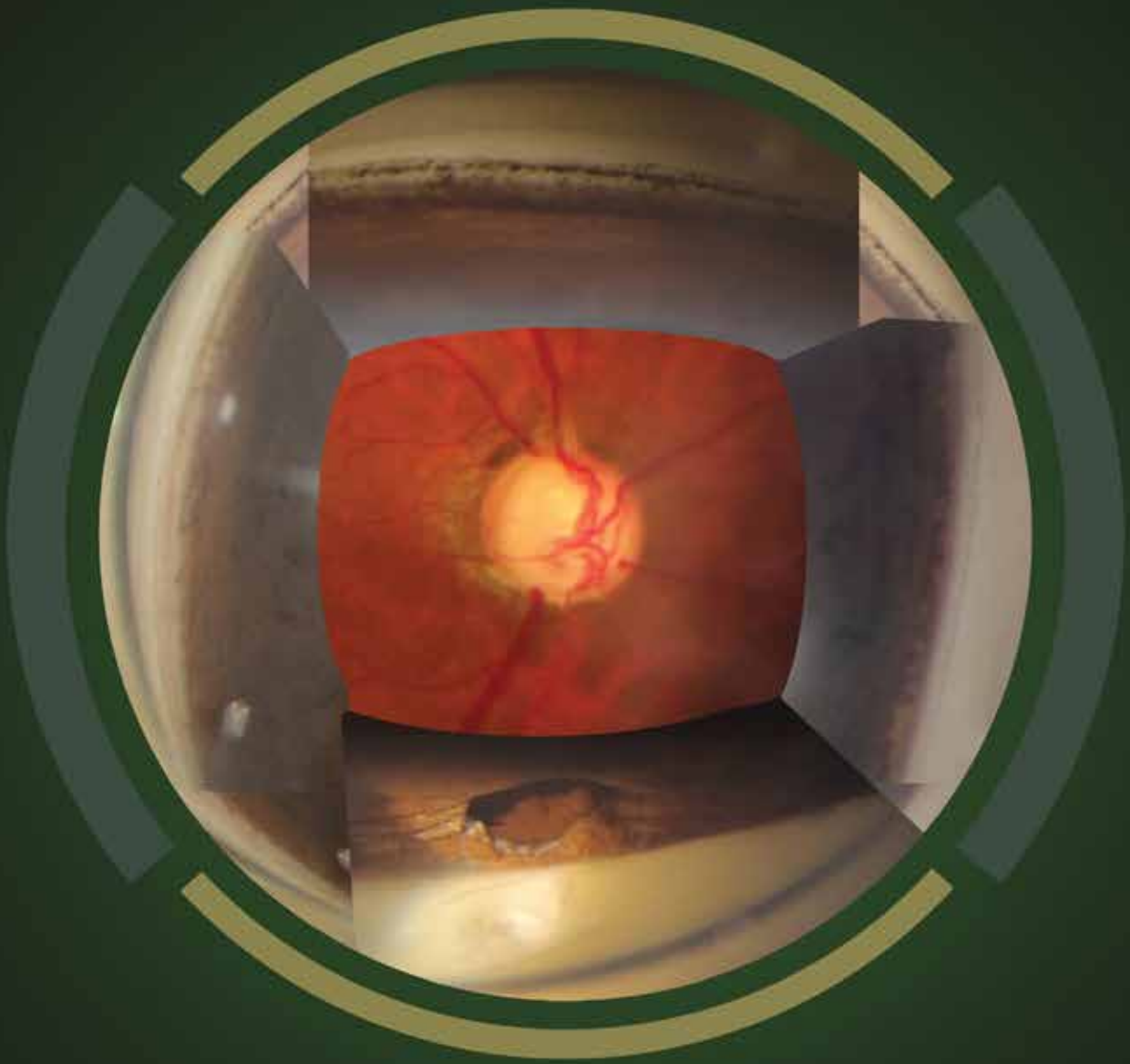
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GLAUCOMA FOCUS ISSUE: HIGHLIGHTS

♦ Pressure centric to patient centric glaucoma care ♦ Evolving role of MIGS in glaucoma treatment: Towards predictive medicine ♦ Secondary glaucoma following vitreo-retinal surgery: A Review ♦ Impact of glycaemic status on CCT and IOP in diabetic patients ♦ Releasable sutures : For safe and titrated trabeculectomy (KJO Video)

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Editorial

Pressure centric to patient centric glaucoma care: Connecting the dots

"Medicines cure diseases, only doctors can cure patients". Carl Jung

Glaucoma is a leading cause of irreversible blindness, affecting an estimated 80 million people globally. The burden of Glaucoma in the Indian context is daunting. One in 8 persons or nearly 40 million Indians aged 40 years or above either has glaucoma or is at risk of developing it.[1] In India, the rate of undiagnosed glaucoma is very high (90%), while the level of glaucoma awareness is only 0.32 to 13.5%.[2]

This editorial intends to high light some ways that might help health care providers move toward patient centric glaucoma care. It will also offer a glimpse into the possibilities of precision medicine for targeted/personalized glaucoma management.

QUALITY OF LIFE: THE MISSING LINK IN PRESENT DAY GLAUCOMA CARE?

Control of intraocular pressure to save the visual field is the basic treatment goal for clinicians, but let us not forget that ultimately a higher quality of life (QoL) could be more important for the patient. So, when it comes to defining treatment goals, are we suffering from tubular vision and missing the big picture? Perhaps yes. Here are a few suggestions to move towards patient centricity in present day glaucoma practice:

- **Look beyond the disease, see the patient**: It is essential that clinicians dedicate more time for the glaucoma patient to bring out his personal needs, concerns and challenges related to the disease and its treatment. Quality of life assessments should be part of the clinical routine at least at baseline and periodically. Intraocular pressure, optic disc and visual fields may not always tell the whole story. The economic feasibility, compliance issues and toxicity of glaucoma medications are problems clinicians should discuss specifically with their patients. Near work and Mobility are top issues that affect the QoL of glaucoma patients. So, treatment goals of a glaucoma patient should include low vision training, mobility training and counselling as needed, apart from the management of the disease proper.
- **How to check QoL? The ideal QoL questionnaire** should be short, simple, and easy to use, with high reliability, reproducibility, sensitivity, and validity. Self-administered questionnaires are preferred. Of the forty odd validated glaucoma QoL questionnaires for the purpose, no single existing questionnaire design suits all purposes.The glaucoma-specific Glau-QoL 36, and the shorter versions GQL-15 and GAL-9 may be the best tools to detect quality of life decline correlating with disease progression, while the MOS SF-36 assesses patient's general health and wellbeing by noting both physical and mental health status.[3] So you need to choose your questionnaire depending on your objective and the patient needs. In a busy outpatient clinic, formal questionnaires may even have to be trimmed further to selected relevant questions.
- **Attitude and communication**: Evidence suggests that glaucoma as well as self-suspicion of it/fear of losing vision can negatively impact QoL and mental health.[4] This means that even glaucoma screening programmed could paradoxically affect some patient's mental health negatively as they make patients aware of the disease/its possibility. So, we need to be extra cautious and sensitive in our attitude and communication while counselling a freshly detected glaucoma patient or suspect.
- **Training the care givers**: It is vital to train care givers on how to make the immediate home environment safe and glaucoma friendly for patients. Our glaucoma awareness activities, currently focused on detection and management of the disease need to include organizing workshops for low vision training/environmental modifications for patients and their caregivers. This can help them face glaucoma related daily life challenges better, thus enhancing QoL.

THE PROMISE OF PRECISION MEDICINE FOR TARGETED GLAUCOMA CARE

At present, clinical examination, intraocular pressure (IOP) measurements, visual field tests, and optical coherence tomography (OCT) drive diagnostic and therapeutic decision making in glaucoma. This disease often baffles clinicians

many a time with its unpredictable nature and varied treatment responses across different patients. It poses difficult questions. Here are a few examples:

- Why is the target pressure so variable across patients?
- Why do different patients have variable responses to the same drug?
- Why do some patients with NTG progress to Primary open angle glaucoma (POAG)?

Questions like these point at many missing links in the glaucoma story. The emerging concept of precision medicine and its role in glaucoma care may provide us with some of the answers. Evidence-based medicine (EBM) is defined as a judicious use of treatment for patients as per the results of clinical trials and research, whereas precision medicine (PM) is an individual based approach for patient's treatment with modifications in the standard treatment after considering variations in genetic makeup, environment, and lifestyle.[5] PM brings in more patient centricity into the treatment paradigm. It is backed by advances in molecular biology and emergence of biomarkers in glaucoma based on Artificial intelligence models, genetic mapping, assessment of NAD/NADH redox state and detection of metabolites/proteins in body fluids.[6]

- **Artificial Intelligence based models**: Deep learning models can detect and quantify early glaucomatous damage using Fundus photos/Optical Coherence Tomography/standard automated perimetry fields. Imaging biomarkers may help clinicians decide upon the initial surgical intervention based on a patient's aqueous vein map.[7]
- **Genetic markers**: Discovery of genetic biomarkers opens the possibility to identify new biological pathways that could influence disease manifestation and progression. This accurate information on genes and pathways that could potentially influence a person's treatment responses can pave way for genetically guided, personalized treatment of glaucoma. Pharmacogenomics may predict the efficacy as well as the vulnerability of a patient to potential side effects of a particular drug. Genetic markers can also allow closer monitoring of family members at risk of developing glaucoma. An excellent example would be predictive gene testing for myocilin-associated glaucoma. MYOC gene mutation is associated with Primary Open Angle Glaucoma, steroid-induced glaucoma, and juvenile-onset glaucoma
- **NAD+/NADH Redox State and Glaucoma**: Reduced levels of NAD + represent impaired mitochondrial activity and, consequently neurodegenerative damages. Nicotinamide, the primary precursor of NAD+, was lowered in the plasma of glaucomatous patients, suggesting its potential as a biomarker and a possible therapeutic role for nicotinamide supplementation for POAG
- **Metabolic biomarkers:** Multiple metabolites and proteins in different biological fluids (tear film, vitreous, aqueous, serum, and plasma) are being investigated as biomarkers for early diagnosis of glaucoma (example: the plasma levels of Homocysteine and Cysteine). In addition, serum antibody profiling is shown to distinguish glaucomatous patients from controls with a specificity of 93%. Certain autoantibodies against ocular tissues implicated in glaucoma are anti-HSP60, anti-a-A-crystalline, a-B-crystalline, glutathione-S-transferase, retinaldehyde binding protein, glial fibrillary acidic protein, retinal S-antigen, and neuron-specific enolase.

Despite all the promise they hold, glaucoma biomarkers need to be considered with caution due to the heterogeneity of disease presentation and the differences in the genetic pattern of individuals. Also, it is unclear whether the alterations in antibody profile are a cause or a consequence of glaucoma-related alterations. Validation studies are currently lacking for biomarkers now available for glaucoma. The next phases of translational research are to integrate the biomarkers with the established clinical risk factors.

In summary, the discovery and validation of biomarkers have the potential to shift the current model of event-based care rooted in visual field change towards preventing glaucoma blindness by intervening at a much earlier and fundamental level. While precision medicine promises to be the future of targeted patient care, incorporating Quality of Life goals at each point of glaucoma care delivery is something we all can practice today to make our treatment more patient centric.

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The evolving role of minimally invasive glaucoma surgery in glaucoma treatment: A shift toward personalized and predictive medicine

Glaucoma is a leading cause of irreversible blindness worldwide, affecting an estimated 76 million people globally in 2020, with projections reaching 111.8 million by 2040.[1] Its management has traditionally encompassed a spectrum of interventions, including pharmacologic therapy, laser treatments, and surgical procedures, each tailored to disease severity and patient-specific factors.[2] This highlights the need for continued advancements in its management strategies.[3] Minimally Invasive Glaucoma Surgery (MIGS) has emerged as a transformative option, offering less invasive alternatives to traditional surgeries like trabeculectomy and tube shunts, with growing evidence supporting its role in earlier intervention and safer surgical outcomes.[4] However, the effectiveness of MIGS is influenced by factors beyond intraocular pressure (IOP) reduction, including patient selection, systemic health considerations, and surgeon expertise, all of which contribute to its variability in outcomes.[5] Understanding these variables is crucial for optimizing patient outcomes and advancing toward personalized glaucoma care, integrating precision medicine and patient-specific treatment strategies.[4]

ADVANCES IN MIGS PROCEDURES AND THEIR COMPARISON WITH TRABECULECTOMY AND TUBE SHUNTS

Unlike traditional surgeries like trabeculectomy and tube shunts that create external drainage pathways, MIGS primarily enhances the eye's physiological outflow structures, such as the trabecular meshwork and Schlemm's canal, aiming to lower IOP while minimizing tissue disruption by using less invasive micro-incisions, serving as an intermediate approach between pharmacologic therapy and traditional surgical interventions. With quicker recovery times and fewer complications, MIGS offers a balance between efficacy and safety, demonstrating promising results in patients with mild-to-moderate glaucoma, by yielding moderate yet sustained IOP reduction and favorable safety profiles across multiple randomized clinical trials, making them a favorable alternative to traditional glaucoma surgeries.[4,6,7]

While traditional trabeculectomy and tube shunts remain the gold standard surgical management for moderate-to-severe glaucoma, providing robust IOP reduction;[8] These procedures carry a high risk of complications, including hypotony, bleb-related infections, and long-term scarring.[9] MIGS, in contrast, prioritizes patient safety but often requires careful patient selection to ensure sustained efficacy.[4,7] This tradeoff underscores the need for individualized treatment pathways based on disease severity, anatomical considerations, and a comprehensive risk-benefit analysis.[7] A growing trend in glaucoma management is the combination of MIGS with cataract surgery, which has demonstrated enhanced IOP reduction compared with cataract surgery alone, making it an attractive option for phakic patients with mild-to-moderate glaucoma.[6]

THE HUMAN FACTOR IN MIGS: SURGEON PROFICIENCY AND CHALLENGES IN PATIENT SELECTION AND LONG-TERM EFFICACY

While much of the research on MIGS focuses on device efficacy, one of the most underappreciated aspects of surgical outcomes is the role of the surgeon and the patient. MIGS outcomes are not solely dictated by device choice but also by how well a particular procedure aligns with the patient's anatomy, lifestyle, and systemic health profile.[10]

One of the critical aspects of MIGS success lies in proper patient selection, as trabeculectomy is widely used for advanced glaucoma while MIGS is generally recommended for patients with mild to moderate disease, where factors such as angle anatomy, prior ocular surgeries, and individual response to IOP-lowering procedures influence long-term outcomes and must be carefully evaluated.[4,11] Moreover, some patients may require additional procedures over time, as MIGS does not provide the same degree of IOP lowering as traditional filtering surgeries; while MIGS offers quicker recovery and fewer complications, their long-term efficacy in preventing glaucoma progression remains a topic of ongoing investigation and requires further validation.[6]

Another major factor influencing MIGS outcomes is the variability in surgical skill and experience among ophthalmologists as each MIGS device requires a different implantation technique

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and learning curve, and surgeons proficient in certain MIGS procedures may achieve better results than those with limited experience, emphasizing the role of surgical training and expertise in optimizing patient outcomes.[10] A MIGS procedure performed by an experienced surgeon comfortable with a specific device is more likely to achieve optimal outcomes than one performed by a less experienced operator.[10,12] This is particularly relevant in the case of the XEN Gel Stent (XEN; Allergan, an AbbVie Company, Irvine, CA, USA) and glaucoma drainage devices (GDDs), where studies have highlighted that complication rates are higher among surgeons still in training, such as fellows learning the procedure.[12] Surgeons performing XEN implantation in fellowship-training programs can face a higher rate of complications in the early postoperative period, underscoring the need for structured training and competency benchmarks in MIGS.[12]

This variability raises important questions about how surgical training should be standardized for MIGS procedures. As more ophthalmologists adopt MIGS into their surgical practice, establishing structured training and competency benchmarks will be critical in maintaining consistent and high-quality patient care in MIGS.[7,12,13]

Future research evaluating MIGS should account for this human factor—not just the device itself but also the level of proficiency in its use, and whether training programs should provide structured MIGS exposure.[7,13]

SYSTEMIC HEALTH CONSIDERATIONS IN MIGS AND GLAUCOMA MANAGEMENT

Glaucoma is influenced by systemic health factors. Comorbidities such as diabetes, hypertension, autoimmune diseases, and obstructive sleep apnea (OSA) have been implicated in glaucoma progression and response to treatment.[14-16] Patients with vascular dysfunction, for instance, may be more susceptible to IOP fluctuations postglaucoma surgery, necessitating individualized surgical planning.[17] The interplay between systemic health and glaucoma progression suggests that treatment decisions should be guided by broader patient health considerations.[14]

Emerging data suggest that neurodegenerative diseases like Parkinson's and Alzheimer's may share common pathophysiological pathways with glaucoma.[18] As our understanding of these systemic connections deepens, future treatment algorithms may incorporate neuroprotective strategies alongside traditional IOP-lowering therapies. This shift toward holistic patient management could significantly affect how MIGS and other glaucoma treatments are tailored to individual needs.[19]

DISPARITIES IN MIGS UTILIZATION: RACIAL, GEOGRAPHIC, AND SOCIOECONOMIC BARRIERS

Despite the advancements MIGS offers in glaucoma treatment, access to these procedures remains uneven due to racial, geographic, and socioeconomic disparities. These inequities can limit the availability of MIGS to certain populations, ultimately affecting glaucoma outcomes and exacerbating health disparities.[20]

Studies have shown that African American and Hispanic patients receive MIGS at lower rates than White patients, despite having a higher prevalence of glaucoma and an increased risk of disease progression.[21] Several factors may contribute to this disparity, including differences in referral patterns, socioeconomic status, healthcare provider biases, and access to specialty care. A large-scale IRIS® Registry analysis confirmed that racial minority groups remain less likely to receive MIGS, even after controlling for disease severity, insurance status, and socioeconomic variables.[22]

In addition to racial inequities, geographic disparities pose significant challenges for MIGS accessibility. Patients in urban centers with well-equipped ophthalmic surgical facilities have greater access to MIGS, whereas those in rural or underserved areas face significant obstacles, such as a lower concentration of glaucoma subspecialists and the need for long-distance travel to high-volume surgical centers.[23,24] A 2025 study on the geographic distribution of ophthalmic subspecialists found a significant shortage of trained glaucoma surgeons in rural areas. This lack of specialists contributes to delayed surgical interventions and increased reliance on medical therapy.[24] The cost-effectiveness of glaucoma interventions also varies between urban and rural settings, further influencing treatment choices and patient outcomes.[25]

Beyond racial and geographic factors, socioeconomic status plays a critical role in determining access to MIGS. Lower-income populations often face barriers such as insurance coverage limitations, high out-of-pocket costs, and reduced access to subspecialists.[20] Even though MIGS can be a cost-effective alternative to long-term glaucoma medication, its higher upfront costs may discourage utilization among socioeconomically disadvantaged groups.[25] Addressing these barriers requires policy-driven interventions, such as expanding insurance coverage and increasing financial support for MIGS procedures. Incentivizing MIGS adoption among at-risk populations could improve access and reduce disparities, advancing equitable glaucoma care.[20,22]

In addition, both systemic and community-based solutions, such as training more glaucoma surgeons in underserved

areas, developing community-based outreach programs to educate at-risk populations on glaucoma and available treatment options, addressing misinformation and promoting early intervention, and strengthening teleophthalmology initiatives to facilitate earlier glaucoma referrals and remote consultations for patients with limited access to subspecialists.

EFFECT OF THE COVID-19 PANDEMIC ON MIGS ACCESS

The COVID-19 pandemic significantly disrupted access to glaucoma surgical care, particularly for patients in rural and socioeconomically disadvantaged communities who already faced barriers to specialty care. Elective procedures, including traditional glaucoma surgeries, were drastically reduced as healthcare systems prioritized emergency care and minimized in-person visits to curb viral transmission.[26]

In response to these limitations, there was a notable shift toward MIGS procedures, as they generally involve shorter surgical times, fewer postoperative complications, and reduced follow-up requirements compared with trabeculectomy and tube shunt surgeries.[26] Surgical volumes among fellowship-trained glaucoma subspecialists had a significant decline during the pandemic, with a global trend toward adopting less invasive options such as MIGS due to its ability to minimize healthcare visits while still providing effective IOP control.[26,27]

The pandemic underscored the vulnerability of surgical glaucoma care in times of healthcare crises and emphasized the need for resilient policies that ensure equitable access to sight-preserving procedures.

THE ROLE OF ARTIFICIAL INTELLIGENCE IN PERSONALIZED GLAUCOMA MANAGEMENT

Artificial intelligence (AI) is revolutionizing glaucoma care by enabling early disease detection, risk stratification, and personalized treatment planning. AI-driven algorithms have demonstrated remarkable accuracy in analyzing large datasets, including optical coherence tomography (OCT) scans, fundus photographs, and visual field tests, to identify glaucomatous damage and predict disease progression.[28,29] These models offer clinicians real-time decision support, facilitating earlier interventions and improved patient outcomes.

AI has also demonstrated significant applications in cataract surgery, a procedure commonly performed alongside MIGS. AI is used to calculate intraocular lens (IOL) power and predict postoperative visual outcomes.[30] Intraoperatively, AI-driven guidance systems can enhance surgical precision, particularly in cataract surgery, potentially leading to better control of IOP and improved visual outcomes.[30.31]

AI-driven models are being explored to enhance personalized glaucoma treatment strategies. By integrating predictive analytics with systemic and ocular health data, AI may provide valuable insights into individualized MIGS outcomes.[32]

Furthermore, AI-powered virtual surgical simulators are being incorporated into ophthalmology training programs, providing risk-free environments to refine surgical techniques. These tools enhance hands-on learning, potentially improving precision and patient outcomes in MIGS and other ophthalmic procedures.[32]

As AI technologies continue to evolve, their integration into glaucoma management—particularly in diagnosis, disease monitoring, and personalized treatment planning—is poised to become a cornerstone of precision medicine. These advancements offer tailored solutions that address the unique needs of each patient, ultimately improving treatment efficacy and patient satisfaction.[29]

REDEFINING GLAUCOMA CARE: THE FUTURE OF PERSONALIZED AND PREDICTIVE MEDICINE

The future of glaucoma management is shifting from reactive treatment strategies to proactive, personalized care. Advances in genetic testing, imaging, and AI are paving the way for precision medicine in ophthalmology, enabling earlier detection, improved risk stratification, and more individualized treatment approaches.[28,29] Genetic markers associated with glaucoma susceptibility are being explored for integration into clinical decision-making. Certain genetic variants linked to IOP regulation and optic nerve resilience—such as thioredoxin reductase 2 (TXNRD2), ataxin 2 (ATXN2), and forkhead box C1 (FOXC1)—may help stratify patients based on their risk of disease progression.[33] By combining genetic risk profiling with AI-powered predictive analytics, ophthalmologists could refine treatment algorithms and offer truly personalized glaucoma management tailored to an individual's genetic predisposition and disease trajectory.[28,33]

The future of MIGS will not be defined solely by device innovation but also by refining patient selection, enhancing surgeon training, and leveraging emerging technologies to optimize outcomes.[7] An important consideration is whether MIGS, particularly devices like the XEN Gel Stent, could one day replace trabeculectomy in certain settings. In developing regions, where access to highly experienced glaucoma surgeons is limited, MIGS may serve as a safer, more

scalable solution compared with trabeculectomy, reducing the burden of postoperative complications and intensive follow-up requirements.[24] However, further studies are needed to evaluate the long-term sustainability of MIGS as a trabeculectomy replacement in such settings.[7]

Moreover, patient counseling should play a more prominent role in MIGS decision-making, as it can significantly affect adherence to postoperative care and overall patient satisfaction. Ensuring that patients are well-informed about the variability in surgical outcomes, potential risks, and realistic expectations can enhance their engagement in the treatment process and improve long-term outcomes. Patients must be informed not only about expected outcomes but also about the variability in response due to individual anatomical and systemic factors. A more personalized approach to patient education may improve surgical satisfaction and adherence to postoperative care.[34]

Future strategies should focus on expanding teleophthalmology, streamlining postoperative care models, and reinforcing healthcare infrastructure in underserved regions to mitigate disparities in surgical glaucoma management.[35]

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Review Article

Secondary glaucoma following vitreo-retinal surgery

ABSTRACT

Vitreo-retinal (VR) surgeries are currently the third most common intraocular surgeries performed worldwide. The most common complication following vitreoretinal surgery is cataract, closely followed by elevated intraocular pressure. The overall incidence of secondary glaucoma post VR surgery in the literature varies widely from 20% to 60% depending on the surgical techniques and the definitions used for glaucoma. The underlying mechanism for elevated intraocular pressure determines the treatment strategy; hence, meticulous history-taking and examination are key to successful management. Mechanisms of glaucoma are varied, and often more than one mechanism can coexist in the same eye. Most eyes respond well to medical management. Laser and surgical interventions may be required in a small proportion of patients. Glaucoma drainage devices are often the ideal surgical option, and surgical techniques will need to be tailored to the requirements of individual patients.

Keywords: Glaucoma drainage device, secondary glaucoma, vitreoretinal surgery

INTRODUCTION

Vitreo-retinal (VR) surgeries are currently the third most common intraocular surgeries performed worldwide. The most common complication following vitreoretinal surgery is cataract, closely followed by elevated intraocular pressure (IOP). The overall incidence of secondary glaucoma post VR surgery in the literature varies widely from 20% to 60% depending on the surgical techniques and the definitions used for glaucoma; the reported incidence of a transient rise in IOP post VR surgery is another 7–48%.[1] The rise in IOP has been reported with all types of VR surgeries, including pars plana vitrectomy, scleral buckle, use of tamponading agents, and post intra vitreal injections.[2] The common preoperative risk factors associated with elevated IOP in VR surgeries include aphakia, pre-existing glaucoma, neovascularization, uveitis, and other surgeries performed along with par plana vitrectomy (scleral buckle, cataract surgery, or endolaser photocoagulation). The postoperative risk factors are hyphema, chronic steroid therapy, neovascularization, silicone oil (SO) in the anterior chamber, and peripheral anterior synechiae.[3,4]

Al-Jazzaf *et al.* in their study on 450 eyes post vitrectomy defined secondary glaucoma post vitreoretinal surgery as an IOP of more than 21 mmHg or the need for antiglaucoma medications at or after postoperative day 42. A rise in IOP or the need for antiglaucoma medications before post operative day 42 was termed as a transient rise in IOP.[3] These glaucomas are some of the most challenging to manage due to the aggressive nature of the disease, unpredictable response to medical therapy, limited glaucoma surgical options due to conjunctival scarring, poor visual outcomes due to the associated retinal pathology, and often the acute symptomatic presentations in the immediate postoperative period. On the other hand, vitreoretinal surgical techniques have evolved considerably in the past few decades with the introduction of transconjunctival suture-less vitrectomy (TSV), high molecular weight SO, nonexpansile concentrations of gases, and a decrease in the number of scleral buckle surgeries. The glaucoma surgeon too has an expanded armamentarium with easy access to cost-effective glaucoma drainage devices (GDDs),

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micropulse laser cyclophotocoagulation (MPCPC), conjunctival sparing minimally invasive glaucoma surgeries (MIGSs), and endocyclophotocoagulation.[5-7] This article discusses the various acute and delayed causes of elevated IOP following vitreoretinal surgeries, their mechanisms, and current principles of management.

CLINICAL EXAMINATION

The underlying mechanism for elevated IOP determines the treatment strategy, hence the significance of a meticulous history-taking and examination. Ocular examination should include the extent of conjunctival scarring, the presence and location of a buckle/sponge, areas of scleral thinning, anterior chamber depth, signs of inflammation, presence of SO in the anterior chamber, iris bombe, peripheral anterior synechiae, surgical peripheral iridectomy, new vessels, the type and position of the intraocular lens, and the axial length.[8-10]

Goldmann applanation tonometry (GAT) remains the gold standard for measurement of IOP in these eyes; however, Icare rebound tonometry and tonopen have been found to be in good agreement with GAT when GAT is precluded. It is to be noted that Icare overestimates IOP more than or equal to 23 mmHg and underestimates IOP less than 10 mmHg.[11] Noncontact tonometers and Schiotz tonometer significantly underestimate the IOP in gas-filled eyes due to compressibility of the gas.[12] Manual digital IOP measurement may also be a useful additional clinical tool in eyes with variable IOP measurements.[13]

Gonioscopy should be performed in all eyes with secondary glaucoma or a transient IOP rise when clinically feasible. Secondary angle closure is often subtle and limited to the peripheral anterior chamber, requiring a thorough and meticulous gonioscopy. A four mirror Sussman or Posner lens allows gentle gonioscopy with and without indentation, document new vessels, peripheral anterior synechiae, presence of silicone oil, and trabecular meshwork pigmentation. However, gonioscopy should be avoided in eyes with inflammation, hyphema, and corneal pathology.[8]

MECHANISM OF GLAUCOMA

Mechanisms of glaucoma post VR surgery are varied, and often more than one mechanism of glaucoma can coexist in the same eye. The common mechanisms of secondary glaucoma and transient IOP rise are listed in Table 1.[4,14,15]

Table 1: Mechanisms of glaucoma post vr surgery

Transient IOP rise or Acute glaucoma	Chronic glaucoma
Inflammation	Trabecular meshwork degeneration
Hyphema	due to silicone oil
SO overfill	Emulsification of SO
SO in the anterior chamber	Neovascular glaucoma
Trabeculitis	Steroid response
Steroid response	Ghost cell glaucoma
Neovascular glaucoma	Tight buckle
Ghost cell glaucoma	Secondary angle closure glaucoma
Tight buckle	Secondary open-angle glaucoma
Suprachoroidal hemorrhage	
Secondary angle closure	
with/without pupillary block	

GLAUCOMA FOLLOWING SCLERAL BUCKLE

Scleral buckle is commonly used in the management of rhegmatogenous retinal detachment. This involves placement of encircling bands or sponges to reduce the traction on a retinal tear and facilitate subretinal fluid absorption. The reported incidence of secondary angle closure following a scleral buckle is 1.4%–4.4%,[16] and that of narrowing of the anterior chamber without secondary angle closure ranges from 14.5% to 50%.[17] The proposed mechanism of secondary angle closure is the constriction of the vortex veins by the scleral buckle that impedes the venous drainage and causes choroidal congestion, swelling, and anterior rotation of the ciliary body. This in turn causes narrowing of the anterior chamber by the forward movement of the lens-iris diaphragm. The choroidal congestion itself can cause exudation of fluid into the suprachoroidal space and cause suprachoroidal effusion.[18] The risk factors associated with shallowing of the anterior chamber in scleral buckle surgeries are pre-existing angle closure, placement of the encircling band anterior to the equator, high myopia, and postoperative ciliochoroidal detachment.[19] The rise in IOP is often transient and responds well to topical cycloplegics, steroids, and aqueous suppressants. Loosening a tight buckle may also help reduce the IOP.

GLAUCOMA FOLLOWING PARS PLANA VITRECTOMY

Pars plana vitrectomy (PPV) can cause IOP rise due to trabeculitis, fibrin membrane from postsurgical inflammation leading to seclusio and occlusio pupillae, steroid response, neovascularization, or presence of ghost cells [Figure 1]. Endolaser photocoagulation can cause choroidal congestion and ciliary body rotation causing anterior movement of the iris lens diaphragm and angle closure.

Studies have shown a delayed IOP response in phakic eyes as opposed to pseudophakic/aphakic as the lens acts as an anatomical barrier to the inflammatory mediators from the posterior segment.[20] The other risk factors that can add to the IOP rise are the placement of a scleral buckle either intraoperatively or before vitrectomy, intraoperative scatter

endo photocoagulation, and an intraoperative lensectomy.[1] Although PPV as such has been proposed to be a risk factor for long-term IOP rise and ocular hypertension, a recent systematic review by Miele *et al*.[21] found the current evidence to be heterogeneous and inconsistent regarding the same.

GLAUCOMA FOLLOWING SILICONE OIL TAMPONADE

SO has been used as an adjunct for internal tamponade in retinal surgeries for the past 5 decades. It is a synthetic polymer made of repetitive Si-O units.[22] Secondary glaucoma has been reported in 7%–48% of eyes that undergo SO tamponade.[23] The mechanisms include

1) SO overfill with or without SO in the anterior chamber
2) Pupillary bock causing secondary angle closure glaucoma [Figure 2a and b]
3) Deposition of the denatured silicone oil in the trabecular meshwork [Figure 3]
4) Inflammation or exacerbation of pre-existing glaucoma
5) SO migration to the optic nerve causing degeneration.[10,24,25]

SO fill in the anterior chamber should be ruled out in aphakic eyes and pseudophakic/phakic eyes with a capsular or zonular breach. A complete SO-filled anterior chamber is characteristically devoid of convection currents and demonstrates a golden sheen over the iris [Figure 4].

Fumitaka Ando in 1985[26] proposed an inferior surgical peripheral iridectomy (PI) in all aphakic eyes with SO to prevent a pupillary block. An inferior PI is preferred since the specific gravity of SO being less than that of aqueous, SO bubbles tend to float superior, which in turn can block surgical iridectomy created superiorly. Al-Jazaff *et al*.[3] in a more recent study reported no pupillary block glaucoma when an inferior surgical PI was done in all aphakics and in eyes with an anterior chamber IOL.

The reported risk factors associated with SO-induced glaucoma are pre-existing glaucoma, diabetes mellitus,[23,26] aphakia, physiochemical properties of SO,[27] and the duration of tamponade. Medical management is successful in many of these eyes. Some may warrant an early partial or complete silicone oil removal (SOR) due to persistently high IOP with maximal medical therapy.

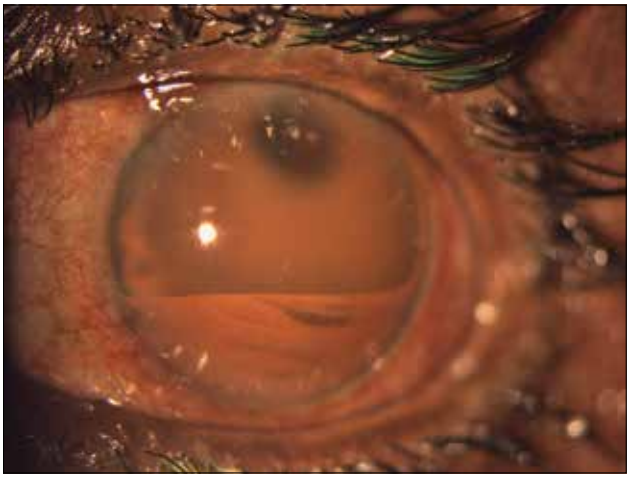


Figure 1: Ghost cell glaucoma with khaki-colored cells in anterior chamber

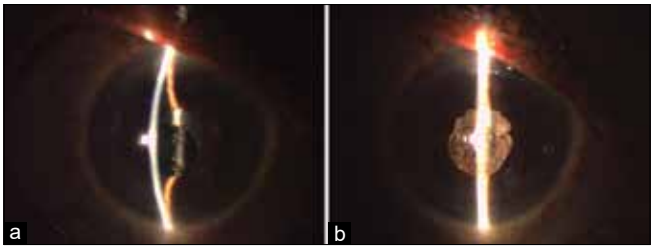


Figure 2: (a) Secondary angle closure with pupillary block before laser peripheral iridotomy. (b) Secondary angle closure with pupillary block after laser peripheral iridotomy

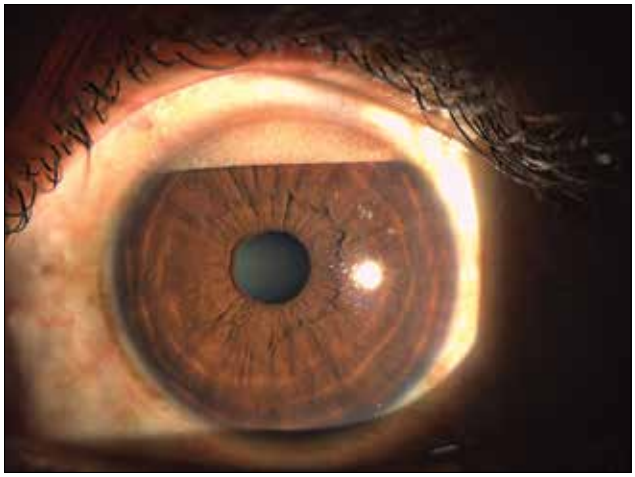


Figure 3: Reverse hypopyon due to emulsified SO

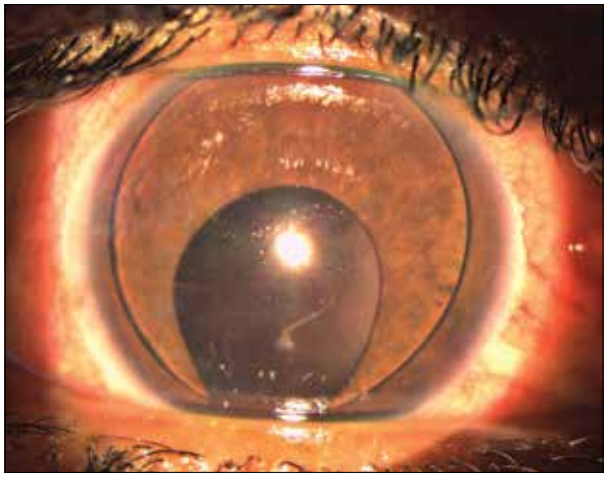


Figure 4: SO in the anterior chamber

GLAUCOMA FOLLOWING GAS TAMPONADE

Expansile gases used in VR surgery, sulfur hexafluoride (SF6) and perfluoropropane, (C3F8) have the advantage of spontaneous resorption as opposed to SO. While SF6 expands two times the injected volume in 1–2 days, C3F8 expands four times in 3–4 days. The non-expansile concentrations of SF6 and C3F8 are 18% and 12-14% respectively and are associated with a lesser risk of IOP rise.[28] The incidence of elevated IOP ranges from 6.1% to 67% after SF6 and from 18% to 59% after C3F8 injections.[29] The expansile property and the longevity of the gas are two factors that contribute to IOP spikes. Mostly, these cause an open angle glaucoma. Secondary angle closure glaucoma with or without pupillary block can also occur due to the mechanical effect of the gas, which leads to anterior pushing of the lens iris diaphragm. Patients with gas tamponade should be instructed to avoid mountain climbing and air travel as the gas expansion on ascent with the drop in atmospheric pressure can precipitate a sudden rise in IOP. Nitrous oxide anesthesia, scuba diving, and hyperbaric oxygen therapy also are to be avoided for similar reasons. Meticulous supine positioning can reduce the incidence of pupillary block glaucoma and gas cataracts.

GLAUCOMA FOLLOWING INTRAVITREAL INJECTIONS

Intravitreal steroids

Intravitreal corticosteroid injections have been used in the treatment of diabetic macular edema, exudative macular degeneration, pseudophakic cystoid macular edema, macular edema associated with retinal vein occlusion, and chronic uveitis. The most common adverse effect reported following intravitreal steroid is an elevated IOP.[30] The most common steroids used for intravitreal injection are triamcinolone acetonide, fluocinolone acetonide, and dexamethasone, and the two modes of delivery are direct injection and the implantation of a sustained-release device. The reported incidence of elevated IOP after intravitreal steroids is 50%, with IOP rise occurring after 1–2 months of injection.[31] An acute elevation of IOP can occur due to the expansion of globe volume during bolus administration; the long-term rise in IOP and steroid induced glaucoma are most commonly reported with triamcinolone acetonide.[32] The risk factors for significant IOP elevations are high myopia, high baseline IOP, multiple injections, the presence of triamcinolone crystals in the anterior chamber, young age, uveitis, family history of glaucoma, and intravitreal steroid implants.[33,34] Trabeculectomy, MIGS, or GDD may be indicated in patients refractory to medical therapy. Patients with unabsorbed depot of the steroid in vitreous will benefit from vitrectomy for removal of the same.

Intravitreal Anti-Vascular Endothelial Growth Factor (VEGF) injection

Intravitreal anti-VEGF injections have been used widely for the treatment of wet AMD, diabetic retinopathy, retina vein occlusion, retinopathy of prematurity, neovascular glaucoma, and other retinal diseases. The usual volume used is 0.05 ml, and multiple injections over many months and years are often required.[35,36] A transient elevation of IOP after intravitreal anti-VEGF is common and has been attributed to an increase in intraocular fluid volume. The reported incidence of IOP >30 mmHg 5 s post injection is 88.9%; this returns to normal levels 30–60 min post injection.[37] The risk factors for a long-term rise in IOP requiring IOP-lowering medications are a high baseline IOP, multiple injections, and short intervals between injections. The incidence of sustained elevation of IOP is 11%.[38-40] The proposed mechanism is the accumulation of anti-VEGF molecules in the trabecular meshwork or Schlemm's canal, causing a direct obstruction or a change in outflow facility.[41]

MANAGEMENT

Medical management

Medical therapy includes topical and systemic antiglaucoma medications, topical steroids to reduce inflammation, and topical cycloplegics, which allow a posterior movement of the iris lens diaphragm. Most patients respond well to medical treatment, Al-Jazzaf *et al*.[3] reported 78% of patients with glaucoma post PPV and SO injection to respond well to medical management. Miotics should be avoided as they cause a forward movement of the lens iris diaphragm and increase inflammation.

Intravenous mannitol can be used to control acute IOP rise and has been proven to cause a significant IOP reduction in vitrectomized eyes, comparable to that in nonvitrectomized eyes. This has been postulated to be due to the IOP lowering effect of mannitol by the direct osmotic mechanism (removal of water from the aqueous humor and vitreous cavity) as well as central mechanism via osmoreceptors in hypothalamus.[42,43]

Laser treatment

In patients with secondary angle closure with pupillary block or an anterior chamber filled with SO and no patent PI, an inferior Nd: YAG laser peripheral iridotomy should be performed to relieve pupillary block. In a retrospective observational study by Senthilkumar *et al*.,[44] the incidence of pupillary block after vitreoretinal surgeries was 0.87%, which resolved with Nd: Yag laser iridotomy in 82% of patients, while 18% needed additional laser or surgical interventions like surgical peripheral iridectomy, trabeculectomy, or diode cyclophotocoagulation alone or in combination with SOR.

The reported risk factors for pupillary block in this study were diabetes mellitus, combined cataract surgery with vitreoretinal surgery, multiple vitreoretinal surgeries, use of 1000 cs SO tamponade, and intraoperative endolaser.

Selective laser trabeculoplasty has been used successfully to reduce IOP postvitreoretinal surgeries in patients with open angles.[5,6] It reduces IOP through the release of chemotactic factors and cytokines, which increases and upregulates the macrophages in the trabecular meshwork, thereby increasing the permeability of trabecular meshwork.[45]

Transscleral cyclophotocoagulation (TSCPC) is a cyclodestructive procedure conventionally used for refractory glaucomas with poor visual potential. A limited TSCPC with lower power settings may be beneficial for a short-term control of IOP in eyes post PPV with visual potential that are at high risk for surgery or not amenable to other surgical options due to extensive conjunctival scarring. Bloom *et al*.[6] reported an overall success rate of 66% after TSCPC for refractory glaucomas. Gangwani *et al*.[46] reported that transscleral TSCPC is useful for short-term IOP control in refractory glaucoma post PPV with silicone oil; however, in the long term, they may require additional antiglaucoma medications. Complications of TSCPC include hypotony, macular edema and phthisis bulbi, the power used should be carefully titrated for optimal IOP control in these patients.

Endo cyclophotocoagulation by direct visualization and photo coagulative destruction of the ciliary processes causes less collateral damage compared to TSCPC. Chen *et al*.[47] evaluated the efficacy of endo cyclophotocoagulation for refractory glaucoma and found the same to be a safe and effective therapeutic modality.

Surgical management

Scarring of the conjunctiva from prior surgeries pose a major challenge for glaucoma surgery in these patients. In a study by Inoue *et al*.,[48] the probability of success of trabeculectomy with mitomycin c (MMC) post vitrectomy was 55% after one year, and the risk factors associated with failure were neovascular glaucoma and a high preoperative IOP. In addition, high myopes were found to be at risk for postoperative hypotony and related complications. However, trabeculectomy with MMC may still be an acceptable primary surgical option in patients with intact superior conjunctiva.

SO-induced glaucoma can be secondary to multiple mechanisms. In eyes with SO overfill with no or early emulsification of oil, a partial or complete SOR can be considered, provided the risk of redetachment is low. Early SOR may reverse the trabecular meshwork changes and prevent further damage. Jonas *et al*.[31] found that 93.4% of eyes with a secondary IOP rise after SO injection had IOP controlled after SOR alone. Long-term contact of the emulsified oil with the trabecular meshwork causes trabecular meshwork sclerosis and collapse, after which SOR alone may not contribute much to the reduction of IOP.[10] In patients with long-standing contact of the trabecular meshwork with SO, uncontrolled IOP on medical therapy, and secondary angle closure, other glaucoma surgical options should be considered. Trabeculectomy and glaucoma drainage devices in SO-filled eyes entail the risk of blockage of the ostium/tube with SO and hence are preferably done post SOR.[4] Budenz *et al*.[49] studied the outcome of glaucoma surgery with and without SOR in vitrectomized eyes and found that eyes which undergo concurrent glaucoma surgery with SOR had a high risk of hypotony.

Glaucoma drainage devices are often the ideal surgical option in vitrectomized eyes. The availability of at least one quadrant of intact conjunctiva for plate implantation is a prerequisite. The presence of a scleral buckle or a sponge in the quadrant of GDD implantation may pose a challenge, and these should be meticulously documented in the preoperative evaluation. GDD can often be implanted over the scleral buckle after careful dissection. In the case of a large sponge, explantation of the same could be considered in consultation with the retina surgeon or an alternate site for the GDD planned.[15] Sidoti *et al*.[50] described the implantation of a silicone tube to shunt the aqueous humor from the anterior segment to the fibrous capsule of a pre-existing episcleral encircling band. Complete or partial success was reported in 85% of the eyes. Scott *et al*.[51] proposed excision of the fibrous capsule of the encircling band and direct suturing of the implant to the episcleral band allowing a larger area of filtration. Trimming of the episcleral plate of the Baerveldt or Aurolab Aqueous Drainage Implant (AADI) implant and the use of smaller GDD like AADI 250 or FP8 model of the Ahmed Glaucoma Valve are other surgical options in eyes with significant conjunctival scarring.

Conventionally, the GDD is inserted into the anterior chamber or into the ciliary sulcus. A longer tube or inferior placement of the tube can reduce the likelihood of tube blockage by SO. In eyes with compromised corneal endothelium, shallow anterior chamber, or when combined with PPV, the placement of the tube in pars plana may be considered.[52]

In a retrospective series of 15 patients with secondary open glaucoma after SOR, Aktas *et al*.[53] reported a qualified success of 93.3% and a complete success of 26.7% with

gonioscopy-assisted transluminal trabeculotomy (GATT) over a mean follow-up period of 37.5 months. The technique being bleb-independent and sutureless, the conjunctiva is spared for a future GDD or trabeculectomy if indicated. However, studies with larger sample sizes and longer follow-up are required to understand the role of MIGS in the management of post vitrectomy secondary glaucoma.

CONCLUSION

Transient IOP rise and secondary glaucoma are very common complications following VR surgeries. Patients with SO tamponade and scleral buckle are at high risk and should be closely monitored. Considering the multiple mechanisms involved in glaucoma following VR surgery, a meticulous clinical examination is key to successful management. Secondary angle closure is often subtle and dynamic and should be ruled out by gonioscopy. Most eyes respond well to medical management. Laser and surgical interventions may be required in a small proportion of patients. GDD are often the ideal surgical option, and surgical techniques will need to be tailored to the requirements of individual patients.

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Declaration of patient consent

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

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Post-operative vision loss after coronary artery bypass graft

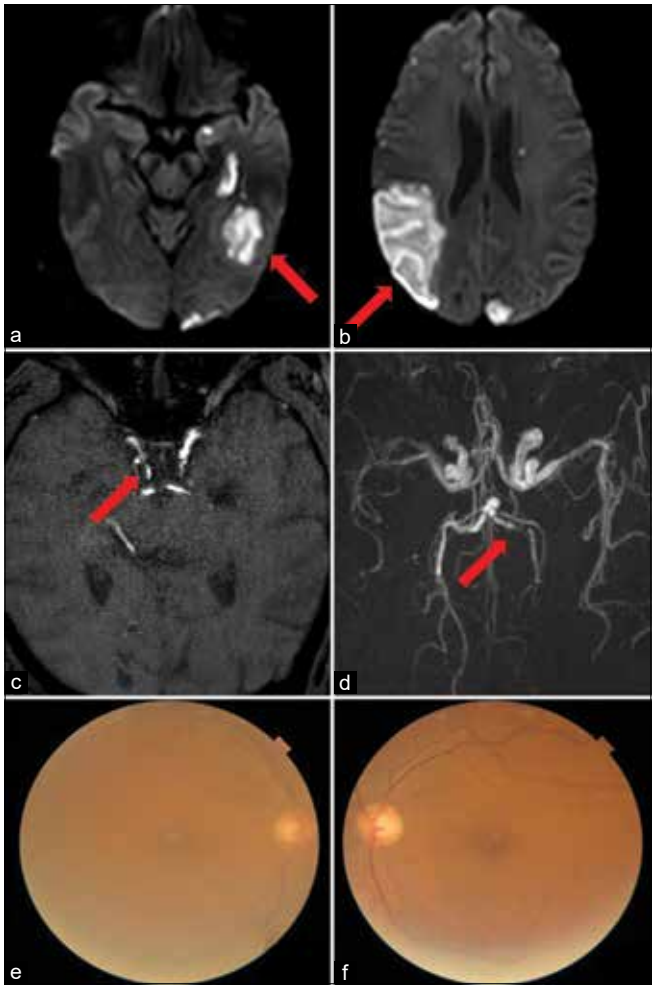


Figure 1: MRI Brain showing multiple subacute infarcts (red arrows) involving the (a) right parieto-temporal lobe, (b) left parieto-occipital lobe, temporal lobe and (c) hippocampus. (d) MRA of carotid and cerebral vessels displaying focal severe (90%) narrowing of the P1 segment of posterior cerebral artery with attenuation of its cortical branch, and focal (60%–70%) luminal narrowing seen in the distal cavernous and clinoid parts of right intracranial internal carotid artery (red arrow). Normal fundus examination (e and f)

CLINICAL SCENARIO

A 61-year-old hypertensive gentleman with a prior history of coronary angioplasty, presented with triple vessel disease and a left ventricular ejection fraction of 50%. He underwent a coronary artery bypass graft (CABG) surgery, and was recovering well. On his 2nd post-operative day, once he regained consciousness, he complained of loss of vision in both his eyes. Visual acuity was only perception of light in both eyes. He was irritable and uncooperative for assessment—bed-side examination revealed a normal anterior segment with a central, circular, briskly reacting pupil, and fundus examination was unremarkable. Blood tests, cardiological and neurological evaluation, and magnetic resonance imaging (MRI) orbit were within normal limits.

What would be your next step?

a. Visual evoked potential (VEP)s
b. Doppler study of carotid and cerebral vessels
c. MRI brain with magnetic resonance angiography (MRA) carotid and cerebral vessels
d. Fundus fluorescein angiography (FFA).

Correct answer

Choice C - MRI brain with MRA carotid and cerebral vessels

MRI brain [Figure 1a-c] and MRA of carotid and cerebral vessels [Figure 1d] are depicted. A diagnosis of embolic stroke was made, and he was managed as per stroke protocol. A week later, his vision improved to counting fingers close to face. He refused any further investigations, and was discharged. Six months later, his vision improved to 6/60 in both eyes with few scotomata. Fundus examination remained normal [Figure 1e and f]. He was advised perimetry, but was unable to perform the test.

Post-operative visual loss after cardiac surgical procedures has a reported incidence of 0.06%–1.3%. In the absence of any anterior or posterior segment findings, a cerebral infarction due to hypoperfusion or micro-emboli may be implicated in its pathogenesis.[1] Cortical blindness may ensue from damage to the primary visual cortex in the parieto-occipital lobe. Embolisms in CABG may develop because of handling of the heart and aorta, atrial fibrillation, hypercoagulability, or substance from the bypass pump. Risk factors include hypertension, diabetes mellitus, renal failure, smoking, Chronic obstructive pulmonary disease (COPD) and cardiac disease.[2] The prognosis is benign, with recovery of good visual function, in majority of the cases.[3]

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Can this happen to you?

A 32-year-old, one-eyed gentleman comes to the retina out-patient department with sudden loss of vision in the seeing eye. Subsequently, he undergoes detachment surgery in that eye and regains 6/12 vision. After about 2 months, he again comes with sudden loss of vision in the seeing eye. Vision now is counting fingers at 3 metres. Examination looks normal, with retina attached. OCT of the eye appears normal. Neurology work-up, including MRI, comes up normal.

The next day, he informs the doctor that he has been on the oral medication *febuxostat* for hyperuricemia (prescribed by a local doctor) for the past 5 days, after which he lost his vision. ChatGPT supplies him with the necessary evidence for his claims, including successful litigation cases in similar scenarios. He intends to sue the general practitioner who prescribed *febuxostat*, and is convinced that his loss of vision is due to the side effect of the drug.

The drug was prescribed to him at 80 mg daily dose, instead of the recommended 40 mg dosage. He was never informed of the possible side effects of the drug, and hence feels medication was started without his consent.

LEGAL SCENARIOS THAT NEED ANSWERS

Is consent required from the patient before starting a drug with possible side effects?

What is the legal standing for such a case in the consumer court?

Consent is perhaps the only principle that runs through all aspects of health care provisions today. It also represents the legal and ethical expression of the basic right to have one's autonomy and self-determination. If a medical practitioner attempts to treat a person without valid consent, then he will be liable under both tort and criminal law.

Often medical practitioners ask for precise prescriptions for the situations when written consent is needed. It is interesting to note that what law demands is mere consent and not written consent and does not prescribe such requirement on a mandatory basis. In fact, the medical practice itself determines the need for written consent. There is no mandate that a doctor should always obtain written consent and failure of which would hold him liable. However, if there is written consent, the medical practitioner would have greater ease in proving consent in case of litigation. To standardize the practice, the Medical Council of India (MCI) has laid down guidelines that are issued as regulations in which consent is required to be taken in writing before performing an operation.[1]

However, the nature of the information that a patient must have in order to give informed consent is a debatable question, as the American and English viewpoints differ to some extent.

Informed consent from the American sense is often described from the viewpoint of a prudent patient, popularly known as the prudent patient test. In this approach, the highest respect for the patient's right of self-determination about a particular therapy is recognized. This will lead to a so-called objective test of disclosure wherein the doctor will keep in mind the patient and disclose all such information which is required to be given.

In contrast to this, the English approach is doctor centric, which is also popularly narrated as the prudent doctor test of disclosure. Here, the doctor is taken as a professional-man endowed with greater prudence to protect the right interest of the patient and bestowed with the final right to decide what information shall be divulged to the patient considering the circumstances and how much information is to be divulged.[1]

In the Indian context, there is a traditional notion of paternalism prevalent among the members of the medical fraternity. It is a notion where the doctor takes-up the role of a parent of the patient and starts deciding on behalf of the patient himself. Unfortunately, the law does not accept this notion. The first priority of law is always the right of autonomy of the patient provided he is endowed with necessary capacity.

Informed written consent while prescribing medications is an open-ended question. Informed written consent while undergoing surgical procedures is accepted practice while the former still remains in the gray zone.

A randomized control trial on patient recall was conducted. Though an informed consent regarding possible side effects

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of the drug prednisolone was taken, the study subjects showed poor recall of side effects when questioned later.[2]

It is not always not practical to obtain an informed written consent while prescribing regular medications, but it must always be kept in mind. *Azathioprine*, an immunosuppressant, has adverse side effects (including bone marrow suppression) which could be fatal to the patient. It is always advisable, in such a situation, to obtain a written, informed consent before starting such medications.

Wrong dosage and related complications are always a case of medical negligence, if it can be proven in a court of law.

CONCLUSION

Informed consent is a must before starting any kind of treatment. It can be an implied or express consent. It can be an oral consent or written informed consent. Written informed consent is usually mandated only for surgical procedures. While using medications with fatal side effects, it is always advisable to obtain an informed written consent since it can be an added advantage to the doctor if at all a litigation arises.

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Original Article

Intraocular pressure variations in rhegmatogenous retinal detachment: Does it point towards an alternate biomechanical response?

ABSTRACT

Objective: To evaluate the variations in intraocular pressure and its relation with the severity and chronicity of Rhegmatogenous retinal detachment (RRD), as well as the type of break and presence of myopia. **Methods:** Design: A descriptive cross-sectional study; sample size 75 (4 pq/d2); duration 1 year; exclusion-combined Retinal Detachment (RD), exudative Rd, associated choroidal detachment, and anterior Proliferative vitreoretinopathy (PVR). Parameters considered were age, duration, IOP by Applanation Tonometry (AT), severity, chronicity, PVR changes, location, number of breaks, and presence of myopia. **Results:** In all, 72% (n = 54) were males; 25 (33%) patients had features of chronicity (PVR, demarcation line, and atrophic retina). Fifty-one patients (68%) had subtotal RD; Superotemporal quadrant rhegma was present in 30 patients (40%). Normal IOP on AT was observed in 42.7%, lower intraocular pressure (IOP) in 37.3%, and hypotony in 20%. Hypotony was more common among total RRD (P = 0.027). None showed evidence of globe distortion. The mean duration of RRD was more among those with total RRD (P = 0.008) and among those with unidentified breaks (P = 0.031). The mean IOP among the total RRD was 9.42 mm Hg (standard deviation [SD] = 3.69, P = 0.011. The mean IOP showed a negative correlation with the duration among those with fresh RD (P = 0.041), and the presence of HST (P = 0.014). The mean IOP showed a positive correlation with age among RRD with features of chronicity (P = 0.012). **Conclusion:** The relationship of intraocular pressure dynamics with the severity, chronicity, and type of breaks suggests the possibility of an alternate hypothesis for lower IOP in RRDs.

Keywords: Biomechanical, intraocular pressure, Kerala, rhegmatogenous retinal detachment, variations

INTRODUCTION

The retina is a major barrier to fluid movement from or into the choroid. In case of retinal detachment, this property of the retina is lost and this is responsible for intraocular pressure (IOP) changes in retinal detachment.[1] Various mechanisms responsible for these observations have been proposed. This study intends to evaluate the intraocular pressure changes in rhegmatogenous retinal detachment and discuss the possible pathophysiology behind such changes.

Objectives

To evaluate the variations in intraocular pressure and its relation with the severity and chronicity of Rhegmatogenous retinal detachment (RRD), as well as the type of break and presence of myopia.

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MATERIALS AND METHODS

A descriptive cross-sectional study was conducted among those patients coming to the Ophthalmology outpatient department (OPD) with rhegmatogenous retinal detachment. Approval of the institutional research committee was obtained. There was no financial burden for the stakeholders. A sample of 75 cases was selected based on the formula (4pq/d2); p being a prevalence of normal IOP among RRD as 25%, q equals

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(100-p), and a precision of 10%. Exclusion criteria: combined Retinal detachment (RD), exudative RD, associated choroidal detachment, and anterior Proliferative vitroretinopathy (PVR).

Detailed history including demography, duration of illness, and symptoms was taken. The examination included IOP measurement by Applanation tonometry (AT), wet retinoscopy for refractive errors, and fundus examination to assess severity, chronicity, proliferative vitreoretinopathy changes, location/number of breaks, and myopic changes. IOP values measured by AT were grouped into three categories: normal: IOP >11 mm of Hg, low hypotony: IOP between 10.9 and 6.4 mmHg, and hypotony: IOP <6.5 mmHg.

All these details were added to an Excel sheet and statistical analysis was performed using the SPSS software version 18.

The study has been approved by the institutional ethics committee on Oct 2014.

RESULTS

Data representation

Out of 75 cases studied, the mean age was 57.32 years and 72% of them were male. A total of 51 patients (68%) had subtotal RD and superotemporal quadrant rhegma was observed in 30 patients (40%). Thirty-two cases (42.7%) had IOP above 11 mmHg, 28 patients had IOP in the range of 10.9 to 6.4 mmHg (37.3%), and 15 cases had hypotony IOP <6.5 mmHg (20%). The frequency of distribution of the cases based on IOP in each category is given in Figure 1.

Twenty-five (33%) patients had features of chronicity such as proliferative vitreoretinopathy, demarcation line, and atrophic retina. The mean duration of RRD was more among those with total RRD ($P = 0.008$) and among those with unidentified breaks ($P = 0.031$).

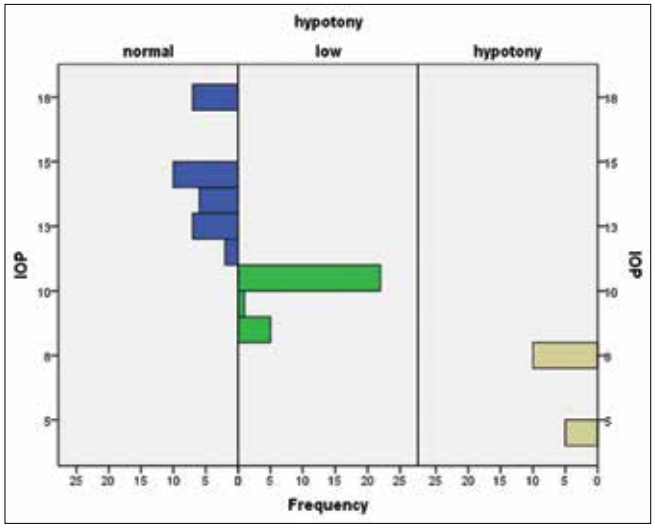


Figure 1: The frequency of distribution of cases based on IOP categories

The distribution of cases among different categories of IO*P* values is given in Table 1.

Those with lower IOP were older. The duration of the disease was longer among those with hypotony. IOP decreased with age, duration, chronicity, and severity of RD. The prevalence of hypotony was higher among those with chronic RD and total RD. This negative relation was statistically significant only for total RD ($P = 0.027$).

The distribution of cases based on IO*P* values is given in Figure 2.

It showed that the mean IOP decreased with increasing severity of the disease, myopia >6D, horseshoe tear, and chronicity. However, a statistically significant negative relation was observed only with total RD and IOP. The mean IOP among total RRD was 9.42 mm Hg (SD = 3.69, $P = 0.011$).

IOP and age of patients

It was observed that IO*P* values decreased as age increased. Table 2 represents the multivariate relation between IOP and age with other factors, namely type of RD, severity of RD, type of breaks, and the presence of myopia.

The mean IOP showed a statistically significant positive correlation with age among RRD with features of chronicity ($P = 0.012$).

IOP and duration of the disease

It was observed that the IO*P* values had a negative relation with the duration of the disease. Figure 3 represents the multivariate relation between IOP and duration with the other factors namely type of RD, severity of RD, type of breaks, and the presence of myopia.

We observed that when the duration of RD increased, IOP decreased in subjects with horseshoe tear (HST) and with fresh RD, that is, IOP had a negative relation with fresh RD and HST. No relation was found between IOP with old RD, other rhegma, and severity of myopia.

Pearson correlation between duration, IOP, and fresh RD was found to be − 0.291 ($P = 0.041$), and that with HST was −0.329 ($P = 0.01$).

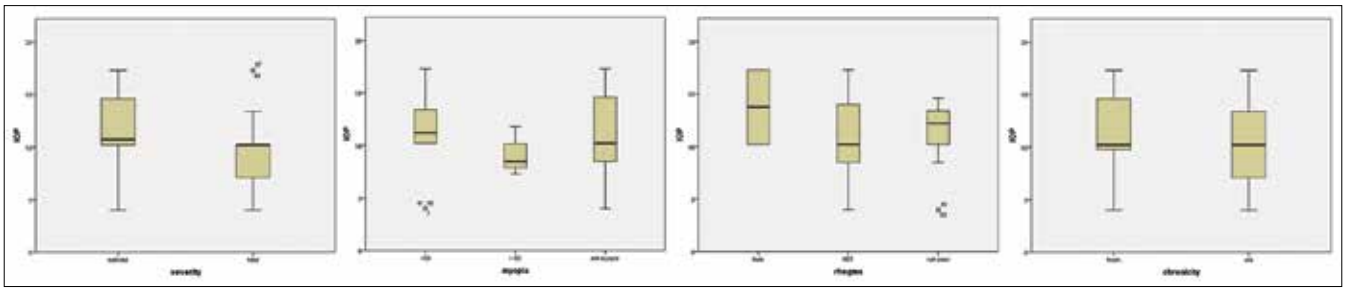


Figure 2: The relation of IOP with the presence of myopia, the severity of RD, the presence of rhegma, and the chronicity of RD

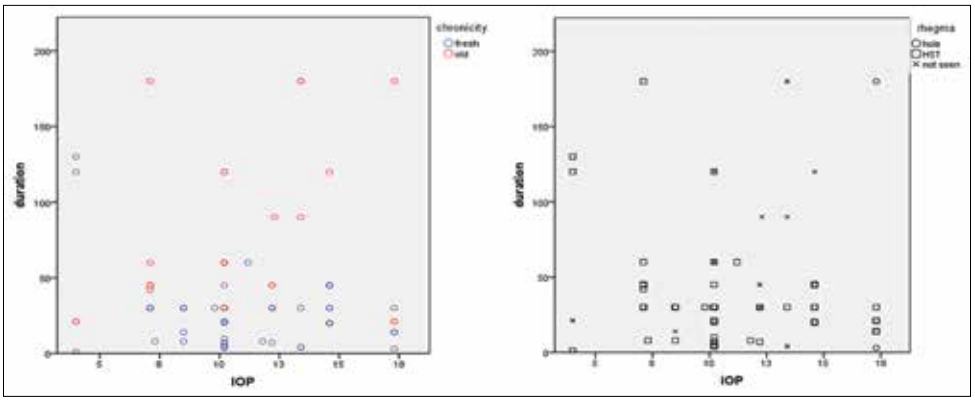


Figure 3: Scatter plots showing the IOP changes with the duration of disease when chronicity and type of regma are considered

Table 1: Distribution of cases among different categories of IOP

Factors	Normal IOP	Low IOP	Hypotony	*P*
Age years mean (SD)	56.03 (10.64)	57.93 (8.54)	58.93 (6.40)	0.547
IOP mmHg mean (SD)	14.25 (1.92)	9.88 (0.661)	6.08 (1.52)	0.000***
Duration in days mean (SD)	48 (50.40)	30.96 (30.27)	53.87 (50.18)	0.187
Fresh RRD *n* (%)	22 (44)	21 (42)	7 (14)	0.162
Old RRD *n* (%)	10 (40)	7 (28)	8 (32)	
Subtotal RRD *n* (%)	27 (50)	20 (37)	7 (13)	0.027***
Total RRD *n* (%)	5 (23)	8 (38.1)	8 (38.1)	

***P-value statistically significant (<0.05).

Table 2: Correlation between IOP and age among various factors

Factors	Pearson	*P*
Type of RD		
Fresh	-0.127	0.378
Old	-0.495	0.012***
Severity of RD		
Subtotal	-0.047	0.737
Total	-0.166	0.472
Rhegma		
Hole	-0.590	0.410
HST	-0.054	0.696
Not observed	-0.290	0.276
Presence of myopia >6D	-0.98	0.402

***P-value statistically significant (<0.05).

DISCUSSION

Studies show RRD is associated with low IOP.[2] The theory behind lower IOP in rhegmatogenous retinal detachment is still a controversial issue. We observed that 42.7% had IOP above 11 mmHg (mean IOP = 14.25 mmHg). Only 20% had IOP less than 6.5 mmHg. None of the eyes lost the ocular tone even among cases with a long duration of the disease. Hypotony from internal aqueous loss has been reported to occur in cyclodialysis cleft and retinal detachment.[3] The subretinal fluid passing through the tear was absorbed faster via the uveoscleral outflow. Relative hypotony in the affected eye was present in 40% of patients with uncomplicated unilateral retinal detachments. The average pressure asymmetry noted by the authors was only 1.3 mm Hg; however, in one out of every four patients, the difference was 3 mm Hg or more. Relative hypotony persisted for longer than 6 months after the scleral buckling operation,[4] occurring even in patients who did not exhibit hypotony preoperatively. Comparison of either eye is essential to identify the relative hypotony.

The extent of vitreous traction is responsible for the promotion of the flow of vitreous fluid into the subretinal space:[5] Either the traction itself causes lower pressure in the subretinal space or the traction positions the retinal hole so that fluid is better directed into the subretinal space with eye movements. The extent of vitreous traction at the break has to be quantified to identify the contribution of this factor in lowering IOP. A lower IOP among those with PVR can be attributed to this explanation.

Those with lower IOP were older. The duration of the disease was longer among those with hypotony. The prevalence of hypotony was higher among those with chronic RD and total RD. This negative relation was statistically significant only for total RD ($P = 0.027$).

The sclera provides stable mechanical support to vulnerable internal ocular structures such as the retina and optic nerve, especially under the complex, dynamic loading conditions imposed by eye movements, and fluid pressures. The changes in the collagen-rich scleral extracellular matrix (ECM) and its resident cells alter the biomechanical properties of the sclera and are considered factors responsible for reduced IOP with aging.[6] In RRD, alterations in the biomechanical properties of the sclera have been studied.[7] The sclera loses its compliance and rigidity similar to high myopia. This can result in falsely low IO*P* values. The conventional Goldmann applanation tonometry is found to underestimate post-buckle IOP measurements due to corneal biomechanics changes. The author suggests the use of an ocular response analyzer for IOP measurements in such cases. Hence, the appearance of normal contour despite the mean IOP being 6.08 mm of Hg among the group with hypotony.

Diurnal fluctuations of the choroidal thickness, though small, have been noticed to be stable and consistent. A negative relation with IOP has been observed in animal studies by Chakraborty *et al*.[8] In acute RD, increased choroidal thickness has been reported due to inflammation. In chronic RD, choroidal thickness is not elevated.[9] This explains the significantly lower IOP in acute RRD and no relation with chronic variations in retroocular blood flow and increased choroidal thickness.[10] This cyclical process may augment the uveoscleral outflow, resulting in lower IOP in acute RRD.

Chakraborty *et al*.[8] have reported that lack of form deprivation can result in significant axial elongation and increased choroidal thickness in chickens. Loss of visual function following RRD may also lead to such changes.

Persistent subretinal fluid is reported to be associated with increased subfoveal choroidal thickness among RRD cases post-surgery.[11] Choroidal thickness may be a predictor for persistent SRF and lower IOP in such cases.[12]

Prostaglandin and chemokine levels are known to increase in concentration in the subretinal fluid after RRD. These inflammatory mediators increase uveoscleral outflow.[13] The effect on uveoscleral outflow by prostaglandins, which are potent vasodilators, may explain increased choroidal thickness and decreased IOP after RRD.

We observed that the mean IOP decreased with increasing severity of disease, myopia > 6D, horseshoe tear, and chronicity. However, a statistically significant negative relation was observed only among those with total RD. The mean IOP among total RRD was 9.42 mm Hg (SD = 3.69, $P = 0.011$).

We observed that when the duration of RD increased IOP decreased with HST and with fresh RD, that is, IOP had a negative relation with fresh RD and HST. No relation was found between IOP with old RD, the presence of atrophic holes, and the severity of myopia.

The mean IOP showed a statistically significant positive correlation with age among RRD with features of chronicity ($P = 0.012$).

Biomechanics is the study of the relationship between forces and function in living organisms.[14] Over the past few decades, basic science research in ophthalmology mostly confirmed that ocular biomechanics could explain in part the mechanisms involved in almost all major ophthalmic disorders including retinal detachment. Scleral wall shrinkage following hypotony has been reported. The ultrastructural remodeling can influence the IOP measurements.[6]

The hydrostatic pressure gradient (ΔP) between ocular vessels and tissue is determined by the difference between the blood pressure in the vessels and the IOP. In the normal eye, the choroidal venous pressure is close to the IOP. When IOP falls, ΔP probably increases and this would cause water to diffuse from the blood vessels into the tissue. Because the choroidal vessels do not regulate the arteriolar diameter, they would receive the full rise in ΔP. The retinal vessels, in contrast, constrict the arteriolar diameter when IOP falls and reduce the rise in ΔP this way. The obstruction of venous outflow from the uveal tract from changes in scleral biomechanics, which occurs subsequently can result in a rise in ΔP, thus a rise in IOP.[15] The mean duration of cases with fresh RD features was around 1 month. Hence, there would have been a fluctuation in IOP over the days.[16]

The subretinal space between the photoreceptors and the retinal pigment epithelium (RPE) is the remnant of the embryonic optic vesicle.[17] In the developed eye, the subretinal space is of minimal size; however, no tissue junctions form across it and it can re-open under pathological conditions of retinal detachment. Absorption via the perioptic space may be a contributing factor.

Multiple factors may contribute to lower IOP in RRD. These include decreased rate of aqueous humour formation, increased drainage through a break, decreased scleral rigidity, corneal biomechanics, scleral biomechanics, retinal tension, increased drainage via peri optic space, and myopic refractive status. IO*P* values measured even by applanation tonometry are dependent on eyewall stress values and viscoelasticity of posterior sclera.

CONCLUSIONS

The relationship of IOP dynamics with the severity, chronicity, and type of breaks performed in its study shows that hypotony is not always associated with rhegmatogenous retinal detachment. It shows that the IOP can vary from normal, low hypotony to hypotony levels and does not correspond always with the severity/duration/type of breaks. This might be due to the fact that IOP variations in retinal detachment at individual levels are also dependent on other factors such as decreased rate of aqueous humour formation, increased drainage through a break, decreased scleral rigidity, corneal biomechanics, scleral biomechanics, retinal tension, increased drainage via the peri-optic space, and myopic refractive status. Thus, it suggests the possibility of an alternate hypothesis for lower IOP in RRDs. Hypotony in RRD could be a prognostic factor but while measuring it we have to take all the above factors into consideration, and the investigation should include an ocular response analyzer for IOP monitoring.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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Original Article

Assay of TORCH profile in patients with congenital cataract aged 0-4 years at a tertiary eye hospital

ABSTRACT

Purpose: To evaluate the TORCH profile (*Toxoplasma gondii*, rubella, *cytomegalovirus*, and herpes simplex virus1 and 2) of all patients with congenital cataract excluding traumatic cataracts aged 0-4 yrs over a period 12 months at a tertiary eye hospital. **Methods:** In this cross-sectional study performed for 12 mo, all patients with congenital cataract aged 0-4 yrs excluding traumatic cataracts were assayed quantitatively and qualitatively at a tertiary eye hospital. **Results:** Of 49 patients with congenital cataract included in the study, 43 (87.75%) were seropositive for TORCH profile. Approximately 7 of 49 presented unilaterally, whereas 42 were bilaterally cataractous. Approximately 37 of 43 seropositive patients (86.04%) were IgG +ve, 6 of 43 (13.95%) were positive for both IgM and IgG, and none were IgM-positive. Approximately 11 of 43 (25.58%) were CMV +ve alone, 5 of 43 (11.62%) were rubella +ve, and 1 of 43 (2.32%) was HSV1 +ve, whereas 25 of 43 (58.13%) were +ve in combinations. **Conclusion:** The positivity rate of TORCH profile is high in patients with congenital cataract. Hence, previous testing and its evaluation need to be performed, so that the clinician can be vigilant and identify the associated syndromic presentation at the earliest.

Keywords: CMV, congenital cataract, HSV, rubella, seropositive, TORCH profile, toxoplasmosis

INTRODUCTION

Congenital cataract refers to the opacification of crystalline lens at birth.[1] When left untreated, it can result in visual deprivation. Worldwide, 20000-40000 children are born with congenital cataract, as stated by Gilbert *et al.*[2]

Hence, early identification and management of cataract is the need of the hour.

Etiology of congenital cataract[3-11] includes idiopathic, genetic, metabolic, trauma, systemic causes, drug reactions, radiation, and intrauterine infections including TORCH agents.

TORCH is an acronym[12-14] for toxoplasmosis, rubella, *cytomegalovirus* (CMV), herpes simplex virus 1 and 2 and others including HIV 1 and 2, *Treponema pallidum*, varicella zoster, and parvovirus b19. These are recognized as potential causative agents of congenital cataract, and hence, investigating all cases of congenital cataract for TORCH agents needs to be performed.

These can be acquired in utero, during delivery, or community-acquired. They can present clinically with varied systemic and ocular manifestations in neonatal period or later in life.

Evaluation of the TORCH serology helps in determining the stage of infection. IgM positivity denotes recent infection. IgG positivity denotes past infection/reinfection/reactivation, and IgM + IgG positivity denotes subacute/progression of infection.

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METHODS

The present study is a cross-sectional study, performed for a duration of 12 months, from January 2021 to December 2021 at a tertiary eye hospital in southern India. All clinically diagnosed patients with congenital cataract aged 0-4 yrs attending OPD *(with informed written consent of the parents/guardians)* have been included in the present study, whereas traumatic cataracts have been excluded from the study.

The study has been approved by the institutional ethics committee and followed the tenets of declaration of Helsinki.

Clinical history including presenting complaint, history, family history, antenatal, natal, and postnatal history, immunization status, and developmental milestones was noted. Ophthalmic evaluation, B-scan, and TORCH serology using ELISA, i.e., enzyme-linked immunosorbent assay for HSV 1 and 2 IgG and IgM and ELFA, i.e., enzyme-linked fluorescent antibody assay for toxoplasmosis, rubella virus, and CMV IgG and IgM were performed for all the patients. (Other agents such as HIV 1 and 2, *Treponema pallidum*, and varicella zoster were not tested.)

Statistical analysis was performed using *SPSS v 21 software*.

T-test for categorical variables and *Chi-square test* for continuous variables were applied.

$P < 0.05$ was considered to be *statistically significant*.

RESULTS AND ANALYSIS

IgG-positive or IgM-positive or IgG + IgM-positive cases were considered to be seropositive. Case definition of congenital cataract is taken as lens opacification at birth.

A total of 49 clinically diagnosed congenital cataract cases in the age group 0-4 years attending the pediatric outpatient department at tertiary eye care center were examined and evaluated for TORCH serology. Of 49 patients with congenital cataract evaluated, 43 were seropositive (87.75%) and 6 were seronegative (12.25%). Of the 43 seropositives, 36 were bilateral (83.73%) and 7 were unilateral (16.27%).

The mean age of presentation among the seropositives is 15.57 months. A male preponderance has been noted, 29 males: 14 females.

Approximately 2 of 49 patients with congenital cataract had a positive family history ($P > 0.5$) and 3 of 49 mothers had fever during gestation ($P = 0.504$), both were statistically not significant. All the cases had an institutional delivery conducted by doctor, and NICU admission (17 of 49) was performed for the required cases. All the patients were immunized till date. Approximately 10 of 49 patients had delayed milestones (20.40%).

The TORCH positives were further evaluated for the IgG and IgM positivity based on the serological tests ELFA and ELISA.

Of 43 seropositive cases, 37 (86.04%) were positive for IgG alone and 6 (13.95%) positive for IgG + IgM [Graph 1].

Seropositivity of individual agents.

CMV alone constituted 11 of 43 TORCH-positive cases (25.6%), whereas a combination of rubella and CMV constituted 12 of 43 cases, and the other coinfections constituted 14 of 43 cases (32.5%) [Table 1 and Graph 2].

Distribution of seropositivity among the individual agents.

Anti-CMV IgG positivity rate is higher among the IgG positives and anti-HSV 1 and 2 positivity rate higher among the IgM positives [Table 2 and Graph 3].

Distribution among the unilateral and bilateral cases [Table 3 and Graph 4].

DISCUSSION

All the patients with congenital cataract attending the pediatric OPD at the tertiary center during the 12-month period were evaluated. Patients were operated for the cataract, and an intraocular lens was placed in feasible cases. Pediatric referral was performed for complete systemic evaluation.

The age group chosen for the study is 0-4 years. Observation, identification, and presentation of the child to the clinician can be delayed. The knowledge, attitude, socioeconomic status, accessibility, and awareness of the parent/guardian play a very important role.

In the present study, an informant in most of the cases is the mother of the child, and in few other cases, the father and the grandmother were the informants. Approximately

37 of 49 parents/guardians complained of whitish reflex in the eye/unable to fixate/not looking at mother, since birth. Approximately 9 of 49 cases presented within 1 year of age, and 3 cases presented within 1-4 years of age. The mean age of presentation among the TORCH-positive cases was 15.57 months.

A male preponderance has been noted in the study.

Approximately 3 of 49 cases had fever during gestation of which 2 were positive for TORCH serology.

Of 49 congenital cataract cases, 43 were seropositive, of which 7 were unilateral and 36 were bilateral.

Of the 43 seropositives, 4 had preterm delivery, and 9 had delayed milestones.

Ocular associations of nystagmus were found in 5 TORCH-positive bilateral congenital cataract cases, of which one patient has [BE] nystagmus with esotropia.

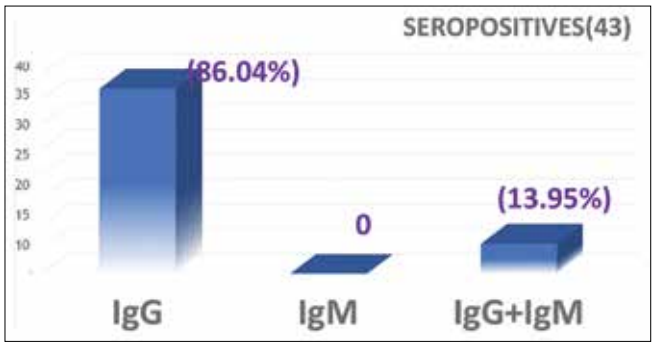
Other ocular associations in the TORCH-positive cases include microphthalmos and exotropia.

The results of the study correlated with that of Mahalakshmi *et al.*,[9] i.e., association of congenital cataract with tox, rubella, CMV, and HSV 1 and 2.

TOX

IgG positivity for toxoplasmosis was 7% in the current study, whereas IgM positivity was 2.3% in previous investigations. According to a study by Gupta *et al.*,[10] 4.3% of them had seroclinical positivity.

The major cause of pediatric cataract, according to Singh MP *et al.*[3] from North India, was TOX, with an IgM-positivity of 8.3%. However, Mahalakshmi B *et al.*[9] reported tox to be 1.7% in their study.



Graph 1: Distribution of seropositivity

Rubella

IgG positivity for rubella in the current study was 58.10%, IgM positivity was 4.70%, and IgG + IgM positivity was 2.3%.

In a study by Gupta *et al.*,[10] 13% have been observed to have IgM (but not IgG) antibodies against RV and 6.5% to have seroclinical antibodies.

The IgM seropositivity was observed in research from North India (Singh *et al.*[3]—5.8%, Angra and Mohon[11]—11%, and Angra[15]—21.5%) while that of South India Mahalakshmi *et al.*[9]—8.4% and Shyamala *et al.*[16]—14%.

CMV

IgG positivity was 79% in the current study, but in previous investigations in southern India, CMV-IgM and IgG positivity rates were 7.8% vs. 66% and 0% vs. 54%, respectively.

HSV

Anti-HSV1 IgG made up 23.30% of the IgG in the current study, whereas anti-HSV2 IgG made up 7%.

Table 1: Seropositivity of individual agents

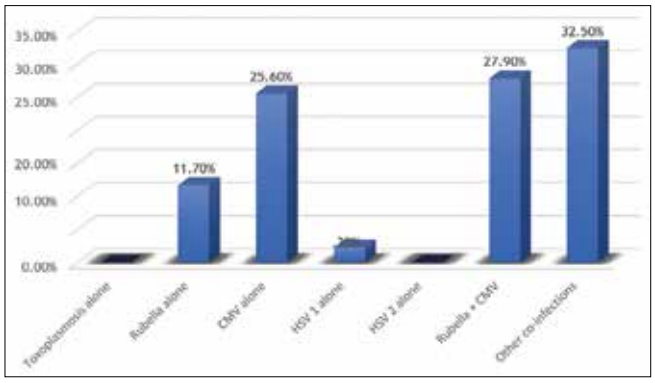
	Frequency	Percent
Toxoplasmosis alone	0	0.0
Rubella alone	5	11.7
CMV alone	11	25.6
HSV 1 alone	1	2.3
HSV 2 alone	0	0.0
Rubella + CMV	12	27.9
Other coinfections	14	32.5
Total	43	100.0

Table 2: Distribution of seropositivity among the individual agents

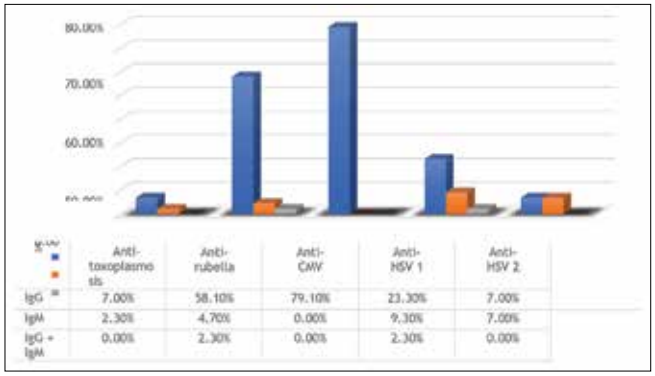
	IgG	IgM	IgG + IgM
Antitoxoplasmosis	3 (7.0%)	1 (2.3%)	0 (0.0%)
Antirubella	25 (58.1%)	2 (4.7%)	1 (2.3%)
Anti-CMV	34 (79.1%)	0 (0.0%)	0 (0.0%)
Anti-HSV 1	10 (23.3%)	4 (9.3%)	1 (2.3%)
Anti-HSV 2	3 (7.0%)	3 (7.0%)	0 (0.0%)

Table 3: Distribution among the unilateral and bilateral cases

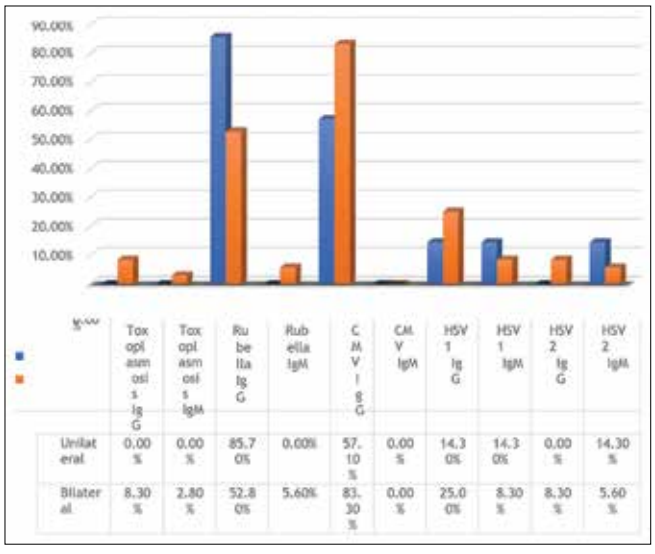
	Unilateral ($n=7$)	Bilateral ($n=36$)
Toxoplasmosis IgG	0 (0.0%)	3 (8.3%)
Toxoplasmosis IgM	0 (0.0%)	1 (2.8%)
Rubella IgG	6 (85.7%)	19 (52.8%)
Rubella IgM	0 (0.0%)	2 (5.6%)
CMV IgG	4 (57.1%)	30 (83.3%)
CMV IgM	0 (0.0%)	0 (0.0%)
HSV 1 IgG	1 (14.3%)	9 (25.0%)
HSV 1 IgM	1 (14.3%)	3 (8.3%)
HSV 2 IgG	0 (0.0%)	3 (8.3%)
HSV 2 IgM	1 (14.3%)	2 (5.6%)



Graph 2: Seropositivity of individual agents



Graph 3: Distribution of seropositivity among the individual agents



Graph 4: Distribution among unilateral and bilateral cases

Anti-HSV 1 IgM levels are 9.3%, and anti-HSV 2 IgM levels are 7%, respectively. Anti-HSV 1 IgM + IgG levels are 2.3% and 0%.

In other investigations, IgM and IgG positives as reported by Mahalakshmi *et al.*[9] were 5.1% and 10% and Shyamala *et al.*[16] 0.0% and 3.0%.

Co-infections

In the current investigation, more than two agents were coinfected with 26 of 43 TORCH-positives.

Of the 26 instances, 12 involved the combination of rubella and CMV, whereas 14 involved other combinations.

Table 4 depicts the observations of various studies. Other studies,[5,8,17-25] also gave an insight into the epidemiology of TORCH serology.

A prospective analytical study was performed by Sushoban das Gupta *et al.*,[10] in northern India. The age group studied ranged between 0 and 15 years. A high IgG positivity reported for CMV and IgM positivity was higher for rubella, among the individual positivity. Coinfection was noted in 91.3% of the sample studied. This study emphasized the importance of conducting serial quantitative and qualitative analyses of TORCH serology to eliminate false positives.

A case-control study performed by Bin Lu *et al.*,[6] at China, on 69 cases and 5194 controls, for a study period of 7 years, revealed higher anti-CMV IgG and IgM positivity rates.

It was also observed in their study that the anti-HSV-2 positivity rates significantly differed among the cases and controls, implying that HSV can be one of the causative etiologies of congenital cataract.

LIMITATIONS OF THE STUDY

Serial qualitative and quantitative analysis of the positive TORCH cases could have eliminated the possibility of false positives.

The possibility of cross-infection with other agents should be considered while analyzing.

IgG levels are significantly higher. Further testing of maternal serology needs to be performed to collaborate its transmission to newborns.

A larger sample size and a longer duration of study could have helped in extrapolating the results to a larger population.

CONCLUSION

There is an increased need of vaccines to TORCH to prevent infections to the mother and newborns. Evaluation of TORCH profile enables the clinician to be vigilant for the other associated syndromic presentations, both systemic and ocular, and hence, timely referral. KAP—knowledge, attitude, and practices among the parents, guardians, and health care workers need to be enhanced. Prevention is better than cure.

Table 4: Observations of various studies

	Present study	Mahalakshmi *et al.*[9]	Sushoban Gupta *et al.*[10]	Bin Lu *et al.*[6]
Study design	Cross-sectional	Retrospective	Prospective analytical study	Case-control
Sample size	43/49 +ve torch	120/593 TORCH +ve	46 TORCH +ve	69 cataracts vs 5194 controls
Duration	12 months	8 yrs	12 months	7 yrs
Age group	0-4 years	10 d-12 mo	0-15 yrs	9 days-7 yrs
seropositivity	CMV 25.58%			anti-hsv 2
Coinfection	58.13%	2.50%	91.3% (42/46)	
IgG	anti-CMV IgG 69.4%	anti-CMV IgG 66.1%	anti-CMV IgG 67.4%	anti-CMV IgG 23.20%
IgM	anti-HSV IgM	antirubella IgM	antirubella IgM 50%	anti-CMVv IgM 15.04%

In TORCH-positive cases of congenital cataract, prevention forms the mainstay. Lifestyle changes, hand hygiene, food hygiene, and safe sexual practices play an important role in preventing these infections in pregnant women and, hence, the newborns. Vaccines to rubella need to be taken before conception among women.

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Conflicts of interest

There are no conflicts of interest.

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Original Article

Comparative evaluation of choroidal thickness in hypertensive individuals and healthy controls using optical coherence tomography

ABSTRACT

Purpose: This study aimed to assess the impact of hypertension on choroidal thickness by comparing hypertensive patients with age-matched healthy individuals. **Methods:** A cross-sectional comparative study included 62 individuals (31 each in the hypertensive and control groups, aged 30–70). Choroidal thickness was measured at subfoveal, 500 µm nasal, and 500 µm temporal to the fovea using optical coherence tomography (OCT). Statistical analysis employed independent-samples t-tests, and correlations were evaluated with Pearson's coefficients. **Results:** Subfoveal choroidal thickness was similar in hypertensives and normals (294.02 ± 61.27 and 295.86 ± 50.86, respectively). Nasal and temporal thickness was significantly reduced in hypertensive patients (256.38 ± 59.32 and 269.53 ± 69.50) compared with controls (285.34 ± 51.37 and 280.34 ± 55.85). Subfoveal thickness had a weak positive correlation with mean blood pressure (MBP) (Pearson's coefficient 0.150), while nasal thickness had a significant but weak negative correlation with MBP (Pearson's coefficient -0.73). No significant correlation existed between hypertension duration and choroidal thickness. **Conclusion:** The study revealed reduced nasal and temporal choroidal thickness in hypertensive individuals compared with age-matched healthy subjects. Subfoveal thickness in hypertensives showed a significant but weak positive correlation with MBP, and nasal thickness had a negative correlation with MBP.

Keywords: Choroidal thickness, OCT, systemic hypertension

INTRODUCTION

Hypertension poses a substantial health risk, defined by the World Health Organization as a systolic blood pressure (SBP) exceeding 140 mmHg and/or a diastolic blood pressure (DBP) exceeding 90 mmHg. Globally, about 1.28 billion adults aged 30–79 are affected, with nearly 46% unaware of their condition.[1] Initial presentations often reveal target organ damage across various organs.[2-4]

Hypertension contributes to acute and chronic abnormalities such as choroidopathy, retinopathy, and disorders affecting retinal veins, arteries, and optic nerves.[5] Hypertensive retinopathy presents with signs such as generalized arteriolar narrowing, copper or silver wiring, arteriovenous nicking, cotton wool spots, and hard exudates.[1]

The highly vascular choroid, crucial for retinal blood supply, undergoes changes in hypertensive individuals.[6,7] Spectral domain optical coherence tomography (SD-OCT) with enhanced depth imaging (EDI) allows clinicians to meticulously examine retinal layers and measure choroidal thickness. Early detection of hypertensive retinopathy and alterations in subfoveal choroidal thickness enhance clinicians' ability to effectively

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manage hypertension and monitor its impact on end organs.

METHODS

This prospective descriptive cross-sectional study took place in the Department of Ophthalmology at a tertiary eye care hospital in North Kerala. Approval was obtained from the institutional research board and ethics committee, adhering to the Declaration of Helsinki principles, and participants faced no financial burden.

The study group involved 31 adults diagnosed with essential hypertension, determined by a SBP ≥140 mm Hg and DBP ≥90 mm Hg.[8] The control group included 31 age-matched normotensive individuals from the ophthalmology outpatient department, categorized as group A for normotensives and group B for hypertensives.

Demographics, including age, gender, and systemic disease status, were recorded for both groups. The duration of hypertension was noted in hypertensive individuals. Patient inclusion criteria encompassed hypertensive individuals aged 18 and above who provided consent and had clear ocular media. Exclusions involved diabetic patients, systemic diseases affecting retinal and choroidal circulation, eyes with hazy media interfering with reliable OCT images, those with a history of ocular surgeries, and any local diseases, including glaucoma.

After obtaining informed consent, the study participants underwent the following procedures:

1. History taking
2. Full ophthalmic examination
   - Best-corrected visual acuity (BCVA).
   - Anterior segment examination using slit-lamp biomicroscopy with no abnormal data.
   - Posterior segment examination using slit-lamp biomicroscopy with a 90 diopter lens and indirect ophthalmoscopy.
3. Investigations:
   - OCT was employed to measure choroidal thickness at three locations in each eye (subfoveal, 500 µm nasal to fovea, and 500 µm temporal to fovea) using the in-built caliper function. Both eyes of each individual were sequentially scanned. Choroidal thickness was measured as the perpendicular distance between the hyperreflective outer border of the retinal pigment epithelial-Bruch's membrane layer (RPE-BM) and the sclero-choroidal interface by a single observer for consistency [Figure 1].

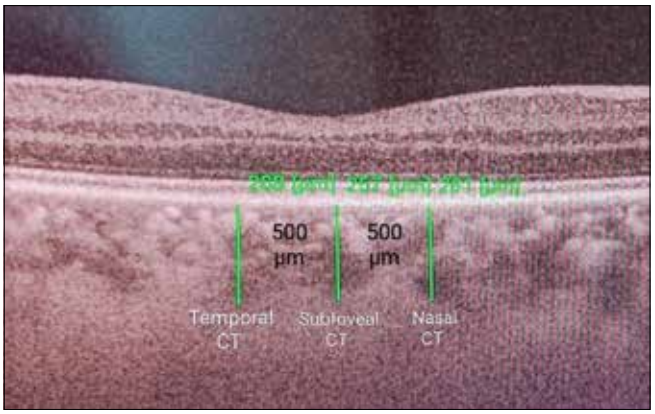


Figure 1: Measurement of choroidal thickness at subfoveal region, 500 µm nasal to fovea, and 500 µm temporal to fovea in a normal individual

Data entry was performed in a Microsoft Excel Spreadsheet, and analysis was conducted using the Statistical Package for the Social Sciences (SPSS). The analysis included the application of an independent-samples *t*-test, and correlations between variables were examined using Pearson's correlation coefficient. A significance level of $P < 0.05$ was considered statistically significant.

RESULTS

Among the 62 patients studied, 20 were males (32.3%) and 42 were females (67.7%). The mean duration of hypertension was 3.67 years. Notably, 22 subjects (70.9%) were hypertensive for ≤5 years, while nine subjects (29.03%) had hypertension for >5 years in the hypertensive group [Figure 2].

In terms of choroidal thickness, the subfoveal region showed similarity between hypertensives and normal subjects (294.02 ± 61.27 and 295.86 ± 50.86, respectively). However, nasal and temporal choroidal thickness was significantly reduced in the hypertensive group (256.38 ± 59.32 and 269.53 ± 69.50, respectively) compared with controls (285.34 ± 51.37 and 280.34 ± 55.85, respectively) [Table 1].

The correlation analysis revealed that subfoveal choroidal thickness in hypertensive patients had a weak positive correlation with mean blood pressure (MBP) (Pearson's coefficient 0.150), while nasal choroidal thickness had a significant but weak negative correlation with MBP (Pearson's coefficient -0.73). However, there was no significant correlation between the duration of hypertension and choroidal thickness [Table 2].

DISCUSSION

The objective of our study was to compare choroidal thickness at various locations (subfoveal, nasal, and

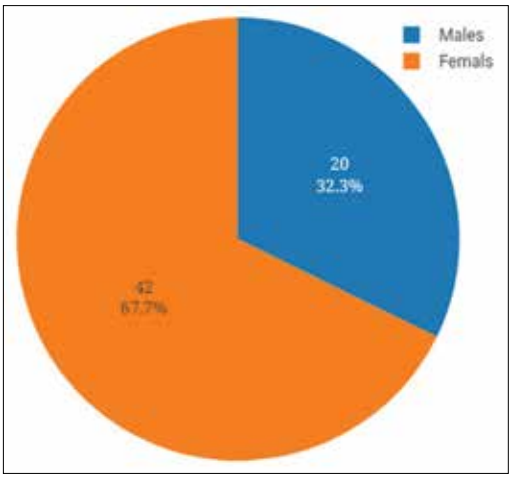


Figure 2: Demographic data showing total males and females studied

temporal) in hypertensive individuals with healthy subjects. We observed significantly decreased nasal and temporal choroidal thickness in the hypertensive group compared with the control group, consistent with findings by Akay *et al*.[9] in a younger age group (20–29 years).

In our study, we identified a weak positive correlation between MBP and subfoveal choroidal thickness, as well as a negative correlation of MBP with nasal choroidal thickness. Similar to our study, Gök *et al*.,[10] in their prospective cross-sectional study with 116 hypertensive patients and 116 control subjects, did not reveal any significant difference in subfoveal choroidal thickness between the two groups. They suggested that the choroidal vasculature remains relatively protected in chronic hypertension due to its intrinsic properties critical for retinal nourishment and optimal perfusion pressure maintenance.

Some studies found the subfoveal choroid to be thicker than the nasal and temporal regions, consistent with findings by Waghamare *et al*.[11] In a cross-sectional study, Waghamare *et al*. demonstrated that the subfoveal choroid was thicker than the nasal and temporal choroid in both hypertensive and healthy individuals. Balmforth *et al*.,in a comparative study, found no difference in choroidal thickness between hypertensive and normal individuals, but they showed reduced choroidal thickness in chronic kidney disease (CKD) patients compared with the hypertensive and normal control groups.[12]

Marisse Masís *et al*.,[13] in a retrospective observational comparative series with 112 hypertensive patients and 15 healthy individuals, estimated subfoveal, nasal, and temporal choroidal thickness using EDI spectral domain OCT. They found that the choroid was thinnest subfoveally in both hypertensives and non-hypertensives, increasing in thickness in the nasal direction. Overall, they concluded that the choroid was thinner in hypertensive patients compared with non-hypertensives in all three evaluated regions.

Table 1: Comparison of choroidal thickness measurements between normotensive individuals (group A) and hypertensive patients (group B)

Parameters	Group A (normotensive) *n*=31	Group B (hypertensive) *n*=31
Average subfoveal choroidal thickness	295.86±50.86	294.02±61.27
Average nasal choroidal thickness	285.34±51.37	256.38±59.32
Average temporal choroidal thickness	280.34±55.85	269.53±69.50

Table 2: Correlation analysis between choroidal thickness and mean blood pressure

	Mean blood pressure	
	Correlation coefficient	*P*
Average subfoveal choroidal thickness	0.150	0.246
Average nasal choroidal thickness	-0.73	0.571
Average temporal choroidal thickness	0.084	0.516

While our study demonstrated valuable insights, it is crucial to acknowledge its limitations. The relatively small sample size and the absence of long-term follow-up for both hypertensive and control groups limit our ability to explore the longitudinal effects of hypertension on choroidal circulation. The strength of the study lies in the consistency of choroidal thickness measurements, performed manually by a single observer, enhancing the reliability of the results.

CONCLUSION

Our study revealed a significant decrease in nasal and temporal choroidal thickness in hypertensive subjects compared with normal individuals. However, choroidal thickness at the subfoveal region did not show any statistical difference between the two groups. This suggests that the measurement of choroidal thickness using OCT can serve as a noninvasive biomarker for assessing the impact of systemic hypertension on the eye.

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Conflicts of interest
There are no conflicts of interest.

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Original Article

Comparison of central macular thickness using spectral domain optical coherence tomography in sickle cell hemoglobinopathy patients and age-matched controls – A comparative cross-sectional observational study

ABSTRACT

Purpose: To compare central macular thickness (CMT) using spectral domain optical coherence tomography (SD-OCT) in sickle cell hemoglobinopathy (SCH) patients and their age-matched controls. **Methods:** A comparative cross-sectional observational study to compare CMT using SD-OCT was done in SCH patients (Sickle cell anemia (SS), Sickle cell trait (AS), HbSC disease (SC), Sickle-Beta Thalassemia (S-Thal)) and their age-matched controls at a tertiary eye care center in Central India for two years. The study included two groups, the SCH patients in Group I and age-matched controls in Group II, with 58 subjects (116 eyes) in each group. SCH patients were recruited from the Sickle Cell Clinic in the hospital. Age-matched controls were selected from a general eye clinic in the same hospital. All patients underwent detailed ophthalmic examination consisting of visual acuity measurement, slit-lamp biomicroscopy, dilated fundoscopy, and central subfield macular SD-OCT imaging of each eye. Analysis of the findings was done by a single vitreoretinal surgeon. Statistical analysis was done. **Results:** The study included total of 232 eyes with 116 eyes in each group. The age ranged from 10 to 60 years with a mean age of 31.55 years in both groups. There were 31 males and 27 females in each group. The mean CMT in the right eye in cases and controls was 224.87 and 244.36 um, respectively, and in the left eye the CMT in cases and controls was 224.86 and 244.31 um, respectively. The *P* value in both groups (cases and controls) in both eyes was 0.001, i.e., statistically significant. **Conclusions:** The CMT in SCH patients was significantly less than that in age-matched controls.

Keywords: Macular thickness, sickle cell hemoglobinopathy (SCH), spectral domain optical coherence tomography (SD-OCT)

INTRODUCTION

Sickle cell disease (SCD) is the most common and serious form of inherited blood disorders. Sickled erythrocytes are responsible for vaso-occlusive, hemolytic, and inflammatory features of the disease.[1]

Retinal findings in sickle cell hemoglobinopathy (SCH) patients are predominantly in the retinal periphery, but macular changes can also occur including macular infarction, macular thinning, and enlargement of foveal avascular zone.[2,3] Sickle cell maculopathy is one of the emerging complications which is a localized macular thinning secondary to ischemia.[4] It is a subtle disease that is not always clinically apparent on indirect ophthalmoscopy; instead, it is more easily detectable using spectral domain optical coherence tomography (SD-OCT) and OCT angiography.[4]

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Based on findings from previous studies, we hypothesize that eyes with SCH will show evidence of subclinical ischemia manifested as retinal thinning on SD-OCT imaging. To the best of our knowledge, this is the first study to compare central macular thickness (CMT) in patients with SCH and age-matched controls in Central India.

METHODS

A comparative cross-sectional study was conducted to analyze CMT using SD-OCT imaging of SCH patients and their age-matched controls over two years from August 2020 to July 2022 in the Department of Ophthalmology at a tertiary eye care center, Central India. It included 232 eyes of 116 subjects. Out of these 116 subjects, 58 subjects (116 eyes) each were included in Group I, i.e. SCH group and in Group II, i.e., age-matched controls. SCH subjects were enrolled in the study from a Sickle Cell Clinic. All patients with hemoglobin (Hb) eectrophoretic confirmation of SCH including AS, SS, SC, and S-thalassemia pattern were included in the SCH group. In the SCH group, 43 subjects had SCD (SS pattern of Hb), 10 patients had sickle cell trait (AS pattern of Hb), 1 patient had SC pattern of Hb, and four patients had S-thalassemia. All patients included in the SCH group were free of any sickle cell retinopathy.

Subjects in the age-matched control group were chosen from a general eye clinic in the same hospital. Controls were required to be free of any SCH or any retinal disease. Subjects in both groups having minimal refractive error and minimal cataracts were included in the study. All uncooperative patients, debilitated patients, children less than ten years of age and adults more than 60 years of age, patients with systemic comorbidities, and any other ocular disease were excluded from the study. The ages of the subjects ranged from 10 to 60 years in both groups.

The study was performed by the tenets of the Declaration of Helsinki and was approved by the Institutional Ethics Committee. Patients were explained about the study. Written informed consent was obtained from all subjects.

Detailed history was taken. Thorough ophthalmic examination consisting of measurement of best-corrected visual acuity on Snellen's chart, slit lamp examination, and dilated fundus examination by indirect ophthalmoscopy was done. Intraocular pressure was measured by applanation tonometry. All patients underwent SD-OCT imaging of each eye to measure the CMT by 3D macular scan by a single vitreoretinal surgeon. SD-OCT was performed on OPTOVUE RTVue 100, 3D OCT System 6.2 version. Thickness profiles were generated from measurements obtained at 100 um intervals laterally along OCT scans and then aligned based on the minimum inner retinal thickness at the fovea center. Thickness was averaged among the central subfield macula (1 mm diameter, centered on the fovea), parafoveal (0.5–1.5 mm eccentricity, nasally or temporally), and perifoveal (1.5–3 mm eccentricity, nasally or temporally) regions.[2] Early Treatment Diabetic Retinopathy Study (ETDRS) subfield measurements were compared between sickle cell and control subjects.[3] In our study, we compared only the central 1 mm subfield macula. All examinations for an individual study participant were performed on the same day. Data were collected and entered in an MS Excel sheet. Statistical analysis was done by unpaired *t*-test and Chi-square test using SPSS software. The mean, standard deviation, and standard error of means were calculated. P value < 0.05 was considered significant.

RESULTS

In the study, comparison of CMT in SCH patients and age-matched controls was done. Fifty-eight SCH patients (116 eyes) in group I and 58 age-matched controls (116 eyes) without clinical evidence of maculopathy by ophthalmic examination in group II participated in the study. The ages of subjects ranged from 10 to 60 years with a mean age of 31.55 years in both groups [Table 1]. Maximum number of patients (60) were in the age group of 10 to 29 years. There were 31 males and 27 females in each group with a male: female ratio being 1.15:1. Majority of patients (30 patients in the right eye and 28 patients in the left eye) had visual acuity ranging between 6/6 and 6/12 in the SCH group [Table 2]. Most of the patients (32 patients in the right eye and 29 patients in the left eye) had visual acuity ranging between 6/6 and 6/12 in the control group. Only seven patients and eight patients in right and left eyes, respectively, had visual acuity </=6/60 in the SCH group and only eight patients in the right and left eye each had visual acuity </=6/60 in the control group due to nuclear cataract.

CMT in the right eye ranged between 199 and 259 um in the SCH group and between 238 and 266 um in the control group. The CMT in the left eye ranged between 177 and 258 um in the SCH group and between 235 and 265 um in the control group.

The mean CMT in the right eye in the SCH group was 224.87 um with a standard deviation of 14.695 and that in the control group was 244.36 um with a standard deviation of 3.264. The P value for both groups was 0.001, which was statistically highly significant. The mean CMT in left eye in the SCH group was 224.86 um with a standard deviation of

Table 1: Age distribution of patients in both the groups

	Age in years					Total number of subjects	Mean age in years±SD
	10–19	20–29	30–39	40–49	50–60		
SCH Group (Group I)	15	15	12	8	8	58	31.55 years±14.36
Age-matched control Group (Group II)	15	15	12	8	8	58	31.55 years±14.36
Total number of subjects	30	30	24	16	16	116	

Table 2: Visual acuity distribution in cases and controls

Visual Acuity	Right eye		Left eye	
	Number of SCH group	Number of controls	Number of SCH group	Number of controls
6/6–6/12	30	32	28	29
6/18–6/36	21	18	22	21
</=6/60	7	8	8	8

14.807 and that in the control group was 244.31 um with a standard deviation of 3.294. The *P* value for both groups was 0.001 which was again statistically highly significant [Table 3].

CMT of the right eye of cases and the right eye of controls was compared in males, and females and CMT of the left eye of cases and left eye of controls was compared in males and females.

Comparison of CMT of cases and controls in females in the right and left eyes was done separately [Table 4]. In the right eye, the mean CMT and standard deviation were 223.25 um and 14.53, respectively, in cases and the mean CMT and standard deviation were 255.37 um and 7.17, respectively, in controls. The *P* value for both groups was 0.000, which was statistically highly significant. In the left eye, the mean CMT and standard deviation were 223.40 um and 15.61, respectively, in cases and the mean CMT and standard deviation were 256.14 um and 6.87, respectively, in controls. The *P* value for both groups was 0.000, which was statistically highly significant.

Comparison of CMT of cases and controls in males in the right and left eyes was done separately. In the right eye, the mean CMT and standard deviation were 226.45 um and 14.90, respectively, in cases and the mean CMT and standard deviation were 254.74 um and 6.87, respectively, in controls. The *P* value for both groups was 0.000, which was statistically highly significant. In the left eye, mean CMT and standard deviation were 226.12 um and 14.20 respectively in cases, and mean CMT and standard deviation was 255.48um and 5.88, respectively, in controls. The *P* value was 0.000, which was statistically highly significant [Table 5].

DISCUSSION

Sickle cells interact with endothelial cells and other blood cells, occluding flow in small and sometimes large vessels causing many complications thought to be a result of vaso-occlusion.[1] SCH has various systemic and ocular manifestations. Systemic manifestations include painful vaso-occlusive crisis, stroke, sickle chest syndrome, sequestration crisis, and aplastic crisis.[5] Ocular anterior segment manifestations include comma-shaped dilations of small conjunctival vessels in inferior bulbar conjunctiva, segmental iris atrophy, hyphemia, elevations of intraocular pressure, and neovascularization on iris. Posterior segment features include optic disc 'sign of sickling,' macular depression sign, enlarged foveal avascular zone, parafoveal thinning, angioid streaks, silver wiring of retinal arterioles, peripheral areas of whitening or darkening, salmon patch hemorrhages, iridescent spots, and sunburst sign.[6]

Sickle cell retinopathy is known to cause retinal pathologies predominantly in retinal periphery, but can also cause macular infarctions. Central macular thinning and foveal splaying (due to thinning specifically in the parafoveal regions) may represent either the residua of or the precursor to macular infarctions. These changes may cause changes in the thickness of retinal layers in the macula and reduce vision.[2]

The retinal vessels supply blood to the ganglion cell layer and inner nuclear layers of the retina, whereas the choriocapillaris nourishes the photoreceptors and the retinal pigment epithelium. As vessels in the choriocapillaris are of larger caliber, it is rare for them to get occluded and cause outer retinal ischemia. However, the inner retinal layers are prone to ischemia, as the retinal vessels are end arterioles and capillaries. Histopathologic studies of sickle cell retinopathy and other vaso-occlusive diseases have previously shown selective atrophy of the inner retinal layers of the macula in several eyes after retinal infarction.[7]

In this comparative cross-sectional observational study, we compared central subfoveal macular thickness using SD-OCT of SCH patients in one group with age-matched controls in another group. The study included a total of 116 subjects (232 eyes). Each group consisted of 58 subjects (116 eyes). Group I included SCH patients from wards or OPDs of a tertiary eye care center in Central India. Group II included age-matched controls from general eye OPD. Subjects in Group II were free

Table 3: Comparison of central macular thickness (CMT) on SD-OCT in the SCH group and controls

	Group	Number of eyes	Range of CMT	Mean	Std. Deviation	*P*
Right eye CMT in microns	SCH Group	58	199–259	224.87	14.695	0.001
	Control	58	238–266	244.36	3.264	
Left eye CMT in microns	SCH Group	58	177–258	224.86	14.807	0.001
	Control	58	235–265	244.31	3.294	

Mean CMT in the SCH group=224.87 um. Mean CMT in the control group=244.34 um

Table 4: Comparison of CMT of males in cases and controls

	Cases		Controls		*P*
	Mean	Standard deviation	Mean	Standard deviation	
Right eye	226.45	14.90	254.74	6.87	0.000
Left eye	226.12	14.20	255.48	5.88	0.000

Table 5: Comparison of CMT of females in cases and controls

	Cases		Controls		*P*
	Mean	Standard deviation	Mean	Standard deviation	
Right eye	223.25	14.53	255.37	7.17	0.000
Left eye	223.40	15.61	256.14	6.87	0.000

of SCD/trait. The ages of subjects ranged from 10 to 60 years with a mean age of 31.55 years in both groups. One study has shown a correlation of the amount of retinal thinning with age and sex.[3] However, such a correlation could not be made in our study due to the small sample size.

In our study, the majority of patients had no symptoms of ocular disease except for patients with mild refractive errors and minimal cataracts. Most of the patients had visual acuities ranging between 6/6 and 6/36. Our study showed that despite lack of symptoms, there was thinning of macula in the central subfield in SCH eyes as compared to age-matched controls. The mean CMT in the SCH group was 224.87 um and the mean CMT in the control group was 244.34 um (*P* value of 0.001). Previous studies demonstrated central subfield thinning of the macula on OCT in asymptomatic sickle cell patients.[2,3]

In our study, fundus examination did not reveal any significant abnormality in any group. According to previous studies, the extent of macular changes has not been shown to correlate with visual acuity, and also macular changes undetectable on clinical examination could be observed by SD-OCT.[3,8]

According to one study, retinal ischemia is sufficient to cause tissue loss even before the patient is symptomatic.[3,9-13] Sickling episodes that occur throughout the capillary bed in the body also take place in the retinal and choroidal vessels. Since there are no pain fibers in the retina, these ischemic episodes result in loss of retinal tissue without pain. The higher metabolic demands of the posterior pole would increase its vulnerability to ischemia theoretically. The finding of thinning of subfoveal CMT on SD-OCT in SCH eyes correlates with foveal depression signs.[10-12] Indeed the transition from infarction of the posterior pole to thinning of inner macular layers, five weeks later has been captured on SD-OCT.[7] Histopathology of sickle cell eyes supports this selective infarction of inner retinal layers.[14] Other studies have reported SD-OCT thinning of inner retinal layers including the nerve fiber layer.[2,7,13,15]

The finding of varying degrees of subfoveal CMT in SCH patients supports the assertion that SD-OCT imaging adds information to the clinical staging of sickle cell retinopathy. Whether this will prove to have prognostic information in terms of future visual loss or retinal complications remains to be seen. In one study, temporal macular atrophy as noted on SD-OCT had a sensitivity of 27%, a specificity of 67%, a positive predictive value of 83%, and a negative predictive value of 13% for identifying neovascularization in sickle cell eyes.[16] Another study has also found significant macular temporal thinning in sickle cell retinopathy eyes.[13]

Previous studies described macular retinal thinning using SD-OCT, in the deeper retinal layers in the central macula and parafoveal retina.[2] Other authors demonstrated paracentral acute middle maculopathy in SCD, which is diagnosed by hyperreflectivity of the parafoveal inner nuclear layer on SD-OCT.[17]

We propose that the measurement of central subfield macular thickness with SD-OCT be used as a screening tool and a supportive investigation for the detection of sickle cell maculopathy in patients having SCH.

CONCLUSION

In our study, we concluded that subfoveal CMT using SD-OCT in SCH patients was significantly less as compared to their age-matched controls. SD-OCT imaging provides information regarding subclinical macular changes and can serve as a supportive investigation to detect subclinical maculopathy. Further studies are needed to evaluate macula in SCH patients in ETDRS subfields over time.

Limitations

Our study had limitations such as limited sample size. We did not compare the subfoveal CMT in different SCH subtypes. Also, we did not compare the ETDRS subfields in both groups. We did not study the subfoveal CMT in patients having sickle cell retinopathy. A prospective follow-up study is needed to analyze the subfoveal thinning of the central macula in SCH patients and its association with visual acuity and progression of sickle cell retinopathy.

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Conflicts of interest

There are no conflicts of interest.

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Original Article

Knowledge, awareness, and clinical practice of ophthalmologists regarding HbA1c estimation in diabetic patients in Andhra Pradesh

ABSTRACT

Background: Ophthalmologists routinely work with diabetes patients and assist in providing comprehensive care in general and ocular disorders like diabetic retinopathy, and therefore, they need to be knowledgeable about the concepts of HbA1c and its significance. **Aim:** To evaluate the awareness and prevailing clinical practices of ophthalmologists about HbA1c (glycosylated hemoglobin) estimation of their diabetic patients. **Setting:** Department of Ophthalmology, Alluri Sitaramaraju Academy of Medical Sciences, Malkapuram, Eluru, Andhra Pradesh, India-534005. **Methods and Materials:** It was a questionnaire-based cross-sectional study, study tool was a validated questionnaire containing ten questions about the normal, and abnormal value of HbA1c, clinical significance, an association of HbA1c with diabetic retinopathy and other co-morbid conditions to evaluate knowledge, awareness, and clinical practice of ophthalmologist in state of Andhra Pradesh of south India. **Statistical Analysis:** All data were analyzed by using a Microsoft Excel sheet. **Results:** 65% of all ophthalmologists correctly recommended to all diabetic patients, and 79.7% chose that no preparation is needed before testing Hba1c. 90.6% chose 8–12 weeks as the average duration of Hba1c, 54.6% chose 5.6–6.5%, and 43.80% chose 4–5.6% as the normal range of Hba1c. 46.9% chose <6.5% as good glycemic control, 100% agreed higher the Hba1c value higher the complications, 56.3% advised Hba1c for every 3 months, and 42.2% (6 months). 46.9% chose option b (25%), 79.7% chose all co-existent diseases (anemia, dyslipidemia, and nephropathy), and 71.9% chose diabetes control and complications trial. **Conclusion:** Ophthalmologists in Andhra Pradesh need to be more enlightened about HbA1c and its significance to prevent diabetic patients from permanently losing their vision.

Keywords: Diabetic retinopathy, Hba1c, ophthalmologists

INTRODUCTION

Diabetes, together with cardiovascular disease (CVD), respiratory disease, and cancer, is one of the biggest global health emergencies of the twenty-first century.[1,2]

The International Diabetes Federation (IDF) estimates that 8.8% of adults worldwide have diabetes, with men being slightly more likely to have the disease (9.6%) than women (9.0%). According to the latest data, 463 million and 374 million people worldwide, respectively, have diabetes and impaired glucose tolerance (IGT), a pre-diabetic condition. By 2045, it is projected that there will be 548 million IGT cases and 700 million cases of diabetes, a 51% rise from 2019.[1]

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According to IDF, 77.0 million people in India have diabetes, and that number is projected to rise to 134.2 million by 2045. In India, the prevalence of diabetes increased from 7.1% in 2009 to 8.9% in 2019, Glycated hemoglobin (HbA1c) has been suggested by the American Diabetes Association as a possible alternative to fasting blood glucose for the diagnosis of diabetes.[1] HbA1c is an important indicator of long-term glycemic control with the ability to reflect the cumulative glycemic history of

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the preceding two to three months and its correlates well with the risk of long-term complications. Ophthalmologists routinely work with diabetes patients and assist in providing comprehensive care in general and specialized care for ocular disorders in particular, including diabetic retinopathy, and therefore, they need to be knowledgeable about the concepts of HbA1c and its significance.

The two primary risk factors for developing diabetic retinopathy are generally considered to be the duration of diabetes and the severity of hyperglycemia. Once retinopathy has developed, glycemic control appears to be a more reliable predictor of how the disease will progress from the early to severe stages.

This study evaluates the awareness and prevailing clinical practices of ophthalmologists about the Hba1c estimation of their diabetic patients and its important relationship with diabetic retinopathy.

SUBJECTS AND METHODS

It was a cross-sectional questionnaire-based study carried out in a Tertiary Care Teaching Hospital and Medical College in Eluru. Three hundred and twenty-six ophthalmologists participated in this study. Ethical clearance for this study (ASRAMS BHR-EC/Approval No. 183/2022) was provided by ASRAMS BHR ethics committee, C/O ASR Academy of Medical Sciences, Eluru, Andhra Pradesh (Chairperson-Dr. M. V. Ramesh Chandrasekhar) on 07/10/2022. A validated multiple-choice questionnaire containing ten questions was taken from the previous study.[3]

The questionnaire was about normal and abnormal levels of HbA1c, clinical significance, and association of HbA1c with diabetic retinopathy and other co-morbid conditions. Each question had 2–4 choices. The questionnaire was sent to all ophthalmologists in Andhra Pradesh through Gmail and WhatsApp in Google Forms.

All the responses were tabulated and analyzed in a Microsoft Excel sheet. Supplementary – [HBA1C QUESTIONNAIRE].

RESULTS

Out of a total of 326 ophthalmologists 320 filled out the questionnaire, almost 65% of Ophthalmologists correctly recommended HbA1c for all diabetics (Option b) while nearly one-third (27%) chose only patients with diabetic retinopathy. Only 6% asked for HbA1c in all patients with Proliferative Diabetic Retinopathy (Option d) and 2% requested it for all patients (Option a).

Nearly 79% said that no special preparation is needed before HbA1c testing (Option d). 10.9% chose 6 hours of fasting (Option a) and 9.4% chose 12 hours of fasting (Option b) even though HbA1c can be done at any time of the day and has absolutely no relation to food.

90% of ophthalmologists correctly said that HbA1c provides an average of plasma glucose control over 8–12 weeks (Option b). Only 1.6% chose 4–8 weeks (Option a) and just over 7.8% chose 16–20 weeks (Option c).

Only 43.8% correctly identified the normal HbA1c range as 4–5.6% (Option a) while more than half (55%) thought it was 5.6–6.5% (Option b). Nearly 1.6% chose 6.5–7.5% (Option c).

Nearly 43.8% chose <6.5% as the HbA1c target for good glycemic control for patients with diabetic retinopathy (Option b), whereas one-fourth (26.6%) chose <6.5% (Option c). nearly one-fourth (21.9%) chose <6% (Option a) and 4.7% chose <7.5% (Option d).

An HbA1c of 5–7% indicates good control, 7–9% indicates poorer control, and levels >9% indicate very poor control of diabetes mellitus. The details regarding the responses of the ophthalmologists for Hba1c questionnaire are listed in two Figures [Figure-1 (Q1-Q5), Figure 2 (Q6-Q10)].

All ophthalmologists (100%) agreed that the higher the HbA1c, the higher the risk of developing diabetic complications (Option a).

Nearly 53% chose 3 months (Option b) as the recommended frequency for testing HbA1c for diabetics, while more than one-third (43.1%) chose 6 months (Option c), and only 1.6% chose 2 months (Option a).

Almost 47% of ophthalmologists chose correctly that improving HbA1c by 1% cuts the risk of microvascular complications by 25% (Option b), 39.21% chose 10% (Option a), whereas 14.1% chose 50% (Option c).

79.7% of ophthalmologists correctly chose all the co-existent diseases that can affect HbA1c.e. Anemia, dyslipidemia, nephropathy (Option d). 6.3% chose anemia alone (Option a) while 6.3% chose dyslipidemia (Option b) and 7.8% chose nephropathy (Option c).

Almost 72% correctly chose the Diabetes Control and Complications Trial (Option c) as the trial which recommended regular monitoring of HbA1c levels in diabetics. 20.3% chose the Diabetic Retinopathy

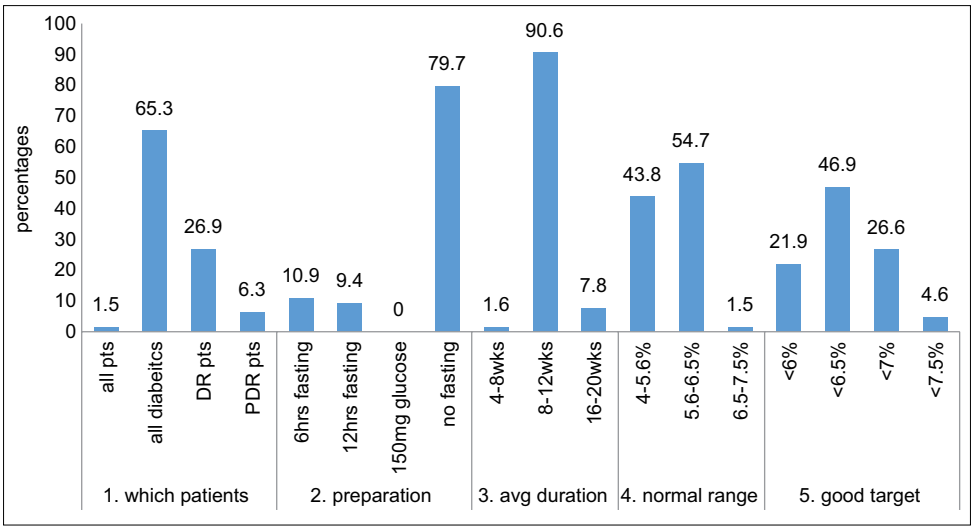


Figure 1: HbA1c questionnaire results for questions 1–5

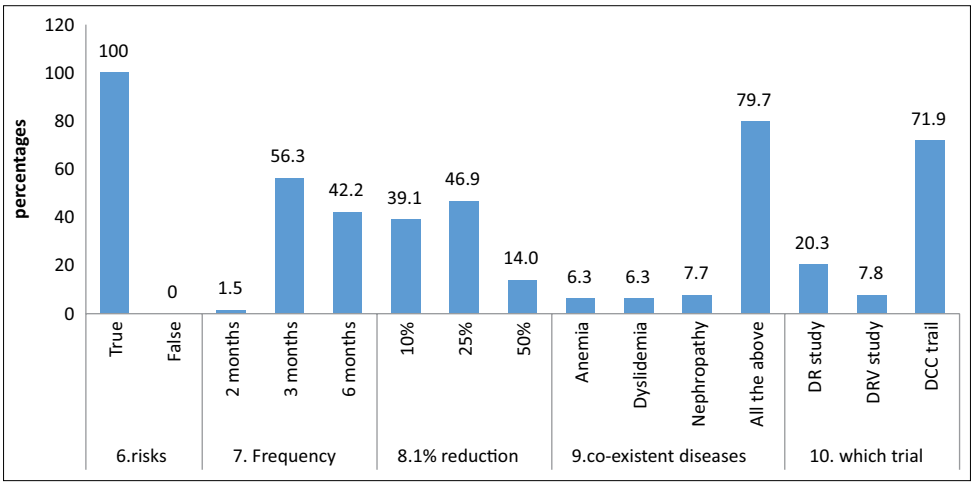


Figure 2: HbA1c questionnaire results for questions 6–10

Study (Option a), whereas 7.8% chose the Diabetic Retinopathy Vitrectomy Study (Option b).

DISCUSSION

Glycosylated hemoglobin is created by non-enzymatically adding a sugar residue to hemoglobin. It is possible to quantify glucose when it is bound non-enzymatically to a terminal portion of the Hb chain. This measurement is directly proportional to blood glucose concentration.[4] As the life span of RBCs is 120 days, this test, with allowances for the dynamics of RBCs production and disposal, indicates mean blood glucose over a 2–3 month period. Currently, the best method for measuring glycosylated hemoglobin is to use a fractionated HbA1c value.

The American Diabetes Association (ADA) recently recommended HbA1c with a cut-point of 6.5% as an alternative to fasting plasma glucose (FPG 7.0 mmol/L)-based criteria for diagnosing diabetes [Figure 3].[5]

Regarding the first question from our questionnaire, 65% of ophthalmologists recommended HbA1c for all diabetic patients, 27% for patients with diabetic retinopathy, 6% for patients with Proliferative Diabetic Retinopathy, and just 2% for all patients, regardless of whether they had diabetes or not! Therein lies the problem.

According to Ghoshal *et al.*[3] study, 61% of ophthalmologists advised HbA1c for all diabetic patients, 31% for patients with diabetic retinopathy, 3% for patients with Proliferative Diabetic Retinopathy, and 5% for all patients. However, ophthalmologists do not advise HbA1c as frequently as they should.

The Diabetes Control and Complications Trial (DCCT) Research Group randomly assigned 1441 type 1 diabetic patients to

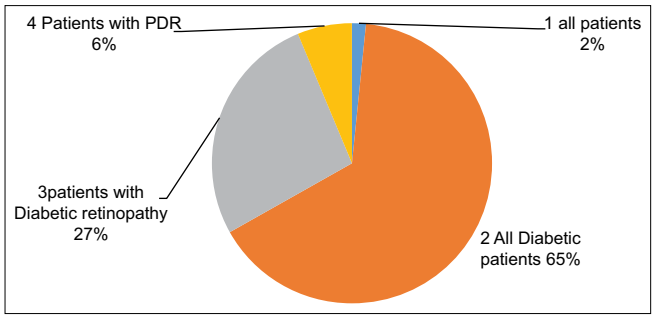


Figure 3: Ophthalmologists recommended HbA1c for which patients?

intensive glycemic therapy or standard care. In comparison with conventional treatment, intensive treatment (median HbA1c (glycosylated hemoglobin A1c), 7.2%) decreased the incidence of diabetic retinopathy by 76% and the progression of diabetic retinopathy by 54% throughout a follow-up period of 6.5 years.[6]

The UK prospective diabetic study[7] and Ohkubo *et al.*[8] both similarly demonstrate that intensive glycemic control [Hba1c <6.5%] significantly decreases the frequency and delays the onset and severity of diabetic microvascular complications. All ophthalmologists, general practitioners, and diabetologists seeing diabetic retinopathy patients should take the stance that proper management of all essential components of modern medical management includes plasma lipids, blood pressure, and blood sugar levels.[9] This is the second study that explores the knowledge and awareness of ophthalmologists regarding HbA1c as a potential index of glycemic control. The main limitation of this study is the small sample size.

CONCLUSION

To prevent diabetic patients from permanently losing their vision, ophthalmologists in Andhra Pradesh need to be more enlightened about HbA1c and its significance. Ophthalmologists should also educate their patients about the importance of regularly monitoring and controlling their HbA1c levels to slow the progression of diabetic retinopathy. All ophthalmologists should emphasize to their diabetic patients the importance of following their primary care physician's instructions to optimize metabolic control and should advise them to get their HbA1c levels checked regularly to prevent blindness.

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Conflicts of interest

There are no conflicts of interest.

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QUESTIONNAIRE

1. In your practice , you recommend HbA1c estimation for-
   a. All patients
   b. All diabetic patients
   c. All patients suffering from diabetic retinopathy(DR)
   d. All patients suffering from proliferative diabetic retinopathy(PDR)

2. Preparation needed before undergoing HbA1c estimation in a lab?
   a. Fasting for 6hours
   b. Fasting for 12hours
   c. 2hours following 150mg of glucose
   d. No preparation needed

3. HbA1c provides an average plasma glucose control for the last-
   a. 4-8weeks
   b. 8-12weeks
   c. 16-20weeks

4. The normal range of HbA1c is –
   a. 4 – 5.6%
   b. 5.6- 6.5%
   c. 6.5-7.5%

5. HbA1c target for good glycemic control for patients with with DR is –
   a. Below 6%
   b. Below 6.5%
   c. Below 7%
   d. Below 7.5%

6. The higher the HbA1c, the higher the risk of developing complications related to diabetics-
   a. True
   b. False

7. How frequently should you advice HbA1c for your diabetic patients?
   a. 2 months
   b. 3 months
   c. 6 months

8. Improving HbA1c by 1% in a diabetic cuts the risk of microvascular complications by –
   a. 10%
   b. 25%
   c. 50%

9. Co-existent diseases that can affect HbA1c value are-
   a. Anemia
   b. Dyspidemia
   c. Nephropathy
   d. All the above

10. Which trail recommended regular monitoring of HbA1c level in diabetics?
    a. Diabetic retinopathy study
    b. Diabetic retinopathy vitrectomy study
    c. Diabetes control and complication trail

Original Article

Dry eye and its association with digital screen exposure among medical students in a tertiary care hospital in South Kerala

ABSTRACT

Purpose: To assess the proportion of dry eye among medical students in a tertiary care hospital and determine its association with digital screen exposure. **Materials and Methods:** A cross-sectional study was conducted in the Ophthalmology department from October 2019 to April 2021 among 145 undergraduate medical students. Data regarding the pattern of digital screen use were collected from the students, and the symptomatology of dry eye was assessed with the help of the SPEED questionnaire. Dry eye tests were done, and data were analyzed. **Results:** Mobile phone was the most frequently used screen type by the medical students (80%). Furthermore, 53.1% spent more than 1 hour using digital screens in the dark, and 59.3% of participants regularly used digital screens for 3-5 years. In addition, 68.3% used the screens at 26%–50% brightness. Results revealed that 42.1% had dry eyes, out of which 53.1% had symptomatic dry eyes, and 49.7% tested positive for dry eyes. Duration of digital screen use in the dark was the factor that was associated the most with dry eye ($P = 0.004$). Increased duration of total screen use in years was found to have a significant association with dry eye among the students ($P = 0.020$). **Conclusion:** In this study, 42.1% of the students were diagnosed to have dry eyes. More than half of them were symptomatic. There is a significant association between digital screen exposure and dry eyes, with the longer duration of exposure and usage of screens in dim light being the key contributing factors.

Keywords: Digital screen exposure, dry eye, medical students

INTRODUCTION

Digital screen devices include computers, mobile phones, laptops, tablets, e-readers, and other electronic devices, and they are now an integral part of the medical field. Screens of such devices are categorized as video display terminals.[1] Reduced blink rates and increased corneal exposure with prolonged screen use result in excessive evaporation of tear film, which in turn causes an increased prevalence of dry eyes among prolonged screen users.[2]

The present study focused on determining the prevalence of dry eye and its association with digital screen exposure among medical students in a tertiary care hospital in South Kerala.

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MATERIALS AND METHODS

This cross-sectional study was conducted in the Department of Ophthalmology from October 2019 to April 2021 among 145 undergraduate medical students. The study was commenced after obtaining Scientific Committee and Institutional Ethics Committee clearance.

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Undergraduate medical students between 18 and 23 years of age who were willing to participate in this study were included. Contact lens wearers, those with a history of LASIK surgery or other corneal refractive surgeries, those with a history of other systemic diseases (such as diabetes, Sjogren's syndrome, connective tissue disorders, SLE, or thyroid-associated diseases), and those who were on psychiatric medications, antihypertensives such as beta blockers or diuretics, estrogens, or lubricant eye drops were excluded from the study.

Participants were randomly selected using a software tool for randomization and were given a brief introduction about the study. Informed consent was taken, and data regarding digital screen use were collected. They were requested to fill up the SPEED questionnaire of ocular symptoms related to dry eye. They also underwent dry eye tests such as Schirmer's test I and II, tear film break-up time (TBUT), and tear meniscus height (TMH) measurement.

According to the NEI workshop grading system, dry eye was diagnosed as one having one or more symptoms (dryness, grittiness, scratchiness, soreness, irritation, burning or watering, eye fatigue) and one or more positive clinical tests (Schirmer's test I and II, TBUT, TMH).[3] Asymptomatic students with positive signs or tests were also considered as having dry eyes for the purpose of this study.

Categorical and quantitative variables were expressed as frequency (percentage) and mean ± standard deviation, respectively. The severity of dry eye was expressed as a percentage. The Chi-square test was used to find the association between categorical variables. For all statistical interpretations, a *P* value of < 0.05 was considered the threshold for statistical significance. Statistical analysis was done using a statistical software package SPSS version 20.0.

RESULTS

Mobile phones were the most frequently used screen type by the students (n = 49, 33.8%). Thirty-four (23.4%) students used both computers and mobiles; 27 (18.6%) used laptops and mobiles; 15 (10.3%) used computer screens only; 11 (7.6%) used computer screens together with tabs/iPad; four (2.8%) utilized laptops, tabs/iPads; and mobiles; three (2.1%) used tabs/iPads alone; and two (1.4%) students used computer screens, mobiles, and tabs/iPads. Table 1 shows the pattern of digital screen use among the students.

In total, 102 students used both devices and books as study tools (70.3%), while 42 students (29%) used only books. Only one student (0.7%) studied solely using devices. Most of the students (n = 109, 75.2%) used digital screens with free intervals in between, while the remaining students (n = 36, 24.8%) used the screens continuously.

Most of the students (n = 94, 68.3%) used the screens at 26%–50% brightness. Thirty-nine students (26.9%) used 11%–25% screen brightness, while 10 students (6.9%) and two students (1.4%) used 51%–75% and >75% brightness settings, respectively.

According to Schirmer's Test 1, 44 (30.3%) students were found to have mild dry eyes, 19 (13.1%) had moderate dry eyes, and two (1.4%) had severe dry eyes. Based on Schirmer's Test II, 39 (26.9%) students had mild dry eyes, while 19 (13.1%) and two (1.4%) students had moderate and severe grades of dry eye, respectively. Furthermore, 78 (53.8%) students showed a normal TBUT, while 67 (46.2%) had abnormal TBUT. TMH was normal in 137 (94.5%) and abnormal in eight (5.5%).

Seventy-seven students (53.1%) had symptomatic dry eyes, and 72 (49.7%) had positive dry eye tests, as shown in Table 2.

Table 1: Pattern of digital screen use among medical students

Duration of screen use	*n*	Percentage
Regular screen use (in years)		
1–2	59	40.7
3–5	86	59.3
Screen use/day (in hours)		
1–2	57	39.3
2–3	60	41.4
3–4	24	16.6
4–6	4	2.8
Screen use in the dark (in hours)		
<1	68	46.9
1–2	77	53.1

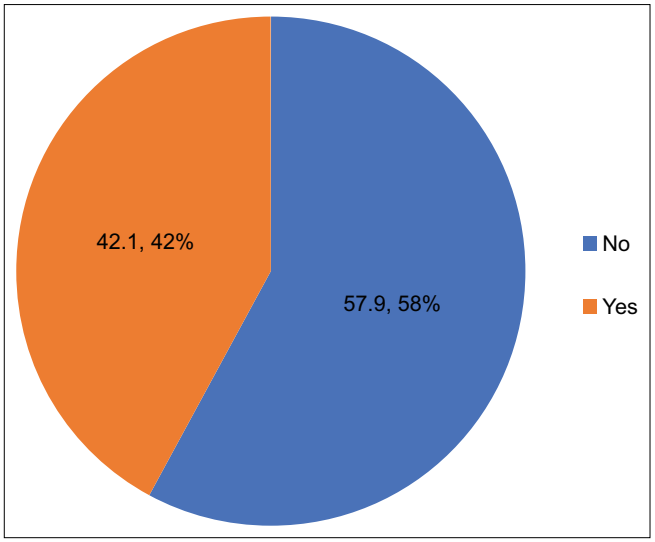


Figure 1: Proportion of medical students with dry eye in percentage

Based on the presence of one or more dry eye symptoms and one or more positive clinical tests, final grading was done. Asymptomatic students with positive signs or tests were also considered as having dry eyes. As shown in Figure 1, 61 (42.1%) undergraduate medical students were diagnosed with dry eyes, whereas 84 (57.9%) students did not have dry eyes.

The most significant factor associated with dry eyes was the duration of digital screen use in dim light, with a *P* value of 0.004. Increased duration of screen use in terms of total years of screen use and in terms of total hours spent using digital screens was found to have a significant association with dry eye among the students ($P = 0.020$ and 0.036, respectively). Continuity of screen use and brightness levels had no statistically significant association with dry eye, as shown in Table 3.

DISCUSSION

This study was done among 145 medical students to assess the presence of dry eyes among them and to find out its association with digital screen use. Digital screens, mainly mobile phones and computers, were being widely used by the medical student population for academic purposes, together with books. The mobile phone was the device that was being used most frequently by the students, followed by computer screens and laptops.

Table 2: Prevalence of dry eye among medical students based on symptoms and tests

Dry eye prevalence	Count	Percentage
No symptomatic dry eye	68	46.9
Symptomatic dry eye	77	53.1
Positive dry eye tests	72	49.7
Negative dry eye tests	73	50.3

The proportion of dry eye among medical students was 42.1% according to this study. Only 53.1% of the students complained of dry eye symptoms; 49.7% of the students tested positive for dry eyes. The prevalence of symptomatic dry eye was reported to be 44.3% in a cross-sectional study conducted by Asiedu *et al*.[4] among 700 undergraduate students at Ghana University, which was comparable to our findings. In another study done by Altalhi *et al*.[5] among Health Science students in Saudi Arabia, the prevalence of dry eyes was found to be 48.3%, which was also consistent with our study.[5]

Most of the students in our study spent around 1–3 hours per day using such devices, with many of them using digital screens in the dark, which was found to have the most significant association with the prevalence of dry eye. Increased duration of screen use in terms of the total number of years of screen use and in terms of total hours spent using digital screens were found to have a significant association with dry eye prevalence among the students. Continuity of screen use and brightness levels were not found to have a statistically significant association with dry eyes. Most of the students used to take breaks in between using screens, and they used the screens at moderate brightness settings. Several studies have reported that people who used digital screens more often had a greater frequency of dry eye disease.[6-14] Incomplete blinking of eyes may be the major factor contributing to dry eyes, and application of lubricating eye drops may reduce symptoms of dry eyes.[15]

In this study, 42.1% of the students were diagnosed with dry eyes, and more than half of them were symptomatic. SPEED questionnaire is an imperative tool in identifying dry eyes in the early stages, and we found that there is a significant association between digital screen exposure and dry eyes,

Table 3: Association of digital screen exposure with dry eye among medical students

Digital screen exposure	Dry eye		χ^2	*P*
	No (Count/Percentage)	Yes (Count/Percentage)		
1–2 hours per day	40/70.2	17/29.8	6.64*	0.036
2–3 hours per day	32/53.3	28/46.7		
>3 hours per day	12/42.9	16/57.1		
<1 h of use in the dark	48/70.6	20/29.4	8.42**	0.004
>1 hour of use in the dark	36/46.8	41/53.2		
Continuous use	25/69.4	11/30.6	2.6	0.107
Interrupted use	59/54.1	50/45.9		
11%–25% brightness	22/56.4	17/43.6	1.62	0.444
26%–50% brightness	57/60.6	37/39.4		
>50% brightness	5/41.7	7/58.3		
Usage during the daytime	41/54.7	34/45.3	0.68	0.410
Usage at night	43/61.4	27/38.6		
1–2 years of regular screen use	41/69.5	18/30.5	5.46*	0.020
3–5 years of regular screen use	43/50.0	43/50.0		

mainly the higher duration of exposure and usage of screens in dim light.

The main limitation of our study was that it was a cross-sectional study and involved only students of a single medical institution, which reduces the ability to generalize. Participants would have either underestimated or overestimated their symptoms while self-reporting the SPEED questionnaire, and this would have had an impact on the outcome.

Despite being perceived as being less tech-savvy than students in hard-core technical fields, medical undergraduates are widely adopting digital gadgets. Therefore, it is important to evaluate the effects of these gadgets on them. If misuse and overuse of these gadgets are not controlled, their benefits might be seriously compromised. As the use of digital screens is now inevitable and has become an essential part of medical education, it is imperative to take a look at the pros and cons of screen use and to make necessary modifications in the pattern of use of these devices.

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Nil.

Conflicts of interest
There are no conflicts of interest.

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Original Article

A study on the impact of glycemic status on central corneal thickness and intraocular pressure in diabetic patients attending a tertiary care center in central Kerala

ABSTRACT

Purpose: This study aims to evaluate the central corneal thickness (CCT), intraocular pressure (IOP), and glycemic status in uncontrolled diabetic patients at baseline, 1 week, and 12 weeks, and to examine the relationship between changes in CCT and IOP with fluctuations in glycemic status. **Methods:** Diabetic individuals with hyperglycemia, defined as having postprandial blood sugar (PPBS) levels exceeding 200 mg/dl and glycated hemoglobin (HbA1c) greater than 7%, aged between 18 and 80 years, were enrolled in the study. Glycemic status was evaluated through fasting blood sugar (FBS), PPBS, and HbA1c levels. Participants underwent a comprehensive ocular examination, CCT measurement utilizing Carl Zeiss Spectral Domain Anterior Segment Optical Coherence Tomography (AS-OCT), and IOP measurement using Goldmann Applanation Tonometer. Participants were advised to pursue treatment for glycemic management. Follow-up evaluations were conducted at 1 week and at 12 weeks. **Results:** The average CCT was 560.9+/ 22 µm during the initial visit, 560.7+/ 22 µm at 1 week, and 539.0+/ 61.1 µm at 12 weeks (p 0.008). The mean IOP was 17.2+/ 2.7 mm Hg at the first visit, 17.1+/ 2.7 mm Hg at 1 week, and 15.3+/ 2.6 mm Hg at 12 weeks (*P* 0.01). **Conclusion:** Achieving optimal glycemic control in diabetic patients leads to a significant reduction in CCT and IOP. Stable euglycemia as evidenced by HbA1c at 3 months is a determinant of reliable IOP measurement. Hence we recommend that healthcare providers should look into the glycemic status, specifically HbA1C once in 3 months during glaucoma related decision making in diabetic patients.

Keywords: Central corneal thickness, diabetics, glaucoma, glycated hemoglobin, glycemic control, intraocular pressure

INTRODUCTION

Diabetic subjects showed higher central corneal thickness (CCT) as compared to non-diabetic subjects and there was an increase in CCT with an increase in duration and uncontrolled diabetes.[1] Thicker or thinner central corneas may lead to either overestimation or underestimation of intraocular pressure (IOP).[2] Very few studies have investigated the changes in CCT in different glycemic status in the same individual. This study reveals how clinically significant it is to know the glycemic status of an individual while measuring IOP. In the present study, we have correlated the changes in CCT and IOP with variations in glycemic status.

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MATERIALS AND METHODS

Sixty diabetic patients attending the Department of Ophthalmology at a tertiary care center in central Kerala between December 2019 and June 2021 were included in this prospective cohort study by consecutive sampling after getting the Institutional Ethics Committee Approval. Diabetic

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patients in hyperglycemic states whose postprandial blood sugar levels were more than 200 mg/dl and HbA1c > 7% were included. Those with corneal edema, pathologies, or any alteration of the corneoscleral architecture, those who were on long-term ocular medications or steroids, dialysis, or had systemic diseases influencing IOP (e.g. thyroid disorders, pregnancy, myotonic dystrophy, etc.) were excluded from the study. Patients who were unable to cooperate or to be positioned for an ideal ocular examination were also excluded.

The glycemic status of diabetic patients was assessed by fasting blood sugar (FBS), PPBS (Glucose oxidase peroxidase method), and glycated hemoglobin (HbA1c) levels (HPLC; High-performance liquid chromatography method). They were then assessed for visual acuity, detailed ocular examination, CCT, and IOP. CCT was measured using Carl Zeiss (Germany) Spectral Domain AS-OCT (5-line raster). IOP was measured using the Goldmann Applanation Tonometer. Patients were advised to undergo treatment for glycemic control. The patient was reassessed for visual acuity, CCT, and IOP along with FBS and PPBS values at 1 week and again at 12 weeks following the initial visit along with HbA1c.

Sample size calculation

Sample size: 60

The sample size was calculated from the variables obtained from the study "The change in CCT after control of hyperglycemia in diabetic patients" by Yesim

Altay *et al*. 2014[3]

$$\text{Npairs} = \frac{(z1+\frac{\alpha}{2}+z1-\beta)2}{\Delta 2}+\frac{z^2_{1-\alpha/2}}{2}$$

$$\Delta = \frac{(\mu 2-\mu 1)}{\sigma} \qquad \sigma = \frac{\sigma 1+\sigma 2}{2}$$

$\mu 1$ = Pre-test mean CCT (552.30)

$\mu 2$ = Post-test mean CCT (542.36)

$\sigma 1$ = Standard deviation in the Pre-test (29.26)

$\sigma 2$ = Standard deviation in the Post-test (27.20)

Δ = Effect size

α = Significance level (5%) = 1.96[2]

$1-\beta$ = power 80% (0.84)

n = 60

Statistical analysis

The data collected was entered into an Excel worksheet and analysis was performed using SPSS software version 23. Descriptive and inferential statistical analysis was carried out. Results of continuous measurements are presented on mean ± SD and results on categorical measurements are presented in number (%). A *P* value of < 0.05 was considered significant. Changes in CCT and IOP have been analyzed using a paired "t" test between each visit and an ANOVA test to analyze across the visits.

RESULTS

Among the 60 patients, 73.3% of our study population was above 50 years and 63.3% of the study population were males [Figure 1]. The major comorbidity was hypertension followed by cardiovascular disease and dyslipidemia. Out of the study population, 28.3% had diabetes for 5 years followed by 25% population who had diabetes for 11–15 years [Figure 2].

Comparison of FBS at different intervals

Table 1 showed that the average FBS level at the initial visit was 240.0 ± 70.4, at 1 week it was 103.0 ± 21.2, and at 12 weeks it was 93.1 ± 11.4, showing statistically significant differences ($P < 0.01$). The mean difference between the initial visit and 1 week was 137, between the initial visit and 12 weeks it was 146.9, and between 1 week and 12 weeks, it was 9.9. These differences in FBS levels between visits were also found to be statistically significant ($P < 0.01$).

Comparison of postprandial blood sugar at different intervals

The average PPBS level at the initial visit was 292.5 ± 58.7 mg/dL, at 1 week it was 160.6 ± 30.0 mg/dL, and at 12 weeks it was 161.3 ± 20.9 mg/dL, demonstrating statistically significant differences ($P < 0.01$). The mean difference between the initial visit and the first week was 131.95 mg/dL, and between the initial visit and 12 weeks, it was 131.18 mg/dL (statistically significant, *P* value < 0.05). However, the mean difference between 1 week and 12 weeks was 0.77 mg/dL, which was not statistically significant [Table 2].

Comparison of Hb A1c (Glycated hemoglobin)

Here in this study, we found that the mean HbA1c level at the initial visit was 10.2 ± 2.5, and at 12 weeks it was 6.5 ± 0.7 [Table 3]. The mean difference in HbA1c between the initial visit and 12 weeks was 4.1, which is statistically significant ($P < 0.01$).

Comparison of CCT at different time intervals

The average CCT during the initial visit (hyperglycemic status) was 560.9 ± 22 µm, at 1 week it was 560.7 ± 22 µm, and at 12 weeks (euglycemic status) it was 539.0 ± 61.1 µm [Table 4].

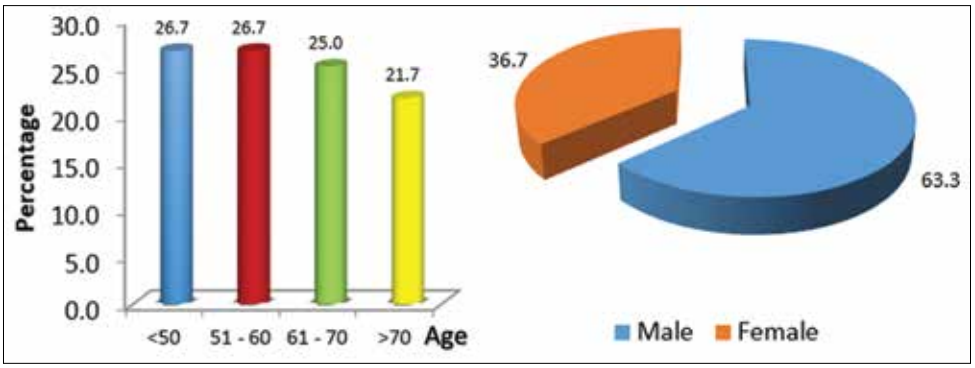


Figure 1: Percentage distribution of age and sex

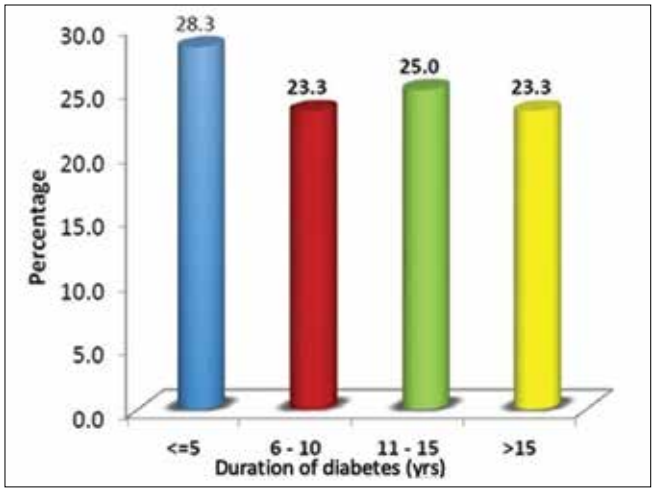


Figure 2: Percentage distribution according to duration of diabetes

The variation in CCT across the three visits was statistically significant ($P<0.008$). The mean difference in CCT between 1st visit and 1 week was 0.18 which is not statistically significant ($P=0.711$). The mean difference in CCT between 1 week and 12 weeks is 21.88 ($P=0.022$) and between 1st visit and 12 weeks was 21.7 which is statistically significant ($P=0.024$).

Comparison of IOP at different time intervals

The average IOP during the initial visit (hyperglycemic status) was 17.2 ± 2.7 mmHg, at 1 week it was 17.1 ± 2.7 mmHg, and at 12 weeks (euglycemic status) it was 15.3 ± 2.6 mmHg. [Table 5] The variation in IOP across the visits was statistically significant (*P* 0.01). The mean difference in IOP between 1st visit and the 1 week was 0.04 which is not statistically significant (*P* 0.54). The mean difference between 1 week and 12 weeks was 1.9 (*P* 0.001) and between the 1st visit and 12 weeks was 1.85 (*P* 0.001) which are statistically significant.

DISCUSSION

In our investigation, the predominant age group comprised individuals aged between 50 and 60 years, with a mean age of 58.5 ± 12.5 years. A study conducted by Sari *et al*.[4] similarly reported an age range of 43 to 79 years. Additionally, Altay *et al*. found a mean age of 58.75 years, ranging from 46 to 73 years. Regarding gender distribution, our study demonstrated a male predominance of 63.3%. Altay *et al*.[3] observed that 51.9% of participants were male and 48.1% were female. Conversely, Prabhavati K *et al*.[5] reported a higher proportion of female patients.

Within our study population, 28.3% had been diagnosed with diabetes for 5 years, followed by 25% who had been diabetic for 11–15 years. In a study conducted by Altay *et al*., the duration of diabetes exceeded 10 years in 32 patients (61.5%). Notably, individuals with diabetes lasting 10 years or more exhibited a marginal elevation in CCT compared to those with duration of diabetes less than 10 years.[3]

Table 1: Comparison of fasting blood sugar (FBS) at different intervals

Stage	FBS (mg/dL)	Pair	Difference	*P*
I st Visit (A) (*n*=60)	240±70.4	A and B	137	0.000**
I week (B) (*n*=60)	103±21.2	A and C	146.9	0.000**
12 weeks (C) (*n*=60)	93.1±11.4	B and C	9.9	0.002**

**Highly Significant at 0.01 level

Table 2: Comparison of postprandial blood sugar (PPBS) at different intervals

Stage	PPBS (mg/dL)	*P*	Pair	Difference	*P*
I st Visit (A) (*n*=60)	292±58.7		A and B	131.95	0.000**
I week (B) (*n*=60)	160.6±30	*P*<0.01	A and C	131.18	0.000**
12 weeks (C) (*n*=60)	161.3±20.9		B and C	0.77	1.000

**Highly Significant at 0.01 level

Table 3: Comparison of Hb A1c (Glycated hemoglobin)

Stage	HbA1c (%)	Difference	*P*
I st Visit (*n*=60)	10.2±2.5	4.1	*P*<0.01**
12 weeks (*n*=60)	6.2±0.7		

**Highly Significant at 0.01 level

Table 4: Comparison of central corneal thickness (CCT) at different time intervals

Stage	CCT (μm)	F#	P	Pair	Difference	P
Ist Visit (A) (n=60)	560.9±22	7.61		A and B	0.18	0.711
I week (B) (n=60)	560.7±22		P<0.008**	A and C	21.88	0.022*
12 weeks (C) (n=60)	539±61.1			B and C	21.7	0.024*

**Highly Significant at 0.01 level, *Significant at 0.05 level

Table 5: Comparison of intraocular pressure (IOP) at different time intervals

Stage	IOP (mm Hg)	F#	P	Pair	Difference	P
Ist Visit (A)	17.2±2.7	55.6	0.000**	A and B	0.05	0.546
1 week (B)	17.1±2.7			A and C	1.9	0.001**
12 weeks (C)	15.3±2.6			B and C	1.85	0.001**

**Highly Significant at 0.01 level

Ultrasound pachymetry (USP) is currently the gold standard to measure CCT. It has some benefits, including its quick measurement time and portability. However, its accuracy is mostly examiner-dependent. Any misalignment in probe placement can cause measurement errors. USP probes placed directly onto the cornea can cause corneal injury. Anterior segment OCT evaluates anterior segment structure and measures corneal thickness in a non-contact manner. CCT readings obtained by AS-OCT (540.86 ± 29.81 μm) were significantly lower than those obtained by USP (554.43 ± 30.42 μm).[6] Though both devices have good repeatability, AS-OCT has slightly better repeatability than ultrasound pachymetry.

Claramonte *et al*.[7] found that diabetic patients had a higher CCT than healthy people. Ultrasound pachymetry was employed in this study. Diabetic patient's CCT averaged 571.96+/−26.81 microns, with a range of 514 to 626 microns. In our study where we used AS-OCT for CCT measurement, the average CCT during the initial visit (hyperglycemic status) was 560.9 ± 22 μm, at 1 week it was 560.7 ± 22 μm, and at 12 weeks (euglycemic status) it was 539.0 ± 61.1 μm. The increased CCT obtained in our study can be attributed to the hyperglycemic status and the prolonged duration of diabetes. In non-diabetic individuals, the average CCT was 544.89+/−35.36 microns, with a range of 448 to 649. They found out that diabetic individuals had a statistically significant increase in CCT as compared to non-diabetic patients and lower endothelial cell counts were linked to higher HbA1c levels in the diabetes group.

Calvo-Maroto *et al*.[8] found that when comparing long-term diabetes patients (10 years or more from diagnosis) versus short-term patients, the CCT was greater and the endothelial cell counts (ECD) were much lower. In both CCT and ECD, multivariate analysis of variance revealed a significant influence on diabetes duration.

In our study, the mean HbA1c level at the initial visit was (10.2 ± 2.5) %, and at 12 weeks it was (6.5 ± 0.7) %. The mean difference in HbA1c between the initial visit and 12 weeks was 4.1, which was statistically significant ($P < 0.01$). Similar findings were reported by Altay *et al*.[3] Previous research has demonstrated a correlation between HbA1c levels and visual acuity (VA), IOP, CCT, and the duration of diabetes. In individuals with poorly controlled diabetes, higher HbA1c levels were associated with elevated IOP. This association was more pronounced when the HbA1c level exceeded 9.5 but not when it was below 9.5.[9] M O Zengin *et al*.[10] also found that diabetic individuals have much greater CCT than healthy people. The Orbscan II corneal topography was used to determine CCT. They also saw that diabetics with HbA1c levels of more than 7% had thicker corneas than diabetics with HbA1c levels of less than 7%. Additionally, Lathika *et al*.[11] suggested that high plasma glucose levels could act as a confounding factor affecting the accuracy of applanation readings in diabetic patients, particularly in cases of severe hyperglycemia.

Several theories have been proposed to elucidate the morphological changes and swelling observed in the corneas of diabetic patients. The accumulation of excess glucose leads to heightened intracellular sorbitol levels in diabetic corneas, acting as an osmotic agent and inducing endothelial cell swelling.[12] Furthermore, decreased adenosine triphosphate production from the Krebs cycle in these patients affects endothelial pump function. Comparative studies between diabetic patients and healthy controls have revealed increased corneal thickness, variability in endothelial cell size, and reductions in endothelial cell density and hexagonality, supporting the notion that type 2 diabetes mellitus can impact corneal endothelial cells.[13] In our study, the mean CCT during the initial visit (hyperglycemic state) was 560.9 ± 22 μm, at 1 week it was 560.7 ± 22 μm, and at 12 weeks (euglycemic state) it was 539.0 ± 61.1 μm. The observed variation in CCT across the three visits was statistically significant (P 0.008). Thus, CCT was elevated in the hyperglycemic state, while it decreased in the euglycemic state over the long term (12 weeks).

In the study by S. Math *et al*.,[14] diabetic subjects compared to non-diabetic subjects showed higher CCT which was related to longer duration and poor glycemic control of diabetes. CCT

was measured by ultrasound pachymetry. A study conducted by Amutha Bharathy *et al*.[15] in one hundred patients revealed that the mean CCT was 502.20 ± 25.05 microns in the hyperglycemic condition and 503.38 ± 25.18 microns in the euglycemic state after a one-month period. Interestingly, no significant difference in CCT was observed between euglycemic and hyperglycemic states within the same group of individuals. Similarly, Dona George *et al*.[16] found that in the hyperglycemic state, the mean CCT was 501.38 ± 25.28 microns, and upon reassessment after one month in the euglycemic state, it was 502.20 ± 25.05 microns, indicating no significant difference in CCT between hyperglycemic and euglycemic states. In our study, CCT values did not exhibit significant differences when assessed shortly after achieving euglycemia (1 week). However, with the attainment of euglycemia for 12 weeks, the results became statistically significant. This underscores the impact of long-term blood sugar fluctuations on CCT rather than short-term fluctuations. Additionally, Chaudhury *et al*.[17] compared CCT and endothelial cell count between diabetics and non-diabetics, noting that the mean CCT was higher and mean endothelial cell density was lower among the diabetic group compared to the non-diabetics group (*P* value < 0.05). M Zulhisham *et al*.[18] found that the mean CCT in children with type 1 uncontrolled diabetes mellitus (T1DM) was significantly higher than that of the control group which was attributed to the imbalance created by the upregulation of matrix metalloproteinases (MMP-3 and MMP-10) and downregulation of tissue inhibitors of matrix metalloproteinases (TIMP-4), thereby increasing the synthesis of the extracellular matrix in the corneal stroma. AS-OCT was used in this study to measure the CCT. Our study too showed similar results.

The prevalence of glaucoma in diabetics is 4.96% to 14.6%.[19] CCT has been shown to be an independent risk factor for the development and progression of glaucoma.[20] According to the Ocular Hypertension Treatment Study, CCTs of 555 ∝m or below had a 3-fold increased risk of development of glaucoma compared to those with CCTs greater than 588 ∝m.[21] According to Prabhakar *et al*.,[22] variations in CCT can result in either overestimation or underestimation of IOP. Studies have indicated that the CCT profile influences IOP measurements obtained using Goldmann applanation tonometry. In the study by Ehlers *et al.*,[23] applanation tonometry underestimated IOP by a mean of 5.2 mm Hg in eyes with a CCT of 450 μm and an overestimation of 4.7 mm Hg when the CCT was 590 μm, at a true IOP of 20 mmHg. Kohlhaas found that there is a change of 1 mmHg for every 25 ∝m increase in CCT in eyes with CCT greater than 550 ∝m.[24] The important fact here is that we should think twice before labeling a diabetic patient as a case of glaucoma unless his blood sugars are controlled. Numerous studies have found a significant association between blood glucose levels and IO*P* values in diabetic patients. Postprandial IOP tends to be notably higher compared to baseline in these patients, revealing a strong link between the magnitude of hyperglycemia and IOP.[25] Larsen *et al*.[26] reported lower IO*P* values during episodes of severe hypoglycemia. Additionally, Traisman *et al*.[27] observed higher IO*P* values when blood glucose levels exceeded 200 mg/dL in patients with hyperglycemia (mean difference = 1.3 mmHg) consistent with our findings. The mean IOP during the initial visit (hyperglycemic state) was 17.2 ± 2.7 mmHg, at 1 week it was 17.1 ± 2.7 mmHg, and at 12 weeks (euglycemic state) it was 15.3 ± 2.6 mmHg. The difference in IOP across the three visits was statistically significant (*P* 0.01).

A significant difference in CCT and IOP was observed between hyperglycemic and euglycemic states at 12 weeks. In the meta-analysis by Doughty and Zaman, a 10% change in CCT resulted in a change in IOP of approximately 3.4 mmHg.[28] Some authors suggest that hyperglycemia creates an osmotic gradient, leading to fluid shifts into the intraocular space. Variations in blood glucose levels may influence changes in IOP, with an average IOP variation (between baseline and postprandial measurements) of 15% for an average glycemic variation of 40%.[29] This may have a significant impact on glaucoma screening and diagnosis in the diabetic population. Ensuring diabetic control is essential before altering treatment decisions in patients with glaucoma and diabetes.

Our study also had a few limitations. First, our sample size was relatively small and following up the same cohort over time could have minimized confounders. Second, CCT-corrected IOP was not taken into account since we did not look into the glaucoma treatment aspects. Thirdly, the status of corneal endothelium was not evaluated in our study, which would have added potential insights into underlying corneal changes among patients with diabetes. Further research on larger groups of diabetic patients for a longer period of time as well as assessment of factors other than those analyzed in this study would provide further clarity to our findings.

CONCLUSION

Short term glycemic fluctuations measured by FBS/PPBS do not influence CCT/IOP significantly where as stable euglycemia as evidenced by HbA1c at 3 months is a determinant of reliable IOP measurement.Hence we recommend that healthcare providers should look into the glycemic status, specifically HbA1C once in 3 months during glaucoma related decision making in diabetic patients.

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Conflicts of interest

There are no conflicts of interest.

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Original Article

Outreach eye camps: Providing comprehensive vision care in underserved areas of North India: Meeting the unmet

ABSTRACT

Purpose: To evaluate the impact and outcome of outreach eye camps in providing comprehensive vision care in underserved areas of North India, where a significant number of individuals lack adequate vision care facilities. **Methods:** The article presents a descriptive study design. Purposive sampling was employed to select participants who had attended outreach eye camps during a specified period. Primary data were collected through structured interviews, clinical assessments, and vision screening procedures. Data analysis was done through descriptive statistical analysis, comparative analysis, and thematic analysis of qualitative data. **Results:** A total of 924 patients participated in the eye camps conducted in underserved communities. Refractive errors, cataracts, glaucoma, and conjunctivitis were commonly observed ocular morbidities. **Conclusion:** These camps bridge the gap between marginalized communities and essential eye care services, offering a range of interventions to address the unmet vision care needs of diverse populations.

Keywords: Cataract, comprehensive eye care, outreach eye camps, refractive errors

INTRODUCTION

Access to comprehensive eye care services is a critical aspect of healthcare that remains a challenge for underserved populations worldwide. In such underprivileged areas, individuals face barriers such as geographical remoteness, limited healthcare infrastructure, and financial constraints, which restrict their access to essential eye care. As a result, visual impairment and preventable blindness disproportionately affect these communities, hindering their overall well-being and socioeconomic development. North India, with its diverse population and challenging socioeconomic conditions, is one such region where a significant number of individuals lack adequate vision care facilities. In response to this urgent need, outreach eye camps have emerged as a promising approach to deliver comprehensive vision care services directly to the doorstep of underserved communities. The core objective of eye camps is to detect and treat common eye disorders, such as refractive errors, cataracts, and infections, while also raising awareness about eye health and promoting preventive measures within these communities.[1,2]

Studies have shown that these camps play a crucial role in reducing visual impairment and preventing blindness by providing timely and appropriate interventions.[3,4] Moreover, they contribute to raising awareness about eye health and promoting preventive measures in communities where eye care knowledge is limited.[5] These camps are typically organized by collaborations between government agencies,

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non-governmental organizations (NGOs), local communities, and healthcare professionals, with the aim of addressing both short-term and long-term eye care needs. Additionally, advancements in technology, such as telemedicine and mobile health applications, have facilitated efficient patient management, remote consultations, and timely referrals to higher-level facilities.[6]

This article highlights the significance of outreach eye camps as a means to provide complete vision care services to underserved areas of North India. By addressing the unique challenges faced by these communities, outreach eye camps have the potential to alleviate the burden of visual impairment and preventable blindness, improve the quality of life for affected individuals, and contribute to the overall development of the region. Through rigorous research and analysis, we can pave the way for sustainable and scalable solutions that ensure equitable access to eye care for all.

METHODS

Study design

This scientific paper utilized a descriptive study design to investigate the provision of comprehensive vision care through outreach eye camps in underserved areas of North India over a period of two years. The study aimed to evaluate the impact and outcomes of these camps in addressing the unmet vision care needs of the target population.

Study setting

The study was conducted in various underserved areas of North India, including rural villages and remote communities. The selection of the study sites was based on the accessibility of the population, considering areas with limited access to eye care facilities and services.

Sample selection

The study employed a purposive sampling technique to select participants who had attended outreach eye camps during a specified period. The sample included individuals of different age groups and both genders, representing the diverse population in the underserved areas.

Data collection

Primary data were collected through a combination of structured interviews, clinical assessments, and vision screening procedures. The interviews aimed to gather information on participants' demographic characteristics, past eye health history, and their experiences with the outreach eye camps. Clinical assessments involved visual acuity testing, refraction measurements, and ocular examinations conducted by trained ophthalmologists and optometrists. Additionally, data regarding the types of services provided, such as refractive error correction, cataract surgeries, and medication distribution, were also collected.

Data analysis

Descriptive statistical analysis is employed to summarize the collected data, including frequency distributions, means, and standard deviations. Comparative analysis is conducted to evaluate changes in visual acuity and assess the impact of the outreach eye camps on the prevalence of ocular conditions. Subgroup analysis may be performed to explore variations in outcomes based on demographic factors, such as age and gender. Qualitative data from interviews are thematically analyzed to gain insights into participant experiences and perceptions.

Ethical considerations

Ethical approval is obtained from the institutional ethics committee to ensure participant protection and compliance with ethical guidelines. Informed consent is obtained from all participants prior to data collection, and confidentiality of their personal information is strictly maintained.

Limitations

This study acknowledges several limitations, including the potential for selection bias due to the convenience sampling method. Additionally, the absence of a control group and the reliance on self-reported data may introduce certain limitations in terms of generalizability and accuracy of outcomes.

RESULTS

A total of 924 patients participated in the eye camps conducted in underserved communities. The demographic characteristics of the participants include age distribution, gender ratio, and socioeconomic background. The majority of participants belonged to low-income households, with ages ranging from 5 to 80 years. Among the 924 patients, 50.9% were female and 48.7% were male, indicating a relatively equal gender representation [Table 1].

The types of eye conditions diagnosed and treated during the eye camps are presented in Table 2. The most common conditions encountered were refractive errors (32.5%), cataracts (21.6%), glaucoma (16.2%), and conjunctivitis (10.8%). Other less prevalent conditions included pterygium, diabetic retinopathy, and corneal disorders. The provision of eyeglasses for refractive errors (43.3%), followed by cataract surgeries were the most frequently provided treatments.

(16.2%) [Table 3]. The outcomes of the interventions provided during the eye camps in underserved communities are presented in Table 4. Among the 924 cases analyzed, the most common outcome was improved vision, accounting for 54.1% of the total. These results demonstrate the effectiveness of the interventions provided during the eye camps in improving the vision and overall eye health of the participants in underserved communities.

DISCUSSION

Eye camps play a crucial role in addressing preventable causes of blindness, particularly in cases of cataracts, and have shown promising results in improving overall visual health and reducing the burden of eye diseases in underserved areas. In the vast expanse of North India, where vibrant cultures and diverse communities flourish, there exists a stark reality: the unmet vision care needs of underserved areas. The participation of 924 patients in these camps demonstrates the substantial demand and need for accessible eye care services in these areas. The demographic characteristics of the participants reveal a diverse population, with a majority of adults (>18 years) comprising 67.5% of the patients. The higher representation of adults emphasizes the importance of addressing visual impairments and eye conditions that can significantly impact their daily lives and productivity. Similar studies have reported the substantial demand for eye care services in underserved areas, as evidenced by the high participation rates observed in our study.[7,8] The demographic characteristics of the participants, including age and gender distributions, are consistent with previous studies that have highlighted the diverse population seeking eye care in such settings.[9-11]

Table 1: Demographic characteristics of participants

Characteristic	Number of Participants	Percentage
Age Range		
Children (0–18 years)	300	32.5%
Adults (>18 years)	624	67.5%
Gender		
Male	450	48.7%
Female	470	50.9%
Other	4	0.4%

Table 2: Types of eye conditions diagnosed and treated

Eye Condition	Number of Cases	Percentage
Refractive errors	300	32.5%
Cataracts	200	21.6%
Glaucoma	150	16.2%
Conjunctivitis	100	10.8%
Corneal infections	50	5.4%
Other eye conditions	124	13.4%
Total	924	100%

Table 3: Interventions provided during eye camps

Intervention	Number of Cases	Percentage
Prescription glasses	400	43.3%
Cataract surgeries	150	16.2%
Medication for eye infections	50	5.4%
Referrals for further treatment	100	10.8%
Health education and awareness	124	13.4%
Other interventions	100	10.8%
Total	924	100%

Table 4: Outcomes of interventions

Intervention Outcome	Number of Cases	Percentage
Improved vision	500	54.1%
Resolution of eye condition	200	21.6%
Reduced symptoms	100	10.8%
Increased knowledge	75	8.1%
Satisfactory patient experience	40	4.3%
Other outcomes	9	1.0%

Refractive errors, cataracts, glaucoma, and conjunctivitis were commonly seen in ocular morbidities in underserved areas.[10-12] These conditions have consistently been identified as major contributors to visual impairment and blindness in low-income communities.[7,9,10] Furthermore, the high prevalence of refractive errors and cataracts underscores the importance of providing accessible and affordable refractive services and surgical interventions, respectively, to address these significant causes of visual impairment.[7-10] The interventions provided during the camps showcased a comprehensive approach to vision care. Prescription glasses were the most common intervention followed by cataract surgeries, emphasizing the importance of correcting refractive errors and improving visual acuity. These interventions reflect the holistic approach of the outreach eye camps in addressing not only immediate eye care needs but also long-term eye health. The outcomes of the interventions demonstrate the effectiveness of the eye camps in achieving positive results. Improved vision was reported in 54.1% of the cases, indicating the success of refractive corrections, cataract surgeries, and other interventions in enhancing visual function. Resolution of the eye condition was observed in 21.6% of the cases, indicating successful management and treatment of various ocular pathologies. Similar results have been reported in previous studies in terms of visual improvement, symptom reduction, and patient satisfaction following interventions provided during eye camps.[7,8]

Overall, the data suggests that the eye camps were able to reach a wide range of individuals within the underserved communities, addressing the eye care needs of different age groups and genders. This highlights the inclusive and

comprehensive approach taken by the eye camps in catering to the vision care requirements of the local population. Together, let us embark on this journey of discovery, as we witness the extraordinary tales of compassion, resilience, and the pursuit of a brighter vision for all in the underserved areas of North India. In conclusion, outreach eye camps are not only catalysts for change, but also they serve as beacons of hope, inspiring a collective call to action to prioritize comprehensive vision care and promote equity in eye health. These camps have demonstrated their potential to transform lives, restore vision, and empower communities by providing comprehensive eye care services directly to those most in need.

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From the sky to the eye: Fascinating journey of the first intraocular lens



Figure 1: Sir Nicholas Harold Lloyd Ridley



Figure 2: British war pilot Gordon Cleaver who inadvertently inspired Dr. Harold Ridley to invent the first intraocular lens

Perhaps no other event has had such a huge impact on the evolution of Ophthalmology as the implantation of the first intraocular lens by the British ophthalmologist, Sir Harold Ridley [Figure 1], on November 29, 1949.[1] This edition presents the fascinating story that led to the successful implantation of the first intraocular lens, an event that has made a paradigm shift in the way millions of eyes perceive vision.

The era of intraocular lens implantation for cataracts started with the power of an idea, followed by astute observations and relentless pursuit of the idea in the most challenging of times. Harold Ridley, when working as the eye surgeon at St. Thomas Hospital during the Second World War, had the opportunity to examine a young British war pilot, Gordon Cleaver [Figure 2].[2] Machine gun fires had shattered his cockpit canopy, and splinters of perspex had destroyed his one eye and had severely damaged the other. The story goes that it was a chance question by his student, Steve Parry, that aroused in Ridley the inspiration toward an invention that was to mark the century. Steve Parry on seeing his first cataract removal surgery exclaimed, "'It's a pity you can't replace the cataract with a clear lens!"[1] Ridley began to ponder over this and observed that even when the cockpit pieces of perspex were inside the eye of the pilot Gordon Cleaver as an intraocular foreign body, they did not elicit any untoward reaction inside his eye. Therefore, would it be possible to use the same material to replace the human crystalline lens following cataract surgery? After the war was over, Ridley was toying with this idea and persuaded his partner

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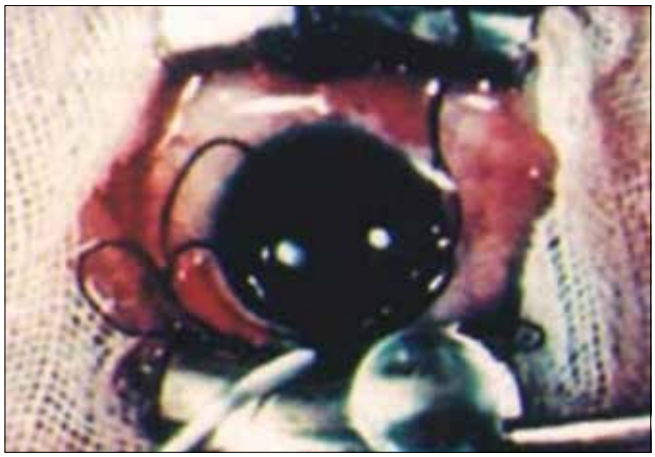


Figure 3: Photograph extracted from an original video tape of Ridley's eighth IOL implantation, performed at St. Thomas' Hospital, May 8, 1951, showing the intraocular lens being inserted through extracapsular cataract extraction incision. This patient had 20/20 vision postoperatively

John Pike, the senior optical scientist at Rayner Intraocular lenses, to design the first implantable intraocular lens.

On November 29, 1949, Ridley performed the first intraocular lens implantation [Figure 3] on a 42-year-old hospital nurse, Elisabeth Atwood, who volunteered for the procedure at St. Thomas Hospital, London,[3] with an intraocular lens made of the material ICI perspex CQ and this event marked a new era in the history of Ophthalmology.[4]

Harold Ridley was born on July 10, 1906, as the eldest son of a naval surgeon who specialized in Ophthalmology at Kibworth Harcourt, Leicestershire, England. He studied at Charter-house school and then Natural Sciences at Pembroke College, Cambridge, and qualified as a medical doctor from St. Thomas Hospital Medical School, London. He decided to specialize in Ophthalmology and became a consultant at Moorfields Eye Hospital, London, in 1938.

He was commissioned in the Second World War as a Major in the Royal Army Medical Corps and was posted in Ghana in 1942, where he made important observations regarding Onchocerciasis. Before going to Africa, Ridley had made observations about the inert nature of perspex inside the eyes of pilots injured during air strikes. He was debating with the idea of an 'inert' lens which could replace the cataractous lens and his observations on Gordon Cleaver's progress and the observations on many other pilots who had sustained perspex foreign bodies during air strikes, which threw some light on a rather dark path.

Ridley envisioned about an intraocular refractive device at a time when the accepted mode of treating cataracts was to remove the cataractous lens and to provide aphakic correction in the form of spectacles. Fearing harsh criticism from the conservative medical professionals, Ridley had kept his work under cover and had repeated the process a few times in 18 months before the news leaked out. He was faced with ridicule, rejection, and professional spite for placing a 'foreign body' inside the eye. There was severe opposition to inserting a new foreign body into the eye, and the medical fraternity viewed it as a dangerous, unnatural, and technically impossible idea.[5]

However, Ridley had support and commanded respect from some progressive and brilliant young ophthalmologists, including Peter Choyce, Eric Arnott, Neil Dallas, and Michael Roper-Hall, in Europe, the United States, and the Soviet Union, who carried forward the idea of intraocular lens implantation. By 1970s, cataract surgery with intraocular lens had been established as a routine and safe treatment or restoring vision after cataract surgery. Since then, intraocular lenses have changed the way in which millions of people see the world and have been an area with continuous evolution in fine tuning of the design. Today, cataract surgery with intraocular lens implantation is the most frequent surgery with widespread economic implications including improvement in quality of lives of the patients and a billion-dollar industry supporting its manufacturing and refinement.

Apart from the invention of intraocular lens, Ridley showed amazing foresight, creativity, and innovation in many other areas. He was a pioneer researcher in the field of Onchocerciasis[6] and ocular involvement in leprosy and the first to televise eye surgeries and to advocate the technique of telediagnosis and to devise a system of posterior segment examination using electronic devices.[7]

However, much later, his contributions were eventually recognized by the establishment with the glory it truly deserved. In 1986, 3 years after his 80th birthday, Ridley was elected to the Fellowship of the Royal Society.[5] The following year, he was awarded the Gullstrand Medal by the Swedish Society of Medicine[5] and he was knighted in 2000.

In the 1990s, he underwent successful bilateral intraocular lens implantation at St Thomas's Hospital by the surgeon Michael Falcon, after which he reflected that "I am the only man to have invented his own operation!"

The story came to a full circle when Gordon Cleaver, the war pilot who was responsible for inadvertently inspiring the journey toward the first intraocular lens, underwent cataract surgery with intraocular lens implantation in his only eye in 1991 and died in 1997, with good eye sight.

Sir Nicholas Harold Lloyd Ridley, whose astute observations and the courage to follow his idea relentlessly despite horrendous opposition, paved the way to the highly specialized cataract surgeries today, died on May 25, 2001, at the ripe age of 95. His wonderful invention still continues to help millions of people across the globe to see the world with astounding clarity.

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Conflicts of interest

There are no conflicts of interest.

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Case Report

Seeing double after sinus surgery

ABSTRACT

A 40-year-old gentleman came to us with protrusion, redness, and mild pain in right eye immediately after undergoing functional endoscopic sinus surgery. He complained of constant horizontal diplopia. He had large angle right eye exotropia (modified krimsy = 50 PD exotropia) with -4 limitation of adduction with palpebral fissure widening on adduction, features suggestive of transection/avulsion of right medial rectus (MR). Magnetic resonance imaging of orbits confirmed the clinical findings. MR exploration was done. Intraoperatively, MR was transected about 15mm from the insertion. A challenging attempt was made to bring back the piecemeal posterior edge and reattach to the anterior edge with hang back sutures. Injection Botulinum toxin was given to right lateral rectus. Diplopia persisted after primary repair and patient was given prism glasses. Eventually a vertical rectus muscle transposition to MR with foster augmentation was done along with injection Botulinum toxin to lateral rectus. Four years postoperatively, patient maintained good alignment in primary position and is diplopia-free in straight gaze for distance and near. Timely exploration with staged squint correction provided acceptable functional outcome to our patient.

Keywords: Diplopia, functional endoscopic sinus surgery, medial rectus transection

INTRODUCTION

Functional endoscopic sinus surgery (FESS) is a minimally invasive procedure to restore sinus ventilation and is the mainstay of treatment for paranasal sinusitis refractory to medical management. The inherent risk of this surgery is injury to the thin medial orbital wall (Lamina papyracea) and damage to the adjacent structures like medial rectus (MR) muscle, orbital fat, nasolacrimal duct, and even the optic nerve. We report a case with complaint of horizontal diplopia following FESS and its management through staged surgeries.

CASE REPORT

A 40-year-old gentleman had right-sided headache and nose block for two years. He consulted an otorhinolaryngologist and was told to have a polyp in his right nose along with right-sided maxillary sinusitis. He underwent FESS as advised. Immediate postoperatively, he started having redness and pain in right eye associated with double vision. He was referred to our hospital for detailed evaluation and management.

On examination, his unaided visual acuity in right eye was 6/9. Pupils were brisk. Right eye examination showed a large angle exotropia with subconjunctival hemorrhage medially [Figure 1]. Anterior chamber and fundus examination were normal. Left eye had an unaided visual acuity of 6/6 and was normal in all aspects. Colour vision was normal in both eyes. In right eye, there was -4 limitation of adduction not crossing the midline with palpebral fissure widening on adduction [Figure 2]. Abduction was full. Modified krimsky was 50PD of exotropia. Patient also had right eye proptosis with proptometry values of 21 mm in right eye and 17 mm in left eye by Hertel's exophthalmometer.

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History and clinical features were suggestive of avulsion or transection of right eye medial rectus during FESS. Magnetic resonance imaging of orbit was advised, which showed a defect in the medial orbital wall and confirmed the transection of medial rectus in right eye [Figure 3]. Patient was admitted, started on intravenous antibiotics and steroids, followed by a course of oral steroids in tapering dose. Exploration of medial rectus and repair was planned under the guidance of an orbital surgeon. Intraoperatively, medial rectus was totally transected 15 mm from insertion and redirected to posterior orbit. A challenging attempt was made to bring back the piecemeal posterior edge and reattached to anterior edge with hang-back sutures [Figure 4]. Botulinum injection was given to right lateral rectus. But diplopia persisted after primary reattachment and repair. Patient was given prism glasses. Eventually a vertical rectus muscle transposition with foster augmentation to medial rectus was done along with Botulinum injection to lateral rectus.

On first postoperative day, patient was orthophoric [Figure 5]. He had a right eye ptosis for a small duration due to Botulinum injection which recovered in a month. Four years postoperatively, patient has good alignment in primary position with adduction improved in right eye (from -4 to -2) and is diplopia free in primary gaze for distance and near [Figure 6].

DISCUSSION

Medial wall is the thinnest wall of the orbit which is also known as lamina papyracea. Injury to medial wall can happen during any procedure near the medial side of orbit.[1] FESS is used to re-establish the ventilation and drainage of paranasal sinuses by widening of osteomeatel complex using a guillotine cutter. Compared to conventional sinus surgery, FESS offer advantages of better visualization, improved cosmesis, and reduced postoperative recovery time.

Modern instrumentation in the form of microdebrider allows precise excision and removal of the tissue. However, these same instruments can create complications if the surgeon is not vigilant. While using these powered cutting instruments, surgeons often lack the feel during tissue removal as compared to manual cutting instruments.[2] The procedure has close proximity to medial orbital wall and chances of injury to medial wall, medial rectus muscle, and even the optic nerve is quiet high.[3]

Optic nerve involvement is the most devastating complication of FESS. Optic nerve is vulnerable because it is in direct contact with the sinus wall which is covered by only 0.5 mm of bone and also there is a very thin mucosa separating it from the sinus cavity. Optic nerve injury can occur as a direct injury due to instrumentation or indirect injury due to compression from orbital hemorrhage. Postoperative complications can be minimized by intraoperative monitoring of pupils and dynamic/multipositional magnetic resonance imaging[4] for functional assessment of extraocular muscles.

In 2004, Kim *et al*.[5] published a series of three cases of diplopia after endoscopic sinus surgery. They discussed the computed tomography findings in each case and compared conservative management versus surgical intervention and suggested the early intervention is advisable in indicated cases.

In 2018, Ben Artsi *et al*.[6] published a series of six cases of traumatic medial wall injury after FESS and discussed the results of different surgical options for the same. They concluded that immediate primary reattachment



Figure 1: Clinical photograph showing right eye exotropia with subconjunctival haemorrhage



Figure 2: Nine gaze clinical photograph showing -4 limitation of adduction in right eye

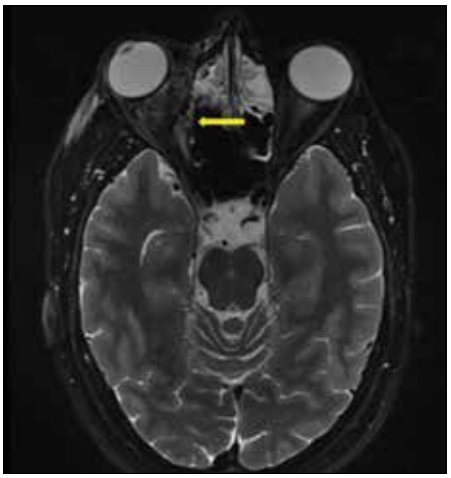


Figure 3: MRI Orbit showing discontinuity of the medial rectus muscle (yellow arrow) in mid-orbit in right eye



Figure 4: Intra-operative photograph showing the medial rectus muscle hooked at the scleral insertion with a Jameson hook and the transected end seen about 15mm from the insertion (yellow arrow)



Figure 5: Clinical photograph of the patient in straight gaze on first post-operative day after primary repair



Figure 6: Clinical photograph of patient after vertical rectus transposition in right eye showing improvement in adduction and good functional alignment

of transected medial rectus muscle with vertical rectus transposition and weakening of lateral rectus either using botox injection or recession gives best results. In our patient, we placed the foster augmentation sutures to improve the resulting adducting force for the patient. Likewise, there are various reports[7-9] stating the importance of early intervention associated with better outcome to the patients.

CONCLUSION

Otorhinolaryngologists and ophthalmologists both should be aware of medial rectus injury being a potential complication of FESS. All steps should be taken to avoid it, but in the event of same, a high index of suspicion, timely exploration, and proper management in a staged fashion with long-term follow-up can provide symptomatic relief, good cosmesis, and acceptable functional outcome to the patient.

Declaration of patient consent

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

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Conflicts of interest

There are no conflicts of interest.

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Case Report

Neurofibromatosis type 1, sarcoidosis, and anterior uveitis: A simple coincidence or coexistence?—An interesting case report

ABSTRACT

Type 1 neurofibromatosis (NF1) is an autosomal dominant multisystem disease caused by a mutation in the neurofibromin 1 gene, which affects tissues derived from the neural crest. Clinically, it is characterized by a spectrum of defects comprising of neural tumors, *cafe-au-lait* spots, intertriginous freckling, and skeletal defects. Sarcoidosis is a systemic disease of unknown etiology defined by the presence of noncaseating granulomatous inflammation and the most commonly involved organs are lungs and lymph nodes. But it can also affect the eyes, skin, heart, and other organs. There are clinical case reports, where various autoimmune diseases are associated with NF1 like—systemic lupus erythematosus (SLE), Hashimoto's thyroiditis, diabetes mellitus, multiple sclerosis, vitiligo, and alopecia. A PubMed search of articles, indexed for MEDLINE using the terms sarcoidosis, anterior uveitis, and neurofibromatosis yielded no such published cases of patients having both sarcoidosis and NF1. We resent a case of neurofibromatosis type 1 in a middle-aged women who presented with anterior uveitis and diagnosed to have underlying pulmonary sarcoidosis. Now it is a rising question, whether neurofibromatosis, sarcoidosis, and anterior uveitis are just a simple coincidence or coexistence?

Keywords: Anterior uveitis, autoimmune diseases, neurofibromatosis type 1, sarcoidosis

INTRODUCTION

Neurofibromatosis type 1 (NF1), also known as Von Recklinghausen's disease, is an autosomal dominant phacomatosis characterized by multiple soft tissue tumors that arise from Schwann cells and fibroblasts.[1] Patients with NF1 carry mutations in the neurofibromin, a gene on chromosome 17, an RasGAP protein that acts to downregulate active Ras signaling. NF1 affects one in 3,500 people and can be inherited or can occur sporadically.[2] NF1 is one of the most common genetic disorders and is not limited to any person's race or sex.[2] NF1 is a multisystemic disease. It may manifest as abnormalities of the nervous tissue, bones, soft tissue, or skin. Although NF1 can affect many different organ systems, effects on the nervous system are a defining feature. In the peripheral nervous system, NF1 is characterized by different Schwann cell tumors, particularly dermal neurofibromas and plexiform neurofibromas (PNF).[3] There are clinical case reports, where various autoimmune diseases are associated with NF1 like—systemic lupus erythematosus (SLE),[4] Hashimoto's thyroiditis,[5] diabetes mellitus,[6] multiple sclerosis,[7] vitiligo, and alopecia.[8] Common symptoms of NF1 include brownish-red spots in the colored part of the eye called Lisch nodules, benign skin tumors called neurofibromas, and larger benign tumors of nerves called plexiform neurofibromas, scoliosis (curvature of the spine), learning disabilities, vision disorders, mental disabilities, multiple

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café au lait spots, and epilepsy.[1-3] NF1 is diagnosed when a person meets the official diagnostic criteria for NF1 that was developed at the National Institutes of Health Consensus Conference in 1987. Based on these criteria, a person who has at least two of the following features is considered to have NF1:[1-3] six or more café-au-lait spots. These spots must be more than 5 mm in diameter in young children and more than 15 mm in diameter after puberty, two or more neurofibromas or one plexiform neurofibroma, Freckling around the armpits or groin, optic glioma, which is a tumor on the optic nerve in the brain that effects vision, two or more Lisch nodules, which are tumors on the iris of the eye, specific bone changes, including sphenoid dysplasia, which is an abnormality of one of the bones forming the skull, or thinning of the long bones, a parent, sibling, or child with NF1.[1-3,5]

Sarcoidosis is a disease characterized by the growth of tiny collections of inflammatory cells (granulomas) in any part of your body—most commonly the lungs and lymph nodes.[9] But it can also affect the eyes, skin, heart, and other organs. The cause of sarcoidosis is unknown, but experts think it results from the body's immune system responding to an unknown substance. This condition mostly affects young adults and a potential abnormal reaction to the body's own proteins (self-proteins) could be responsible for the formation of granulomas.[9] Sarcoidsis is characterized by the presence of noncaseating granulomas and characteristically presents with reticular opacities in the lungs and bilateral hilar lymphadenopathy.[9] Herein, we present a case of classical NF1 and anterior uniilateral uveitis in a middle-aged woman with underlying etiological diagnosis of sarcoidosis. A PubMed search of articles, indexed for MEDLINE using the terms *sarcoidosis, anterior uveitis,* and *neurofibromatosis* yielded no such published cases of patients having both sarcoidosis and NF1. Now it is a rising question, whether neurofibromatosis, sarcoidosis, and anterior uveitis are just a simple coincidence or coexistence?

CASE REPORT

A 45-year-old female patient presented with complaints of pain, redness, photophobia, and drooping of upper eyelid of 7 days onset. She is a homemaker and there is neither any history of ocular trauma nor any preceding history of any fever or general illnesses. There is no associated ocular discharge. She gives a history of on-off respiratory distress and dry cough for past 1–2 months for which she did not seek any medical advice. There is no history of night fever, weight loss, or loss of appetite. She does not give any history of double vision and difficulty in swallowing and deglutition. There was no history of such recurrent illness in the past. She had multiple nodular skin lesions throughout her body with a nodular mass in her left upper lid since long back with mild drooping of upper eyelid. There was no history of any seizure episodes. She overlooked this condition and had an opinion long back. On examination, her distance visual acuity was 6/6 in right eye and 6/12 in the left eye. Intraocular pressure was 14 mmHg in both eyes. There was classical S-shaped moderate ptosis in the left eye with rounded nodular mass in the left upper eyelid with feeling of bag of warm [Figure 1]. It was not tender and not fixed to the underlying structure. On lid evertion, the underlying surface was congested and lacrimal gland was found enlarged with multiple nodular infiltrate [Figure 1]. Lacrimal gland was nontender. There was marked circum ciliary congestions. Cornea was mild lustreless with presence of multiple mutton fat keratic precipitates (KPs) over the endothelium. There was grade two to three anterior chamber reaction with flare [Figure 1]. Her blood pressure was 120/78 mmHg and pulse rate was 77/min. On detailed slit-lamp examination, she had multiple Lisch nodules (flat or mild raised iris surface glistering nodule) in her right eye [Figure 1], whereas no such nodules were seen in the left. There was also lacrimal gland swelling in the right eye [Figure 1]. On dilated fundus examination, there was festooned pupil in the left eye [Figure 1] Detailed fundus evaluation was normal in both the eyes. She had



Figure 1: Classical S-shaped ptosis, eyelid nodular mass, congested eye, festooned pupil along with Lacrimal gland enlargement with multiple nodular infiltrate, multiple mutton fat KPs in left eye, and in the right eye there was Lisch nodules and lacrimal gland swelling

multiple nontender skin nodules suggestive of plexiform neurofibroma with multiple café-au-lait spots (more than six in number) [Figure 2]. Suspecting a case of sarcoidosis chest X-ray, high-resolution computed tomography (HRCT) chest, Montoux, angiotensin-converting enzyme (ACE) estimation, and complete blood count was advised. Chest X-ray was suggestive of sarcoidosis (ground glass appearance) [Figure 3]. On HRCT chest, multiple bilateral hilar lymphadenopathy along with multiple nodular lesions along the peribronchovascular and para septal region in both the lungs were noticed. In addition, multiple fibrotic patches were also noticed in both the lung, suggestive of pulmonary sarcoidosis [Figure 4]. Her ACE level was high = 117 U/L. Blood neutrophil was 62, lymphocyte = 27, monocyte = 06, and eosinophil = 05. Erythrocyte sedimentation rate (ESR) was 13. Serum calcium level was 4.5 mg%, which was lower in side. Her Montoux test was negative. She was prescribed topical prednisolone acetate; one drop six times a day for 7 days along with homatropine 2%; one drop three times a day. Topical steroid was tapered over 6 weeks and homatropine was stopped after 7 days. She was referred to pulmonary physician and was prescribed oral steroid 1 mg/kg/body wt. She responded well with topical steroid. After a month her respiratory symptoms also were improved and she was put on low dose steroid (10 mg daily) for 1 year.



Figure 2: Plexiform neurofibroma with multiple café-au-lait spots

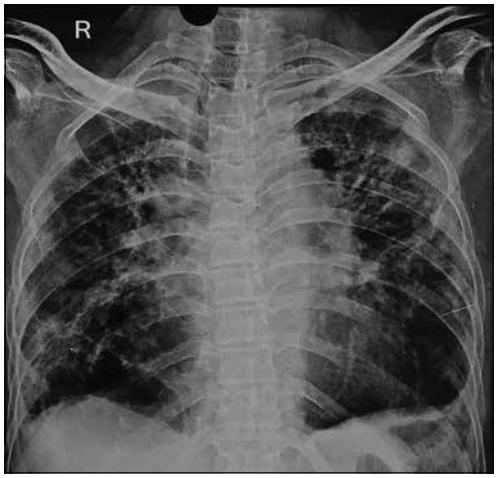


Figure 3: Chest X-ray was suggestive of sarcoidosis [ground-glass appearance]



Figure 4: On HRCT chest, multiple bilateral hilar lymphadenopathy along with multiple nodular lesions along the peribronchovascular and para septal region in both the lungs were noticed. In addition, multiple fibrotic patches were also noticed in both the lung, suggestive of pulmonary sarcoidosis

DISCUSSION

NF1 is a complex multisystem human disorder caused by the mutation of neurofibrin, a gene on chromosome 17. NF1 is an autosomal dominant disorder, and is not limited to any person's race or sex.[1] The disorder affects up to 2% of the general population and is more common in older women and 10 times more frequent in women than in men.[2] Our patient was 34-year-old female presented with left-sided granulomatous uvetis with lacrimal gland swelling with nodular infiltrates in the lacrimal gland. People affected by NF1 also tend to develop varying numbers of neurofibromas, on the covering of the nerves.[3] Our patient had multiple such lesions over forearm, eyelid, and rest of the body. There was classical S-shaped ptosis in the affected eye. NF1 is a hereditary condition commonly associated with multiple café-au-lait spots on the skin. About 10% to 25% of the general population have café-au-lait spots; NF1 is suspected when a person has six or more.[4] Our patient also had multiple café-au-lait spots (more than six in numbers) over forearm and the rest of the body. In our case, there was no such positive family history. Eye findings in NF1 are Lisch nodules in the iris, S-shaped ptosis, optic nerve glioma along the one or both optic nerve or optic chiasma can cause proptosis, squinting, and vision loss.[1-3] Our patient presented with unilateral anterior granulomatus uveitis, lacrimal gland swelling with nodular infiltration, Lisch nodule, and classical S-shaped ptosis.

Sarcoidosis is a disease characterized by the growth of tiny collections of inflammatory cells (granulomas) in any part of your body—most commonly the lungs and lymph nodes. But it can also affect the eyes, skin, heart, and other organs.[9] The cause of sarcoidosis is unknown, but experts think it results from the body's immune system responding to an unknown substance. Symptoms are variable; typically, patients present with a persistent dry cough, fatigue, and shortness of breath. Other symptoms include painful red lumps on the skin, uveitis with the blurring of vision, hoarseness of voice, palpable lymph nodes at multiple sites including armpit, neck, painful swollen joints, hearing loss, seizures, or psychiatric disorders could be observed as part of neurological manifestations.[9] Ocular manifestations are seen in close to 50% of patients, of which the most common clinical feature is uveitis and lacrimal gland swelling.[9] In our patient, it was unilateral granulomatous uveitis with lacrimal gland enlargement with nodular infiltrates. Patient's posterior segment was absolutely normal with vision of 6/12 in the affected eye. On further investigation, there was bilateral hilar lymphadenopathy, reticular opacities along with features of lung fibrosis on HRCT chest. Chest X-ay had a ground-glass appearance. Both the radiological picture was suggestive of sarcoidosis. The patient had high serum level of ACE estimation (117 U/L). Montoux test was negative. So far a PubMed search of articles indexed for MEDLINE using the terms *sarcoidosis, anterior uveitis* and *neurofibromatosis* yielded no such published cases of patients having both sarcoidosis and NF1. The disease sarcoidosis is diagnosed by a constellation of radiographic, clinical, and histopathological findings; it is most often defined by the presence of noncaseating granulomatous inflammation that occurs in the absence of infection, exposures, malignancy, or alternative immune-related disease.[9,10] Lung and thoracic lymph nodes are most often involved. The basis of treatment of sarcoidosis is regulation of[9,10] order to prevent dangerous interference with organ function (as seen in the eye or the heart) and to prevent eventual scarring and fibrosis as seen in the lungs.[10] A number of suggested treatment algorithms have been published, most of which are based upon initiation of corticosteroids in a symptomatic patient with abnormal function or imaging studies, followed by tapering of steroids over a minimum of 1 year.[10] Clinicoradiological findings alone are often insufficient to confirm the diagnosis of sarcoidosis. It needs to be supported by histological evidence showing noncaseating granulomas. This warrants a tissue biopsy which is invasive. A number of immunological biomarkers have been recognized in sarcoidosis. Among all biomarkers, ACE is the most contentious, as it is elevated in sarcoidosis patients and could therefore serve as a sarcoid biomarker.[9,10] In our patient, it was highly radiological suggestive of sarcoidosis.

Pulmonary sarcoidosis is often asymptomatic, nonprogressive disease and requires no treatment, as a majority of patients undergo spontaneous remission.[10] Close monitoring of symptoms, chest radiograph, and pulmonary function is continued at 3- to 6-month intervals should be considered in asymptomatic patients.[9,10] For patients with pulmonary sarcoidosis causing worsening symptoms, stage II to III radiographic findings should be considered for oral glucocorticoids at 0.3 to 0.6 mg/kg for 4 to 6 weeks.[10] In our case, patient was asymptomatic from pulmonary site. If there is no improvement in symptoms, maintenance steroids usually 10 to 20 mg per day should be considered over a period of at least 6 to 8 months.[10]

CONCLUSION

Findings of NF1 and sarcoidosis together in our patient has raised a question whether is it just a coincidence or coexistence? A PubMed search of articles indexed for MEDLINE using the terms *sarcoidosis, anterior uveitis* and *neurofibromatosis* yielded no such published cases of patients having both sarcoidosis and NF1. In medical science, suspect, clinical evaluation, diagnostic tests, and final conclusion of diagnosis and its association with other rare disease is not a new one and we should always work hard to get this answer.

Declaration of patient consent

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

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There are no conflicts of interest.

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Case Report

Things that do not fit well, tend to "stand out" – A case report of extrusion of glaucoma drainage device

ABSTRACT

To report a rare case of extrusion of Aurolab aqueous drainage implant (AADI). A 65-year-old male presented with a history of blunt trauma, loss of vision, and irritation in his left eye (oculus sinister [OS]) for 1 year. Two years prior to the presentation, he was diagnosed with pseudophakic secondary glaucoma, for which he underwent implantation of an Aurolab aqueous drainage implant (AADI) in his left eye. Examination revealed phthisis bulbi and a partially extruded body of the AADI plate and tube (OS). The patient underwent surgical explantation of the glaucoma drainage device (GDD) and, subsequently, ocular prosthetic rehabilitation. Extrusion of GDD is a rare occurrence, and timely intervention is necessary to avoid grave consequences.

Keywords: AADI, extrusion, glaucoma drainage device

INTRODUCTION

Glaucoma drainage devices (GDDs) are commonly used in the management of eyes with refractory glaucomas.[1] Aurolab aqueous drainage implant (AADI; Aurolab, Madurai, India) is a cost-effective version of a non-valved implant based on the Baerveldt model, which has shown good outcomes.[2] Though efficacious, GDD implantation is associated with many serious complications like hypotony, shallow anterior chamber, corneal decompensation, inflammation, and implant-related problems.[3] Tube exposure is one of the common implant-related complications, but plate or GDD extrusion has been rarely reported.[4-6] We intend to report a case of spontaneous extrusion of AADI following blunt trauma.

CASE REPORT

A 65-year-old male presented to our outpatient services with a history of blunt trauma, loss of vision, and irritation in his left eye (oculus sinister [OS]) for 1 year. Two years prior to the presentation, he was diagnosed with pseudophakic secondary glaucoma, for which he underwent implantation of an Aurolab aqueous drainage implant (AADI) in his left eye. On examination, he had a vision of 20/20 in his right eye (oculus dexter [OD]), and OS had no perception of light. Examination revealed a disorganized globe with a shrunken appearance, indicating phthisis bulbi and a partially extruded body of the AADI plate and tube [Figure 1a]. The prognosis was explained to the patient and subsequently planned for surgical explantation of the glaucoma drainage device. Under peribulbar anesthesia [Figure 1b], the remnant fibrotic capsule and adhesions surrounding the body of the AADI plate were carefully excised, and the AADI plate and tube

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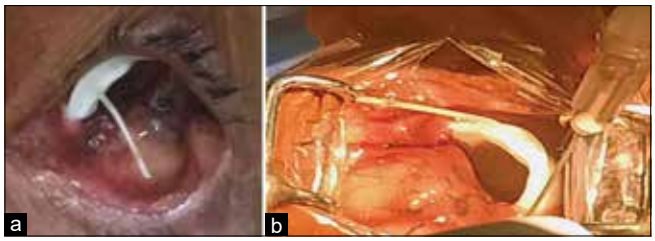


Figure 1: (a) Slit lamp image showing extruded AADI with pthisical globe. (b) Intraoperative image showing removal of the extruded AADI. AADI = Aurolab aqueous drainage implant

were removed in toto. Following this, the patient was treated with topical steroids and antibiotics. On further follow-ups, the patient remained free of symptoms and underwent ocular prosthetic rehabilitation.

DISCUSSION

Both the primary and refractory forms of glaucoma are being treated more frequently using GDD in recent years. With the increase in their use, it becomes essential to identify and manage their complications as well. Although the Tube versus Trabeculectomy Study (TVT) demonstrated that tubes have a lower rate of problems than trabeculectomies, there is still a chance that tube implantation will have unfavorable effects.[7] The commonly reported late postoperative complications following GDD implantation are tube block, exposure, migration, and corneal decompensation. Extrusion of GDD is an extremely rare complication, with only 3 reports involving Ahmed glaucoma valve and PAUL shunt.[4-6] To the best of our knowledge, this is the first report of extrusion of an AADI implant. The reported cases were following retrobulbar hemorrhage,[6] multiple interventions,[5] orbital cellulitis.[4] In our case, it was a sequela following blunt trauma and the resultant Pthisis. The shrinking eyeball and the low scleral rigidity would have contributed to the expulsion. The erosion or extrusion of a GDD poses a serious risk of endophthalmitis, warranting prompt surgical intervention. In one study, 9 out of 542 eyes that received GDD implantation experienced endophthalmitis. Six of the 9 cases had tube erosion.[8] In some cases, when this complication occurs, the GDD simply needs to be removed. With meticulous and timely removal, grave consequences were avoided for our patients. This reiterates the importance of regular follow-up and monitoring, as unforeseen events can happen at any time in the postoperative period.

CONCLUSION

Extrusion of GDD is a rare presentation. Regular follow-up and timely surgical intervention are necessary to avoid serious complications.

Ethical considerations

A written informed consent was obtained from the patient.

Declaration of patient consent

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

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There are no conflicts of interest.

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Case Report

‘PRES’SING problems in pregnancy: A case report

ABSTRACT

Posterior reversible encephalopathy syndrome (PRES) earlier called as reversible posterior leukoencephalopathy syndrome is a clinicoradiologic syndrome of heterogenous etiologies grouped together because of similar clinical and radiologic findings. Patients present with features of headache, confusion or decreased consciousness, visual complaints, and sometimes seizures of acute or subacute onset. The underlying etiology or inciting factors are variable, the most common being hypertension, renal disease, and immunosuppressive drugs. They are diagnosed based on the common radiologic finding of bilateral posterior cerebral white matter edema. Other radiologic findings may also be present. Such patients may present first to an ophthalmologist with complaints of sudden onset of defective vision or double vision. It is a reversible entity if identified early. Hence, high index of suspicion, quick diagnosis, and management are important. Here, we present the case of a 23-year-old primigravida with chronic hypertension who presented with sudden onset of defective vision followed by headache. Visual acuity was only perception of hand movements in both eyes. Ophthalmologic evaluation showed no abnormality to account for the visual loss. Brain imaging showed bilateral parieto-occipital white matter edema consistent with clinical diagnosis of PRES. After treatment of hypertension and supportive measures, the patient’s symptoms recovered, and her vision was 6/6 in both eyes on 1-week follow-up. Follow-up MRI also showed resolution of edema.

Keywords: Edema, hypertension, MRI, posterior reversible encephalopathy syndrome, pregnancy

INTRODUCTION

Posterior reversible encephalopathy syndrome (PRES) also earlier called as reversible posterior leukoencephalopathy syndrome is a clinicoradiologic syndrome of heterogenous etiologies grouped together because of similar clinical and radiologic findings. Patients present with features of headache, confusion or decreased consciousness, visual complaints, and sometimes seizures of acute or subacute onset. The underlying etiology or inciting factors are variable, the most common being hypertension, renal disease, and immunosuppressive drugs. It is usually reversible[1] but with reports of permanent visual loss in untreated cases.[2]

CASE REPORT

A 23-year-old primigravida of gestational age 25 years was referred to ophthalmology casualty with complaints of sudden-onset defective vision in both eyes of one-day duration. It was associated with diffuse headache of moderate severity, not relieved by analgesics. She did not have any other symptoms. Medical history was nonrelevant except for detection of hypertension in the first month of pregnancy, and she was on nifedipine 20 mg twice daily. Other medications included iron, calcium, and folic acid.

On evaluation, blood pressure was 150/100 mm Hg. Other vitals were stable. Higher mental function was also normal. Vision was perception of hand movements in both eyes with accurate projection of rays. External eyes were within normal limits with no relative afferent pupillary defect.

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Dilated fundus examination showed normal disc with grade I hypertensive retinopathy changes [Figure 1].

After ruling out ocular causes of visual loss, a magnetic resonance imaging (MRI) of the brain with orbit was advised. MRI showed hyperintense lesion on T2-weighted images in bilateral parieto-occipital region probably because of white matter edema consistent with PRES [Figure 2]. She was referred back to obstetrics department for control of hypertension and close monitoring. She was managed with addition of labetalol. On follow-up after one week, her vision was 6/6 in both eyes. Follow-up MRI showed complete resolution of white matter edema after 6 weeks.

DISCUSSION

Posterior reversible encephalopathy syndrome (PRES) has been reported in patients as young as 2 years to as old as 90 years and seen more among females than males. Pathophysiology is believed to be disordered cerebral autoregulation and endothelial damage. This can lead to arteriolar dilatation and increased and unregulated cerebral blood flow, leading to breakdown of blood-brain barrier and leakage of fluid into brain parenchyma. The parietal and occipital lobes are most commonly affected, followed by the frontal lobes, the inferior temporo-occipital junction, and the cerebellum.[3] Edema is characteristically seen as bilaterally symmetrical parieto-occipital edema. However, the imaging findings are variable and may occur in other locations such as the frontal lobes, thalami, basal ganglia, and brainstem.[4] Important risk factors include systemic hypertension, immunosuppressive drugs, and cytotoxic drugs such as cyclosporine, tacrolimus, platinum containing drugs, renal disease, and pre-eclampsia, all of which cause endothelial damage and thus lead to cerebral edema as mentioned above.

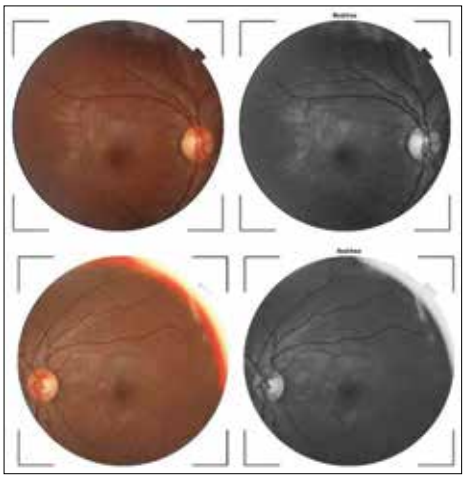


Figure 1: Fundus images of the patient

Clinical features evolve rapidly over hours to days. The patient presents with complaints of headache, which is constant, nonlocalized, and not relieved by analgesics. Altered sensorium may vary from mild somnolence to confusion, agitation to even stupor and coma. Visual complaints include defective vision, hemianopia, quadrantanopia, diplopia, visual neglect, cortical blindness, aura, and visual hallucinations. Seizure can be generalized tonic clonic, focal, or even status epilepticus.[5,6]

On examination, visual acuity may be severely impaired to the extent of no light perception in both eyes. External eye and fundus are usually normal. Hypertensive retinopathy changes may be seen in patients with chronic hypertension. Papilledema may be present. Sometimes, retinal detachment may coexist. Hypertension may not be a finding at presentation because it is the rapid fluctuation in blood pressure that is more contributory than the absolute blood pressure itself, and the fluctuation may have been hours to days before. PRES can also occur in patients with other risk factors even with no history of hypertension. Differential diagnosis includes hypertensive retinopathy, hypertensive encephalopathy,

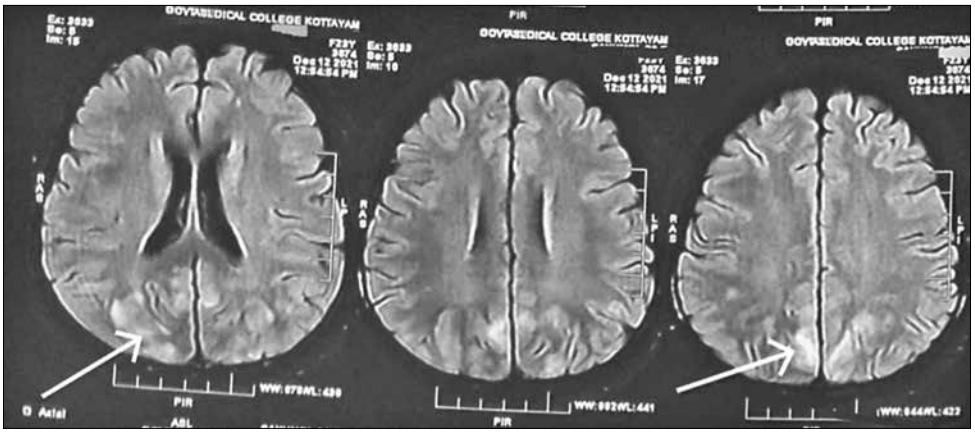


Figure 2: T2-weighted MRIs showing white mater edema in the occipital region denoted by white arrows

reversible cerebral vasoconstriction syndrome, posterior circulation stroke, demyelinating disorder, and vasculitis.[7]

After thorough examination to rule out ocular causes of defective vision, neuroimaging must be done. Neuronal current MRI best depicts the changes. Bilateral white matter edema in posterior cerebral hemispheres, particularly parieto-occipital region, is seen as hyperintense signals in T2-weighted images and hypointense signals in T1-weighted images. Magnetic resonance angiograph and venographs are usually normal. Follow-up imaging is mandatory and is necessary to look for resolution with treatment in PRES. With reduction of blood pressure with parenteral antihypertensives, control of seizure, discontinuation of inciting drugs, and other supportive measures on time, the condition is completely reversible in most patients.[8,9]

As vision loss is an initial symptom, ophthalmologist may be the first physician to see the patient and diagnose the condition. Hence, an ophthalmologist must be aware of PRES and include it in the differential diagnosis of acute or subacute vision loss in patients with risk factors. The potential for complete reversibility of vision loss makes early diagnosis and treatment all the more important, and the outcomes are extremely satisfying. Associated findings such as hypertensive retinopathy and retinal detachment must be addressed, and the patient should be referred to a neurology or obstetrics OPD, in case of preeclampsia, eclampsia, for further management. Although most patients recover PRES, it is not always reversible and may be associated with considerable morbidity and even mortality.[10] It is concluded that with high index of suspicion, early diagnosis, and prompt treatment of PRES, visual loss in PRES can be reversed without any permanent damage.

Declaration of patient consent

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

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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Case Report

Double crisis: A case of late bilateral consecutive spontaneous intra-ocular lens dislocation in the absence of identifiable systemic disease

ABSTRACT

A 56-year-old gentleman presented with decreased vision in the right eye (OD) since 1 month and in the left eye (OS) since 1 year. He denied history of trauma. Fifteen years ago, he had undergone uneventful phaco-emulsification in both eyes (OU). Slit-lamp examination showed OU aphakia. Fundus examination revealed an OU posteriorly dislocated intra-ocular lens (IOL). He underwent vitrectomy with IOL removal, followed by scleral fixated IOL placement, initially in OS, followed by OD after 1 week. At 6 weeks, he had 20/30 vision in OD and 20/40 in OS.

Keywords: Dislocated intra-ocular lens, phaco-emusification, scleral fixated IOL

INTRODUCTION

Late intra-ocular lens (IOL) dislocation is a known complication of cataract surgery, the reported incidence being around 0.032–0.28%.[1] It usually occurs due to progressive zonular weakness.[1] Most of the reports on late IOL dislocation describe unilateral cases of IOL dislocation; however, bilateral late dislocation of IOL has also been reported.[2-5] Bilateral dislocation of IOL is usually associated with a systemic condition such as pseudo-exfoliation,[2] retinitis pigmentosa,[2] gyrate atrophy,[4] uveitis,[5] and so on. We report a rare case of late bilateral spontaneous posterior dislocation of IOL in the absence of any identifiable systemic disease.

CASE REPORT

A 56-year-old gentleman presented to the clinic with complaints of decreased vision in OD since 1 month and in OS since 1 year. The onset of the decrease in vision in OU was sudden, painless, and non-progressive. The patient did not consult anywhere for OS as the symptoms occurred during the coronavirus disease (COVID) pandemic and he could manage his daily activities with the good vision in OD. He denied history of any trauma. Fifteen years ago, he had undergone uneventful phaco-emulsification with +21D polymethylmethacrylate IOL placed in the bag, initially in OD, followed by OS after a gap of 11 months. Post-operatively, he had 20/20 vision in OU and was asymptomatic till date. He was a known case of hypertension on treatment since 15 years. On general examination, there was no abnormality noted in his stature/skeletal structure. Ocular examination revealed the unaided visual acuity for distance as 20/400 OU. Slit-lamp examination revealed OU vitreous in the anterior chamber, sectoral iris atrophic patches, and aphakia. Fundus examination revealed OU 'in-the-bag' posteriorly dislocated IOL; the rest of the retina was unremarkable. The patient underwent three port pars plana vitrectomy with IOL removal [Figure 1], followed by scleral fixated IOL

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placement [Figure 2], initially in the left eye, followed by the right eye after 1 week. The surgery and the post-operative period were uneventful. At 6 weeks follow-up, the patient had 20/30 vision in OD and 20/40 in OS.

DISCUSSION

The first description of dislocation of the IOL capsule complex dates back to 1993 and has been reported with increasing incidence in recent years.[6] The mean time interval between cataract surgery and surgery for IOL dislocation has been reported to be 6.9–8.5 years.[6] The final common cause for late IOL dislocation is zonular instability, which may be exacerbated by the presence of capsular contraction.[6] Bilateral dislocation of IOL has been described in systemic conditions which may contribute to zonular weakness.[2-5] Though posterior dislocation is more common, bilateral anterior dislocation of IOL with a capsular bag has been reported in a patient with pseudo-exfoliation.[7] Certain connective tissue disorders such as Marfan syndrome, Ehlers-Danlos syndrome, and Weill-Marchesani syndrome are known to be associated with increased risk of lens dislocation;[1] however, our patient did not have systemic features of the same. Spontaneous bilateral posterior dislocation of the cataractous lens in the absence of any risk factor has been described in the literature.[8] To the best of our knowledge, this is the first case of bilateral consecutive spontaneous dislocation of IOL in the absence of any identifiable systemic or ocular risk factors.

Our case had iris atrophic patches in both eyes [Figures 3 and 4], which were present prior to cataract surgery. However, there was no evidence of pseudo-exfoliative material over the pupillary margin or lens. The intra-ocular pressure was normal, and the optic nerve head was healthy. Pupils were well dilating, and there was no documented intra-operative complication during cataract surgery. The distribution of iris atrophic patches was predominantly sectoral and not typical of the iris atrophy near the pupillary margin seen in pseudo-exfoliation. There was no history of trauma, heterochromia, or intra-ocular inflammation. Whether the presence of the iris atrophic patches was an associative or causative factor for the IOL dislocation is uncertain. Subjecting the dislocated IOL for histopathology may help in detecting sub-clinical pseudo-exfoliation;[9] however, it could not be done in this case due to financial constraints. Unilateral late spontaneous posterior IOL dislocation in the absence of risk factors has been reported,[10] and the probable cause hypothesized is "Capsular contraction syndrome". This entity has increased in incidence after the introduction of continuous curvilinear capsulorhexis,[10] which on one hand has made "in the bag" IOL fixation more secure but on the other hand also induces some amount of capsular fibrosis.[6] Capsular contraction is more common in cases with small capsulorhexis as compared to those with large ones.[10] The use of high vacuum, irrigation, and aspiration or the unbalance between centripetal and centrifugal forces during phaco-emulsification may contribute to zonular weakening,[10] which may slowly progress over years, ultimately leading to late IOL dislocation.

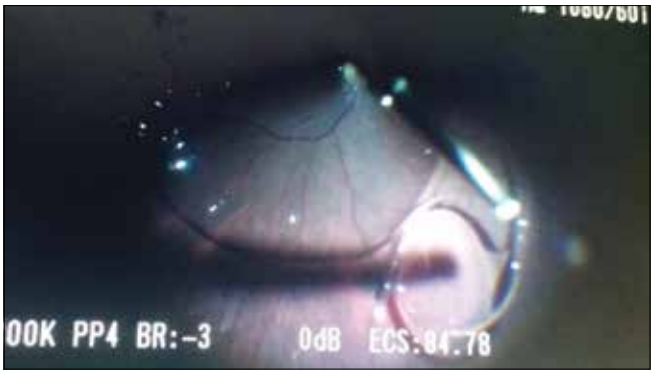


Figure 1: Removal of dislocated IOL



Figure 2: Scleral fixated IOL placement

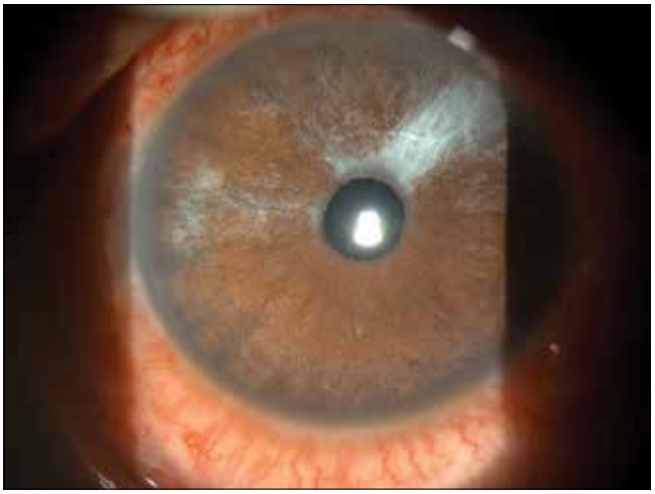


Figure 3: Right eye showing iris atrophic patch

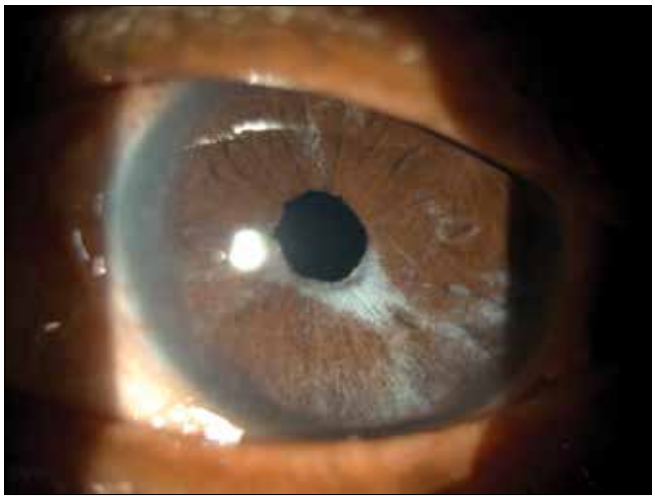


Figure 4: Left eye showing iris atrophic patch

The management of dislocated IOL can be broadly classified into IOL repositioning or IOL exchange. There is no conclusive evidence that one technique is superior over the other and the choice of surgery is best individualized for each case. The common options to replace the dislocated IOLs are angle-supported anterior chamber IOL, sulcus repositioning, scleral fixated IOL, and posterior chamber iris sutured IOL.[10] We chose to perform IOL explantation, followed by a scleral fixated IOL placement. The patient had an uneventful post-operative period and good visual recovery.

To summarize, late IOL dislocation may occur in the absence of identifiable risk factors. The fellow eye must be carefully monitored at regular intervals. Capsular contraction syndrome may be responsible for late IOL dislocation in those cases with no ocular/systemic risk factors. Good visual outcomes can be obtained with timely and appropriate intervention.

Declaration of patient consent

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

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Nil.

Conflicts of interest

There are no conflicts of interest.

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Case Report

A wriggle underneath the conjunctiva

ABSTRACT

The Indian subcontinent is well known for various parasitic infections, given the myriad of favourable conditions which enable the parasite to complete its life cycle. However, multiple textbooks in ophthalmology do not provide adequate amount of information about the same. This manuscript is an attempt to bridge this gap and enable ophthalmologists to treat a common but yet underreported clinical scenario.

Keywords: Dirofilaria, parasitology, worm

INTRODUCTION

The Indian subcontinent is very well known for a multitude of parasitic infections, given the ready availability of vectors and suitable environment for the completion of the parasite's life cycle. However, many textbooks in ophthalmology fail to shed light on some commonly encountered parasitic infections which are often seen in a rural setting, where a comprehensive ophthalmologist is more likely to practice. This case report is an attempt to fill this existing gap—that is, to enable a primary-level ophthalmologist to better understand and manage this clinical condition.

CASE REPORT

A 55-year-old lady, native to Amaravila in Kerala, presented with complaint of redness—which would wax and wane—in her right eye (RE) for the past 2 weeks with accompanying foreign body sensation. Slit lamp examination of the affected eye showed a diffuse episcleral congestion involving the temporal bulbar conjunctiva within the interpalpebral zone. Tenderness was absent over the involved area, but a repeat examination revealed a transparent, coil-like structure wriggling beneath the area of interest [Figure 1].

A clinical diagnosis of a subconjunctival worm was made, and a plan to surgically extract the worm was chalked out. She consented to the procedure, and under the effect of topical 0.5% proparacaine, a conjunctival nick was created over the predicted location of the worm. In its complete entirety, a long, white-colored, tubular worm was glided out of the subconjunctival plane with a forceps and promptly sent for a microbiological evaluation [Figure 2]. Under the microscope, the wet mount preparation of the worm showed a rounded end [Figure 3] along with thick cuticle and longitudinal striations [Figure 4]. The microbiologist identified the worm to be a female *Dirofilaria repens* [Figure 5].

The patient was put on a 1-week course of 0.5% moxifloxacin. She did not report for follow-up.

DISCUSSION

Let's dissect this case a little more and attempt to improve our comprehension about *Dirofilaria*.

In India, a majority of reporting about *Dirofilaria* has been from Kerala, coastal Karnataka, and Maharashtra, while only minimal cases are reported from North India.[1] Mosquitoes

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Figure 1: Slit lamp photograph showing a coiled S-shaped translucent worm within the encircled zone

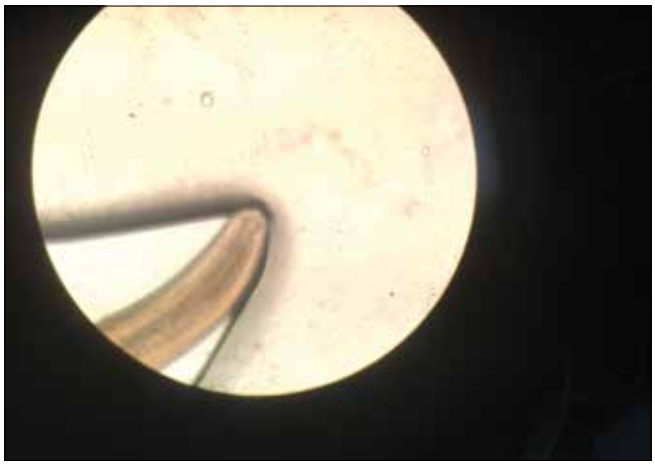


Figure 3: Tapering end of worm seen under the microscope

of the *Culex* and *Anopheles* breeds are involved in the transmission of this disease.[2] Our patient is a resident of a coastal area called Amaravila, which itself heightens the possibility of *Dirofilaria*. Enquiry about a patient's geography and travel history is of utmost importance as was noticed in a case of subconjunctival *Dirofilaria* being reported in a 43-year-old woman at the Skane University Hospital, Sweden,[3] who had a history of travel to Pondicherry, India.

Dirofilaria is known to house itself at multiple sites in the human body within a subcutaneous/submucous plane,[4] thereby undermining the need for the treating clinician to have an insight about worms, especially when the clinical presentation also shows features of motility attached to it.

The treatment advised is a simple extraction of the worms with there being no definitive role for systemic antihelminthic drugs. The rationale for the same can be exemplified by exploring the life cycle of *Dirofilaria*.



Figure 2: Photograph showing the entire worm

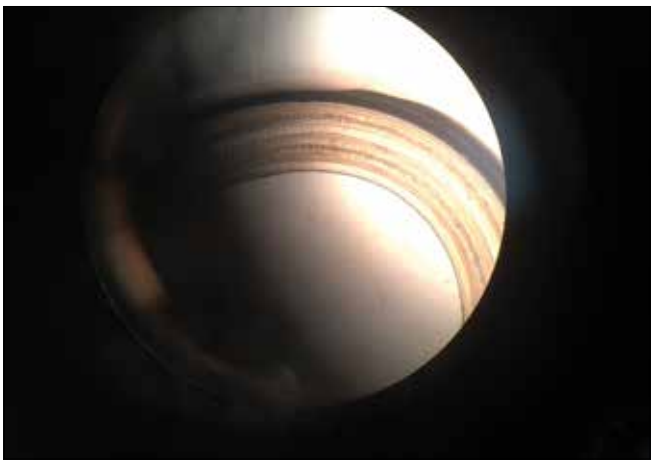


Figure 4: Longitudinal striations and thick cuticle of worm seen under the microscope

Dirofilaria is a zoonotic disease affecting animals—both domestic and wild, chiefly dogs and cats. Humans are only aberrant, accidental dead-end hosts. The microfilaria—which refers to the larval stage of the worms—is picked up by the mosquitoes from the infected animals and introduced into the human body during a blood meal of the mosquito. The microfilaria immediately enters a subcutaneous plane and grows into adult worms. Fortunately, the cycle ends there as these worms are incapable of sexually reproducing in humans, and hence, the only treatment is surgical removal of the worm.

However, with recent reports of human filarial worm *Brugia malayi* now being reported in dogs in Kerala,[5] it is possible that *Dirofilaria* may attain the ability to further propagate its life cycle in man and thereby making it even more easier for the disease transmission as there is neither a dearth for man nor mosquitoes.

In this context, it might be prudent to carry out two more investigations in such patients—eosinophil count and peripheral blood smear—which I had failed to do in this patient due to lack of insight. A high eosinophil count and/

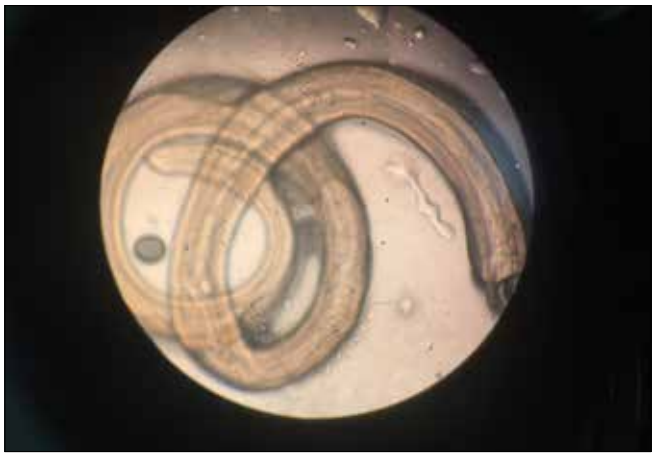


Figure 5: View of the worm under the compound light microscope (wet mount)

or the presence of microfilaria in the smear report would not only necessitate the need for systemic antihelminthic drugs, but a public health expert would also need to be notified on a remote possibility of *Dirofilaria* now having attained the capability of reproducing sexually in humans.

Declaration of patient consent

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

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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Case Report

From sunset to sunrise

ABSTRACT

A 38-year-old male patient presented with blunt trauma in right eye (RE), 3 days back. On examination of RE, the best corrected visual acuity was light perception and intra-ocular pressure (IOP) was 42 mmHg by applanation. Anterior segment examination showed dislocated lens in anterior vitreous cavity. Ultrasound sonography showed no evidence of vitreous hemorrhage or retinal detachment. Maximum medical anti-glaucoma therapy was started. After 1 week, due to uncontrolled IOP and dislocated lens, pars plana vitrectomy with lensectomy with scleral fixated intra-ocular lens implantation was done. On 2-week follow-up, IOP was 18 mmHg on monotherapy, with stable lens and retina.

Keywords: Closed globe injury, dislocated lens, scleral fixated intra-ocular lens

INTRODUCTION

The lifetime prevalence of ocular trauma is estimated to be 19.8% with a 5-year incidence of 1.6%.[1] A high rate of ocular trauma occurs in young males due to their outdoor-related work activities. Even minor ocular injuries may result in significant economic burden to families. Cataracts or glaucoma can result from blunt or penetrating ocular trauma. Post-traumatic glaucoma is usually caused by an increase in intra-ocular pressure (IOP) by mechanisms such as inflammatory cells blocking the trabecular meshwork, leading to trabeculitis and angle recession. Early or delayed elevation in IOP can cause damage to the trabecular meshwork in the injured eyes. This can further lead to glaucomatous changes in the optic nerve head. Early detection and timely management are key to preventing further complications.[2] Lifetime regular follow-up is needed for the affected individuals to rule out any traumatic-related changes in the optic nerve head (traumatic optic neuropathy and glaucomatous changes) and the peripheral retina (retinal tears, dialysis, and detachment).

CASE REPORT

A 38-year-old male patient presented with defective vision in (RE > LE) both eyes. The patient had a history of blunt trauma 3 days ago in RE. He was diagnosed with type-2 diabetes 10 years back. The best corrected visual acuity in the right eye (RE) was perception of light, and that in the left eye (LE) was hand movements. The IOP by Goldmann applanation tonometry was 42 mm Hg in RE and 14 mmHg in LE. On anterior segment examination, the right eye showed corneal haze with a dislocated lens in the anterior vitreous cavity and pseudo-exfoliative material in the pupillary area [Figure 1a]. The patient had a contusion (zone 1, closed globe injury as per the Birmingham Eye Trauma Terminology classification system) due to an accidental hit by a stick at his workplace. The left eye had mature cataract [Figure 1b]. Injectable and oral carbonic anhydrase inhibitors in the form of mannitol (20% w/v) and tablet acetazolamide 500 mg were given. Maximum medical topical anti-glaucoma medication was started (a combination of dorzolamide (2%)

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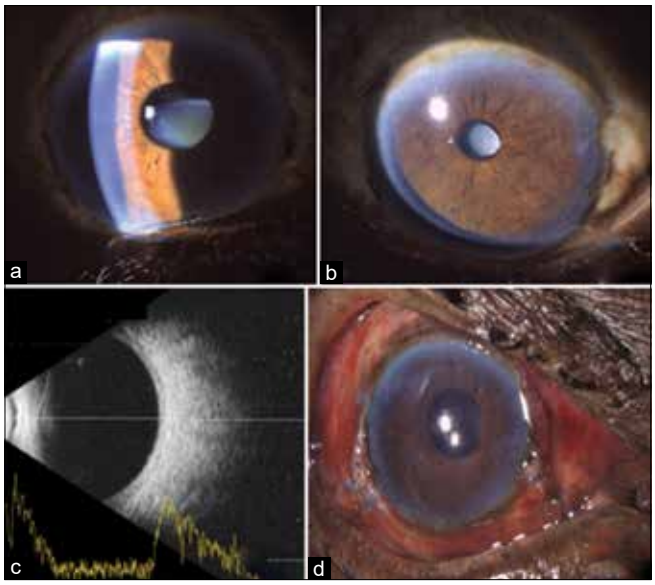


Figure 1: (a) – Slit lamp image of RE showing dislocated lens in anterior vitreous cavity with pseudo-exfoliative material in pupillary area. (b) – Diffuse slit lamp image of LE showing mature cataract. (c) – Normal ultrasonography of RE. (d) – Diffuse slit lamp image showing scleral fixated intraocular lens

+ timolol (0.5%) and brimonidine (0.1%)). Ultrasonography was normal in RE [Figure 1c]. After 1 week, IOP was 36 mmHg and considering the dislocated status of the crystalline lens in the anterior vitreous cavity, pars plana vitrectomy with lensectomy along with implantation of the scleral fixated intra-ocular lens was done [Figure 1d]. On a 2-week follow-up, the visual axis was clear, the best corrected visual acuity was 6/18, and IOP was 18 mmHg with monotherapy (Timolol (0.5%)). Dilated fundus examination in RE revealed a cup: disc ratio of 0.6 and a stable peripheral retina.

DISCUSSION

Blunt injury results in sudden compressive deformation of the eye. The cornea and anterior sclera are displaced posteriorly, and there is a compensatory expansion of the globe in the equatorial direction.[3] Campbell[4] has graphically described seven rings, or circles, of tissue anterior to the equator of the globe that suddenly expand with blunt trauma impact. The global annual incidence of ocular trauma is around 55 million, of which 750,000 cases require hospital admission every year.[5] The pathophysiology of traumatic glaucoma is multi-factorial. Poor initial visual acuity, advancing age, lens injury, angle recession, higher initial IOP, and hyphemas are highly predictive factors for developing glaucoma following ocular injury.[6] A myriad of conditions which include ocular trauma, inherent zonular weakness, and complicated cataract surgery can compromise anterior and posterior capsular integrity.

In our patient, the rise in IOP can be due to secondary inflammation following blunt trauma. Apart from that, the pseudo-exfoliative material from the pupillary margin can block the trabecular meshwork and cause an increase in IOP after a dislocated lens following blunt trauma.

In our patient, zonular laxity due to the presence of pseudo-exfoliation has contributed to the lens dislocation in the anterior vitreous cavity.

The reported incidence of phacodonesis and/or lens subluxation ranges from 8.4% to 10% in patients with pseudo-exfoliation syndrome.[7,8] In our patient, considering the status of the dislocated lens (sunset) [Figure 1a], pars plana vitrectomy along with lensectomy was done. A scleral fixated intraocular lens (Sunrise) [Figure 1d] was implanted, and visual acuity of 6/18 with an IOP of 18 mmHg was maintained on single anti-glaucoma medication (Timolol 0.5%).

CONCLUSION

Effective IOP control and early surgical intervention are the key to maintain visual functions and save the eye.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

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Case Series

Case Series: EDTA chelation for band keratopathy

ABSTRACT

The purpose of this study was to report a case series of visually significant calcific band keratopathy (CBK) associated with chronic uveitis and systemic hypercalcemia, which markedly decreased in severity after dipotassium ethylenediaminetetraacetic acid (K2 EDTA) chelation. The results included six eyes (five patients) with a moderate-to-severe grade of CBK who underwent K2 EDTA chelation. Of five patients, two had chronic uveitis, two had absolute glaucoma, and one had old corneal opacity. After K2 EDTA chelation therapy, the CBK lessened in severity with clearing of the central cornea and improvement in visual acuity. It was concluded that chelation of calcium with topical EDTA is a safe and effective treatment for band keratopathy. Because of the difficulty in acquiring disodium EDTA and the easy availability of K2 EDTA in medical facilities, K2 EDTA chelation is preferred over disodium (Na2) EDTA chelation. Other than easy availability, K2 is also preferred because the preparation of the final EDTA dosage is performed under relatively sterile conditions.

Keywords: Band keratopathy, chelation, chronic uveitis, EDTA

INTRODUCTION

Calcified band keratopathy (CBK) was first described by Dixon in 1848 as a degenerative condition of the cornea, characterized by the deposition of grey–white opacities in the superficial corneal layers, most commonly in the exposed interpalpebral region.[1,2] It can be classified as primary or secondary, with a variety of predisposing systemic and ocular conditions, including renal failure, elevated serum calcium levels, chronic ocular inflammation, corneal ulcers, alkali chemical burns, and a compromised ocular surface due to adverse reactions to preservatives or buffers in ocular drops, particularly in patients on lifelong therapy.[1-5] Familial calcified band keratopathy has also been reported.[6]

In most cases, band keratopathy is superficial. Histological analysis of affected corneas has revealed a selective, fine granular calcification of Bowman's membrane.[1,2]

In the early stages of the condition, the patients remain asymptomatic; however, extension into the visual axis can result in glare and reduced visual acuity. A breakdown of the ocular surface can result in painful recurrent corneal erosion-like symptoms and predispose to corneal ulceration.

Earlier mechanical debridement was performed to remove calcium deposits from the superficial cornea.[7] The most popular current method involves the use of a metal ion chelator known as ethylenediaminetetraacetic acid (EDTA).

EDTA for ophthalmic use was originally derived from Na2 EDTA, but it has lost approval from the Food and Drug Administration (FDA) and now can only be obtained at compounding pharmacies. Lee *et al.*[8] proposed a novel and useful method of producing EDTA by dissolving the K2 EDTA lining found in blood collection tubes.

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In our study too, we used the same technique of obtaining K2 EDTA from vacutainer tube and using them to treat moderate-to-severe CBK in six eyes of five patients [Figure 1].

MATERIAL AND METHODS

The selected patients underwent the following examinations: visual acuity (distant or near), intraocular pressure, slit-lamp examination, fundoscopy, and B scan. Systemic investigations like renal function tests and serum Ca/PO4 levels.

Firstly, K2 EDTA was prepared by injecting 0.3 ml of sterile water into a 2-ml K2 EDTA vacutainer blood collection tube, containing 3.6 mg of K2 EDTA coated inside the tube, and this solution was swirled to dissolve it. This solution was then transferred from tube to tube using five tubes to prepare an end concentration of 6% [Figure 2].

Secondly, patient preparation was performed. The patient's eyes were anesthetized by topical proparacaine hydrochloride (0.5%) and painted with 5% povidone–iodine and draped, and a speculum was applied. The epithelium was debrided with 20% ethanol. A copious wash was performed to remove any residual alcohol.

A sterile cotton swab dipped in the final solution containing K2 EDTA was rubbed and kept over the CBK for 5 minutes. It was removed once it adequately infiltrated the space and dissolved the calcium impaction [Figure 2].

An iris repository was used to scrape further, a normal saline wash was performed, and a bandage contact lens (BCL) was placed.

Patients were advised topical moxifloxacin (0.5%), fluorometholone (0.1%), and lubricating eye drops.

After 5–7 days, BCL was removed and patients were further called for follow-up after 1 week, 1 month, and 3 months.

CASE SERIES

The results included six eyes (five patients) with a moderate-to-severe grade of CBK who underwent K2 EDTA chelation. Of five patients, two had chronic uveitis, two had absolute glaucoma, and one had old corneal opacity [Table 1].

RESULTS

After K2 EDTA chelation therapy, the CBK lessened in severity with clearing of the central cornea and improvement in visual acuity with no postoperative (post–op) complications [Figure 3].

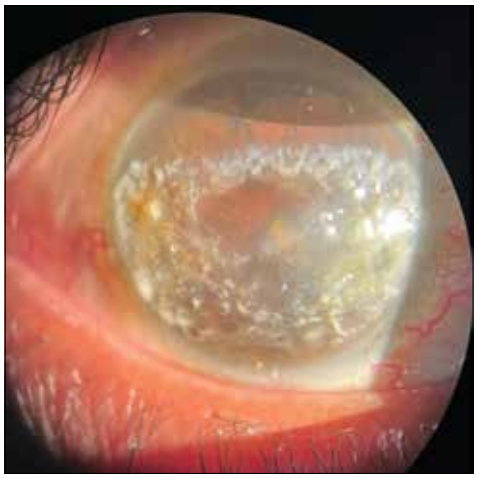


Figure 1: Patient's right eye pretreatment (original)

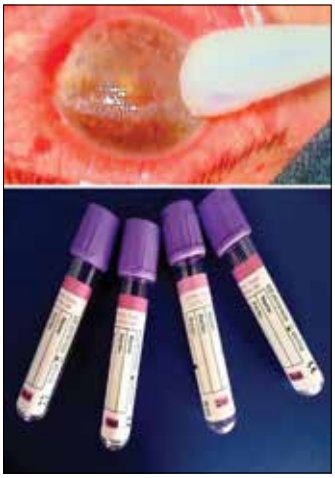


Figure 2: Material used 2ml, 3.6mg EDTA blood collection tube

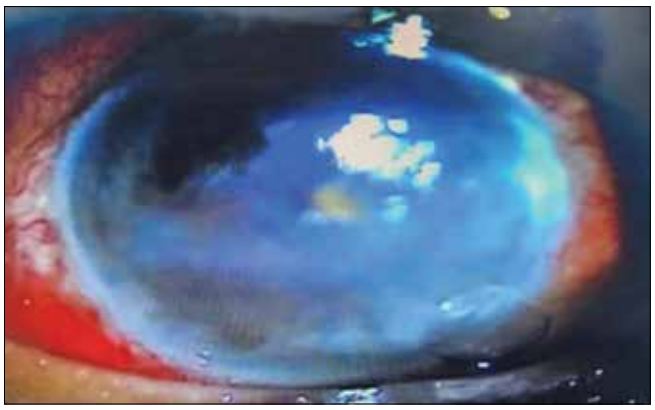


Figure 3: Patient's right eye posttreatment

DISCUSSION

The use of EDTA in the treatment of CBK seems to have begun in 1952, when it was noted that EDTA's lack of lipid solubility prevented it from penetrating the corneal epithelium, necessitating early epithelial removal in the procedure.[9]

Table 1: Cases : Six eyes (Five patients)

Gender	Age	Diagnosis	Visual acuity pre-EDTA chelation	Visual acuity post-EDTA chelation
Male	27 years	RE and LE chronic uveitis	RE 6/36, LE 6/12	RE 6/12, LE 6/9
Female	50 years	RE absolute glaucoma	PL negative	PL negative
Male	55 years	RE absolute glaucoma	PL negative	PL negative
Female	35 years	LE chronic uveitis	6/24	6/9
Male	48 years	LE corneal opacity	6/60	6/24

RE = Right Eye, LE = Left Eye, PL = Perception of light

Superficial debridement is the least invasive surgical management for band keratopathy and can be conducted in a clinic. To remove the deposits, the affected areas are scraped with a beaver blade and then treated with a sponge or filter paper soaked in 2% EDTA. EDTA application occurs every 3 minutes until satisfactory removal of calcium deposits. EDTA can be toxic to the corneal surface; thus, utilizing a soaked method and performing thorough irrigation post-procedure limit its contact with the cornea and prevent toxicity.

Safety of EDTA solution used

According to a retrospective case series study of 20 patients performed by Cartes C *et al.*,[10] superficial keratectomy with topical 12.3% K2 EDTA chelation serves as an efficient and "safe" surgical technique for band-shaped keratopathy clearance, and no complications were observed.

According to the study by Narvaez J *et al.,* K2 EDTA has been shown to be an effective alternative to Na2 EDTA, which is both expensive and inconvenient to obtain. It is also cost-effective and widely available.[11]

CONCLUSION

Chelation of calcium with topical EDTA is a safe and effective treatment for band keratopathy. Because of the difficulty in acquiring disodium EDTA and the easy availability of K2 EDTA in medical facilities, K2 EDTA chelation is preferred over Na2 EDTA chelation. Furthermore, K2 is also preferred because the preparation of the final EDTA dosage is performed under relatively sterile conditions.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal.

Acknowledgements

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Ethical approval

The study was approved by the Institutional Ethics Committee.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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AI for a clear eye: Revolutionizing refractive surgery

ABSTRACT

Advancements in Artificial Intelligence (AI) and Machine Learning (ML) have significantly impacted the field of ophthalmology, particularly in the treatment of refractive errors and cataracts, which are major causes of treatable blindness. With groundbreaking advancements in refractive surgery, including phakic intraocular lenses and LASER Vision Correction (LVC) surgeries—such as flap-based procedures(LASIK), surface procedures(PRK,LASEK), and lenticule extraction procedures—AI-driven innovations have emerged as transformative techniques. There are multiple Kerato Lenticule Extraction (KLEx) surgeries available now - SMILE, SILK, CLEAR and SmartSight. These advancements aim to enhance precision, reduce risk, and improve efficiency in correcting vision impairments like myopia, hyperopia, and astigmatism. AI's role in preoperative screening, surgical planning, and postoperative care promises clearer vision and an improved quality of life for numerous individuals. This article reviews the integration of AI in refractive surgery, highlighting its contributions to patient outcomes and the future potential of AI in ophthalmology.

Keywords: Artificial Intelligence, CLEAR, KLEx, LASEK, LASIK, machine learning, Phakic IOL, PRK, refractive surgery, SILK, SmartSight, SMILE

INTRODUCTION

In recent years, Artificial Intelligence (AI) and Machine Learning (ML) have revolutionized various fields of medicine, and ophthalmology is no exception. Much of the treatable blindness in the word today is due to uncorrected refractive errors and cataract. Refractive errors can be treated by spectacles, contact lenses, or refractive surgery. Refractive surgery has undergone groundbreaking advancements in recent years. There are newer advances in phakic intraocular lenses and LASER Vision Correction (LVC) surgeries including flap-based procedures, surface procedures, and lenticule extraction procedures. There are significant advancements driven by AI in refractive surgery—a transformative technique aimed at correcting vision impairments like myopia, hyperopia, and astigmatism. By enhancing precision, reducing risk, and increasing efficiency, AI is paving the way for clearer vision and improved quality of life for countless individuals [Figure 1].

PREOPERATIVE SCREENING

Starting from the easily accessible Large Language Models (LLMs), one study tried to validate ChatGPT-4's capabilities in categorizing refractive surgery patients based on commonly used clinical parameters. Information supplied included age, sex, manifest refraction, visual acuity, and various corneal measurements and indices from Scheimpflug imaging. Compared to a refractive surgeon, ChatGPT had an accuracy of 68% and a precision of 75% only and responses were also variable.[1]

The significance of preoperative assessment and screening cannot be overstated, with the primary focus being on identifying patients who should be excluded, rather than merely selecting those who are deemed safe. AI is currently being researched for its potential to enhance preoperative

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screening for the risk of ectasia following corneal laser refractive surgery. By guiding the selection of the appropriate type of refractive surgery and automating refraction, AI aims to improve patient outcomes. Iatrogenic ectasia, which can occur after laser refractive corneal surgery, may result from either an undiagnosed preoperative biomechanically weak cornea, such as subclinical keratoconus, or the surgical procedure itself weakening a significant portion of the cornea.

Various ML algorithms have been used to identify eyes with preclinical or subclinical keratoconus. Pentacam Random Forest Index (PRFI), which is an ML algorithm, was shown to be more sensitive and specific than Belin-Ambrosio enhanced ectasia Display – Total Deviation (BAD-D) in detecting keratectasia. Pentacam Inception ResNetV2 Screening System (PIRSS)[2] and Screening Corneal Objective Risk of Ectasia (SCORE) Analyzer were also better than BAD.[3]

Advancements in the field of optical coherence tomography (OCT) have expanded its application in refractive surgery, with Pentacam-OCT and polarization-sensitive OCT (PS-OCT) enabling epithelium and Bowman's layer mapping. High-resolution epithelial mapping has the potential to identify the earliest signs of ectasia. By integrating corneal biomechanics (using Corvis-ST (Corneal Visualization Scheimpflug Technology)) with tomography, a fourth dimension of time is added to preoperative assessment, allowing for the detection of subtle subclinical ectasia. All these valuable Big Data are best analyzed with the help of AI.

GENERATING PATIENT EDUCATIONAL VIDEOS

One interesting project by Han *et al*. looked at three AI text-to-video platforms (InVideo (San Francisco, CA), ClipTalk (San Francisco, CA), and EasyVid (Los Angeles, CA)) and used them to create patient educational videos for Laser-Assisted *In Situ* Keratomileusis (LASIK), Photo Refractive Keratectomy (PRK), and SMall Incision Lenticule Extraction (SMILE). Control videos for LASIK and PRK from the American Academy of Ophthalmology (AAO) and a SMILE video from Zeiss were used for comparison.

All three AI models performed similar to the control videos in terms of script accuracy, image clarity, and script alignment, while medical image accuracy was much worse compared to the control videos.[4]

ANSWERING PHYSICIAN EXAMS ON REFRACTIVE SURGERY

Sensoy *et al*.[5] asked ChatGPT, Bing (Copilot), and Bard (now Gemini) chatbots 70 text-based exam questions related to lens, cataract, and refractive surgery from American Academy of Ophthalmology 2022 to 2023 Basic and Clinical Science Course Lens and Cataract and Refractive Surgery Books. They scored 35 (50%), 43 (61.4%), and 50 (71.4%), respectively. This will presumably keep improving with time as these LLMs voraciously devour information from the Internet.

RECOMMENDING APPROPRIATE REFRACTIVE SURGERY

There are currently many options in refractive surgeries including PRK, microkeratome-LASIK, femto-LASIK, various kerato lenticule extraction (KLEx) surgeries, phakic intraocular lens (IOL), and their many variations.

KLEx surgeries now include SMILE (Zeiss Visumax 500), SMILE Pro (Zeiss Visumax 800), SILK (Smooth Incision Lenticule Keratomileusis) (Johnson and Johnson ELITA), CLEAR (Corneal Lenticule Extraction for Advanced Refractive correction) (Zeimer Z8), and SmartSight (Schwind ATOS).

Kundu *et al*.[6] showed that AI and predictive analysis using a Random Forest ML classifier had a good accuracy in stratifying patients' decisions while selecting a refractive surgery. Factors identified included income, insurance, time spent in the clinic, age, occupation, residence, and source of referral. It correctly predicted 93% of the cases that did undergo refractive surgery.

In South Korea, Yoo *et al*.[7] trained an ML algorithm using data from 18,480 subjects to select the refractive surgery option appropriate for a candidate. It classified patients into LASEK (Laser Epithelial Keratomileusis), LASIK, SMILE, and contraindication groups with an accuracy of 81%.

IMPROVING NOMOGRAMS FOR REFRACTIVE SURGERY

Cui *et al*.[8] used an ML model to develop a nomogram for SMILE surgery to achieve the desired visual outcome. They reported that 93% eyes in ML group and 83% eyes in the surgeon group had a postoperative refractive error within ±0.50D. The safety and efficacy indices were found to be better in ML group than in the surgeon group. Similar models for SMILE were developed by Park *et al*.[9] and Luft *et al*.[10]

CALCULATING POSTREFRACTIVE SURGERY IOL POWER FOR CATARACT SURGERY

The patients who underwent laser corneal refractive surgeries will eventually need to undergo cataract surgery where calculation of corneal power is paramount for IOL selection. Four new-generation formulas with post-laser refractive surgery (LRS) versions have been recently made freely available online: the Barrett True-K, Emmetropia Verifying Optical (EVO 2.0), Hoffer QST, and Pearl DGS.[11] AI-based

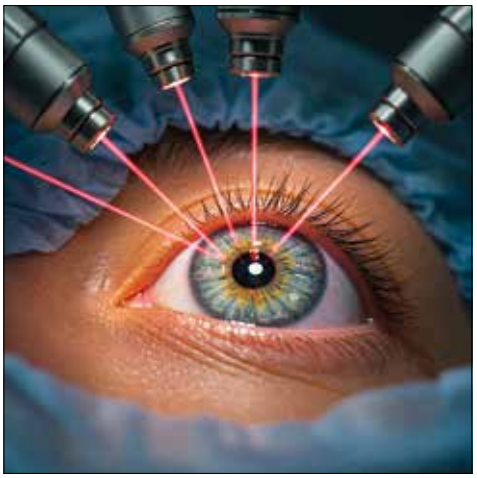


Figure 1: AI-generated image by Google Gemini. Prompt "Create a realistic image of an eye undergoing refractive surgery using lasers. Make sure surgeons fingers are not visible. More lasers"

formulae can help prevent calculation errors and accurately predict IOL power.

AI IN IMPLANTABLE COLLAMER LENS (ICL) SURGERY

Jiang *et al*.[12] developed ML models that provide a novel nomogram for phakic IOL power calculation. Kamiya *et al*.[13] developed an ML-based algorithm to predict the postoperative posterior chamber phakic IOL vault better than manufacturers' nomogram using preoperative AS-OCT images of patients undergoing phakic IOL implantation.

LIRIC (LASER-INDUCED REFRACTIVE INDEX CHANGE)

IRIS (Intratissue Refractive Index Shaping) and LIRIC (Laser-Induced Refractive Index Change) performed at low power at 405nm wavelength are newer refractive surgeries that aim to change the refractive index of the cornea at each point to modify the wavefront. Their application would probably need phase resolved OCT and AI analysis of the cornea and its refractive index.[14,15]

CONCLUSION

AI is ushering in a new era of refractive surgery, characterized by meticulous screening, unparalleled precision, personalized care, and improved patient outcomes. By harnessing the power of AI, ophthalmologists can provide clearer vision and a better quality of life for their patients. As technology continues to advance, the future of refractive surgery looks brighter than ever, promising a world where visual impairments are effectively and efficiently corrected, thanks to the transformative capabilities of AI.

The integration of AI into ophthalmology exemplifies the profound impact of technology on healthcare, ultimately enhancing the human experience and enabling a clearer view of the world.

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Conflicts of interest

There are no conflicts of interest.

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Posterior ectopic cilia causing linear corneal abrasion

A 30-year-old male presented with a foreign body sensation in his left eye for one month. It was aggravated during eye and lid movements. He denied any foreign body falling into the eye or any other form of ocular trauma. He was shown outside the hospital 20 days ago, for which they gave him hydroxypropyl methylcellulose 0.3% and moxifloxacin 0.5%, with which the symptoms were not relieved. Unaided visual acuity (UAVA) for the left eye was 6/18 with normal intraocular pressure. Slit lamp examination showed an apparently normal cornea with normal bulbar conjunctiva, but on fluorescein staining, there was a dense linear epithelial erosion [Figure 1c] seen on the temporal cornea. On everting the upper eyelid, there was a localized erythematous hyperemia covered with slough material on the upper palpebral conjunctiva [Figure 1a]. The slough material was removed using a sterile cotton bud, and a small pointing hair was noted [Figure 1b]. The hair was removed using the forceps [Figure 1d] and was given topical moxifloxacin 0.5% + loteprednol 0.5% eye drops four times a day with carboxymethylcellulose 0.5% four times a day for one week. At the next follow-up of one week, there was no corneal erosion on fluorescein staining [Figure 1f], and the erythematous lesion on the upper palpebral conjunctiva was healed [Figure 1e]. Ectopic cilia is a rare condition of the cilia, present in an abnormal location of the eyelid.[1] Based on their location to tarsal plate, ectopic cilia is classified into two types: the anterior type, which is located anterior to the tarsal plate, and the posterior type, which is located posterior to the tarsal plate. Anterior forms of ectopic cilia are usually of congenital origin, and posterior types are usually acquired.[2] Posterior ectopic cilia usually presents with ocular irritation,[2] foreign body sensation,[3] and pain[2] and also causes superficial punctate lesions of the cornea.[2]

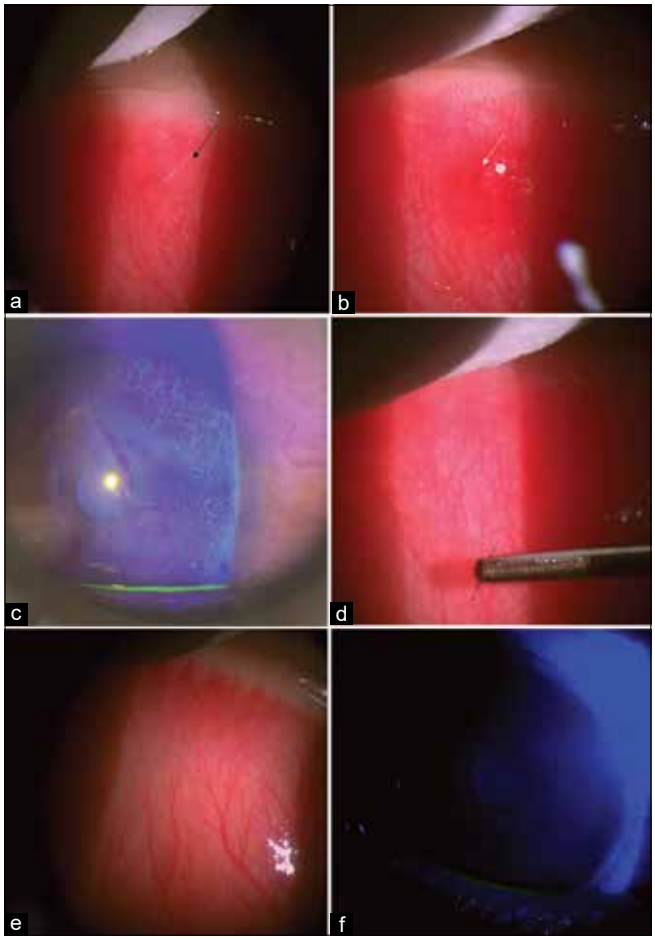


Figure 1: (a) Localized erythema with slough material in upper-left palpebral conjunctiva. (b) Pointed single ectopic cilia which was visible after cleaning the slough with sterile cotton buds. (c) Dense linear abrasion with fluorescein stain under cobalt filter on the temporal cornea, which corresponds to the location of the ectopic cilia. (d) Removal of the cilia using forceps. (e) Healing lesion without any scar. (f) No cornea epithelial defect or abrasion on fluorescein staining

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Surgical excision is the preferred method for the treatment of ectopic cilia.[1] The recurrence of the ectopic cilia was also reported.[1,2] In our case, the diagnosis of ectopic cilia was missed at the initial visit in an outside hospital due to the covering of the slough material, and because of this, the symptoms were not relieved with topical medications. Also, only a single cilia was pointed out, which was easily removed using the forceps, and no other cilia was seen; hence, no incision or surgical excision was done in this case. There was no recurrence noticed at the one-month follow-up. Hence, we suggest that posterior ectopic cilia should be considered as one of the causes of linear corneal erosion in the absence of any other ocular foreign body.

Declaration of patient consent

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

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Nil.

Conflicts of interest

There are no conflicts of interest.

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Materials and method/methodology part in research article

ABSTRACT

The Materials and Method section is the foundation of any rigorous research article. It is where you describe how you did your research, providing critical information that helps others to evaluate and, if required, reproduce it. Its value cannot be emphasized.

Keywords: Materials and method, methodology, research

INTRODUCTION

The Materials and Method section provides a detailed overview of your techniques, allowing other researchers to replicate your studies and validate your results. It also builds up transparency to your research. It also allows the reviewers of the journal to critically evaluate the validity and reliability of your results.[1]

SALIENT POINTS

Materials and Method section in an original research article is discussed in the following sub-headings:

a. **Research design:** Explain the overall structure of your study, including the type of study (e.g., experimental, observational, qualitative), the research setting, and the study population.
b. **Sample Size and Sampling Technique:** A well-calculated sample size ensures that your study has adequate power to identify a true effect and helps in resource optimization. A proper sampling technique guarantees that a representative subset of the population is studied, allowing researchers to draw valid conclusions about the entire population.
c. **Data collection:** Explain how you collected data, including the devices or tools used, the procedures followed, and the data sources.
d. **Data analysis:** Describe the methods you used to analyze the data, including statistical software used and qualitative analytic techniques.
e. **Ethical Considerations:** Discuss any ethical implications for your research, such as informed permission, data privacy, and confidentiality.

COMMON MISTAKES/PITFALLS

- Mismatched research design and research question
- Failing to provide insufficient information for replication of the study
- Lack of justification in explaining sample size, data collection tools, and statistical test used
- Inadequate clarity and organization in methodology.

TAKE HOME MESSAGES

- Logical flow of steps ensures readers and reviewers to understand your research process more clearly.
- **Justification is key:** Every methodological decision, including sample size and statistical tests, must be justified and related to the study issue. Explain why you took a particular strategy.
- **Citing Credible Sources:** Cite the parameter used for sample size calculation and standard data collection tools or methods used in research.

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- **Replication is the goal:** The ultimate purpose of the Materials and Method section is to allow other researchers to duplicate your findings. Provide all of the necessary information to accomplish this.

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Conflicts of interest

There are no conflicts of interest.

REFERENCE

1. Patel M, Patel N. Exploring research methodology: Review article. Int J Res Rev 2019;6:48-55.

Curated article summaries

1. HERPES ZOSTER OPHTHALMICUS RECURRENCE: RISK FACTORS AND LONG-TERM CLINICAL OUTCOMES

Scott DA, Liu K, Danesh-Meyer HV, Niederer RL. Herpes zoster ophthalmicus recurrence: Risk factors and long-term clinical outcomes. Am J Ophthalmol 2024;268:1-9.

SUMMARY

Herpes zoster is caused by the reactivation of the Varicella zoster virus in patients who have been infected with chickenpox. Herpes zoster ophthalmicus (HZO) occurs due to the reactivation of the virus within the trigeminal ganglion, affecting the ophthalmic division of the nerve. Approximately 20% of herpes zoster patients have HZO. Complications include postherpetic neuralgia and vision-threatening conditions such as corneal scarring, perforation, secondary glaucoma, and uveitis. There is a 25% increased risk of HZO among immunocompromised patients.

Recurrence of HZO is a nightmare for both the patient and clinician. It is presumed that the reactivation of the virus occurs due to an immune-mediated response to the VZV virus within the ocular tissues.

This is a retrospective cohort study of 869 patients with HZO from Auckland, New Zealand, who were identified with the disease between 2006 to 2016 and were followed up until 2019. Out of these, 551 recurrences were noted. They observed that uveitis was the most common form of recurrence. The important ocular risk factors identified for recurrence were high baseline intraocular pressure/ocular hypertension, uveitis, and chronicity of ocular disease. Cataract surgery, prolonged usage of topical steroids, and systemic immunosuppression were also found to trigger recurrences. Interestingly, the timing of steroid cessation also contributed to the relapse of HZO. They also found a significant association between the number of recurrences and degree of vision loss in the patients. Moderate to severe vision loss was reported in patients who had more than one recurrence.

WHY THIS ARTICLE?

This study is the largest in the literature to evaluate the risk factors and long-term clinical outcomes of patients with HZO recurrence. An in-depth knowledge and prompt recognition of these risk factors will go a long way in reducing the rates of HZO recurrence. Judicious use of topical steroids and close monitoring in high-risk individuals will help decrease visual morbidity and improve their quality of life.

2. INCREASED SERUM FERRITIN IS ASSOCIATED WITH SEVERITY OF ORBITAL DISEASE IN COVID-19–ASSOCIATED RHINO-ORBITO-CEREBRAL MUCORMYCOSIS: A QUANTITATIVE ANALYSIS

Tanwar S, Mishra N, Sharma P, Kaur A. Increased serum ferritin is associated with severity of orbital disease in covid-19–associated rhino-orbito-cerebral mucormycosis: A quantitative analysis. Indian J Ophthalmol 2024;73:223-7.

SUMMARY

Mucormycosis is a potentially devastating fungal infection that has been increasingly reported during the Covid and post-Covid times. The most common presentation is rhino-orbito-cerebral mucormycosis (ROCM). There was an unprecedented rise in incidence in India, especially during the the second wave of Covid. Approximately 81% of Covid-associated ROCM cases were reported from India. Its rapid progression and high mortality rate caused significant concern among healthcare professionals worldwide.

The important risk factors identified are Covid -19 infection, uncontrolled diabetes mellitus, iron overload, neutropenia, the use of steroids/immunomodulators, prolonged oxygen therapy, and mechanical ventilation. Poor glycemic control is the most important risk factor, as the fungus thrives in a hyperglycemic environment. High blood sugar levels also cause impaired sequestration of iron. As iron sequestration is one of the major host defense mechanisms against fungi, this impairment leads to a further worsening of the condition. Raised serum ferritin is observed when iron levels are increased.

This study was a cross-sectional tertiary care study conducted to assess the role of serum ferritin in Covid-19 associated ROCM cases as a biochemical marker of the severity of orbital disease. It was conducted at King George Medical University, Lucknow, Uttar Pradesh, from May 2021 to June 2021. A total of 100 patients with signs of ROCM were included in this study.

The following biochemical parameters were assessed in all patients: serum ferritin, fasting blood sugar, postprandial blood sugar, C-reactive protein, D-dimer, and interleukin-6. All these parameters were elevated in the patients analyzed in this study, but only serum ferritin showed a statistically significant increase and positive correlation with the severity of orbital involvement. Multiple regression analysis confirmed that serum ferritin could independently contribute to the severity of orbital disease.

WHY THIS ARTICLE?

Orbital mucormycosis is a dreaded complication of Covid-19 infection. This study reveals that, apart from uncontrolled blood sugar levels, serum ferritin also plays a significant role in the pathogenesis and mortality associated with Covid-19–associated ROCM.

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Unlocking the pars plana

ABSTRACT

An overview on the applied anatomy of the pars plana.Algorithms of the various drugs delivered through this route are also provided.

Keywords: Antibiotics, antifungals, antimetabolite, antivirals, intravitreal drugs, pars plana, steroids

THE VITAL GATEWAY

Introduction

The pars plana or orbicularis ciliaris is a relatively avascular smooth pigmented zone that is 4 mm wide and extends from the teeth of ora serrata to the valleys between ciliary processes. It is the safest route for a posterior surgical approach which is located 3–4 mm from the limbus.[1]

ANATOMY OF CILIARY BODY AND PARS PLANA

The ciliary body is the middle portion of the uveal tract which is anteriorly continuous with iris and posteriorly with the choroid. The two principle functions are aqueous humor production and accommodation. It is 6-7 mm wide and has two parts

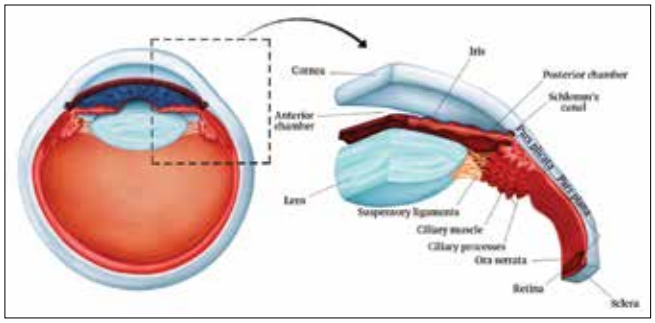


Figure 1: Anatomy of pars plana

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i. Pars plicata - It is the richly vascularised anterior part, and exhibits 70–80 corrugated fin-like folds arranged circumferentially around the crystalline lens (2-2.5 mm).

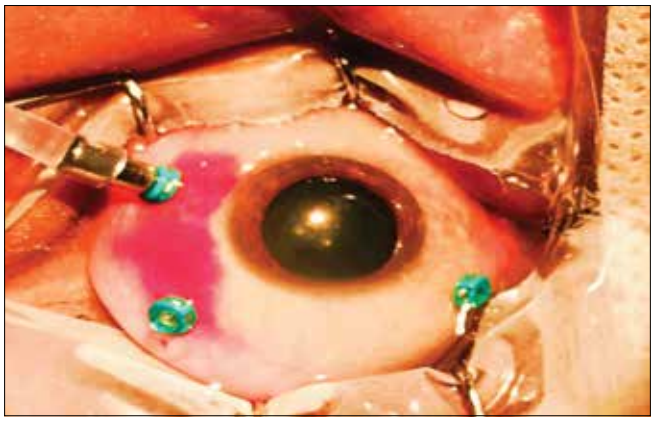


Figure 2: Typical sclerotomy positions with infusion line inferotemporal, cutter and light above horizontal in a 3-port vitrectomy (as viewed by the surgeon)

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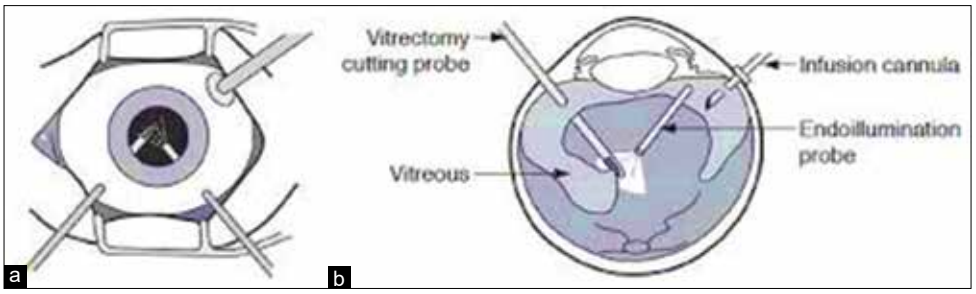


Figure 3: Pars Plana Vitrectomy Ports a-(as viewed by surgeon) with Axial View-b (courtsey: AA0 BCSC chapter 12; retina and vitreous- illustration by dave yates)

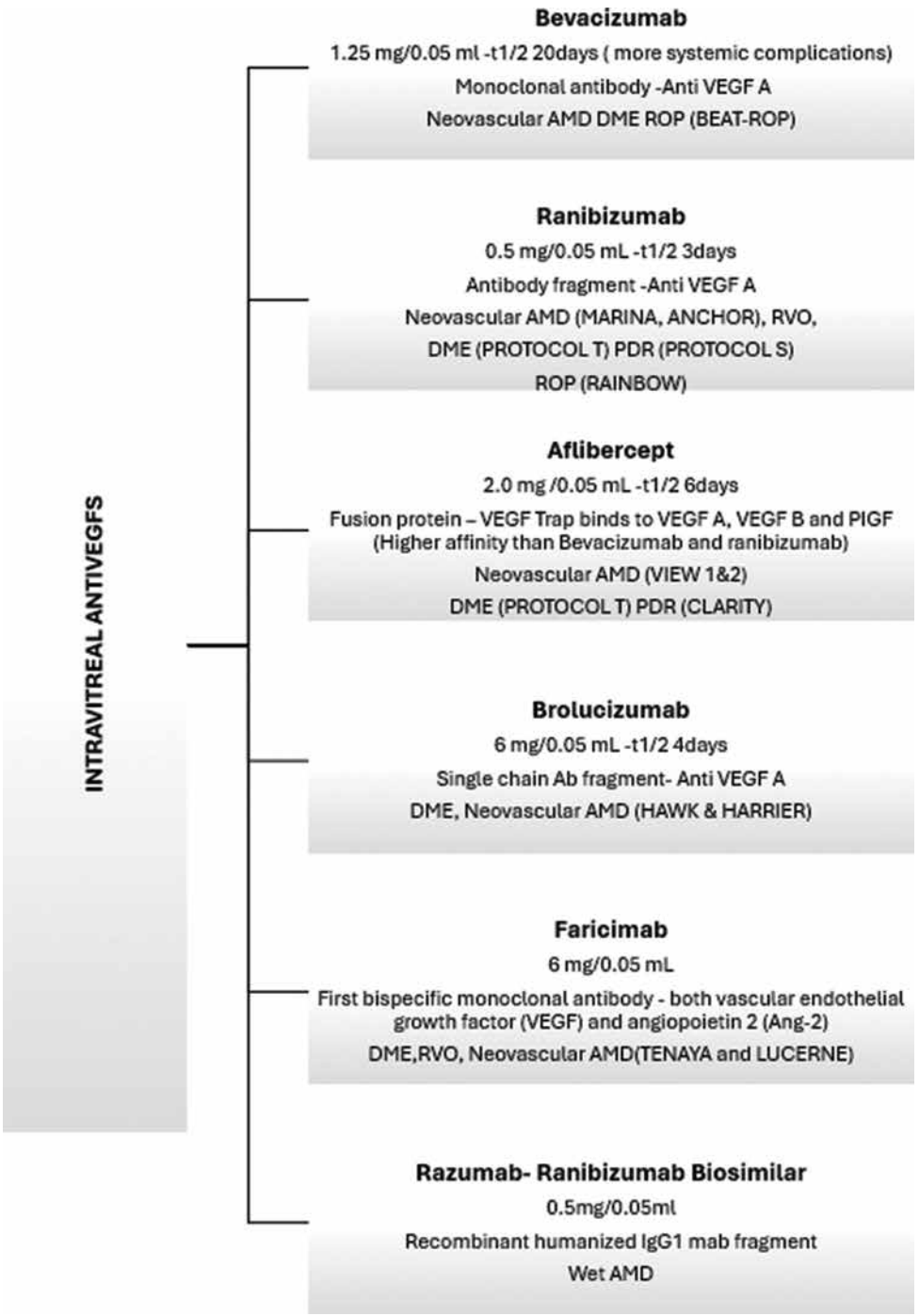


Figure 4: The Intravitreal Antivegfs

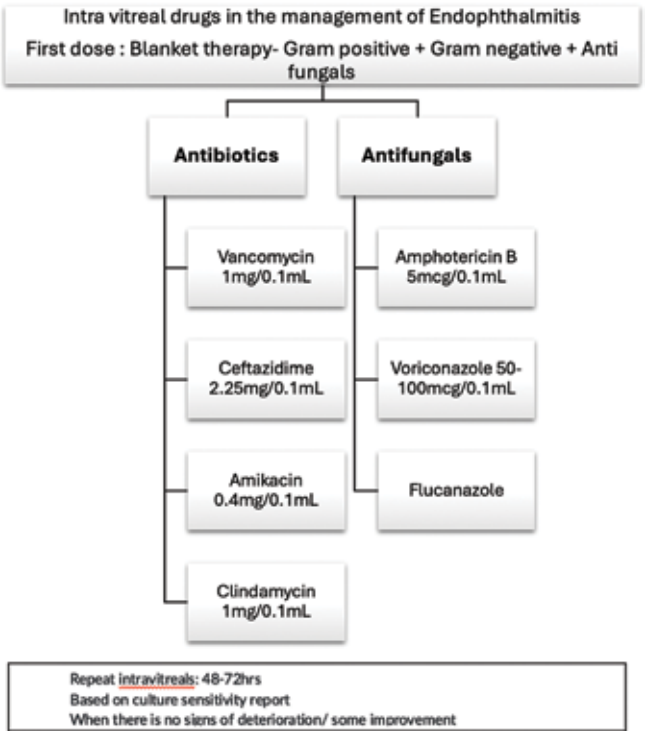


Figure 5: Intravitreal anitbiotics and antifungals

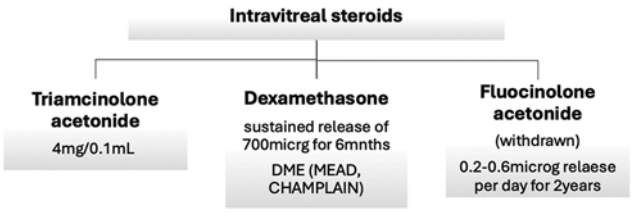


Figure 6: Intravitreal steroids

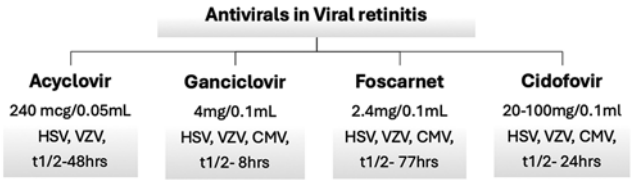


Figure 7: Intravitreal antivrials

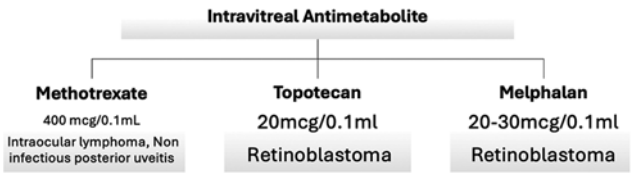


Figure 8: Intravitreal antimetabolites

ii. Pars plana - Posterior smooth part (4.5 mm wide temporally and 3 mm wide nasally). It is a relatively avascular, smooth-pigmented zone that extends from the ora serrata to the ciliary process.[1]

[Figure 1] Anatomy of pars plana.

SURGICAL ANATOMY

Pars plana is chosen for various intraocular procedures as it is the safest point of entry into the eye which causes minimal ocular trauma. Also, it does not serve a sensory purpose and is not involved in the production of aqueous and vitreous humor.

The posterior part of the pars plana is covered by the vitreous base which extends both anterior and posterior to ora serrata. Most needle punctures and incisions are made 3 to 4 mm posterior to the limbus through the anterior part of the pars plana to avoid injury to the vitreous base.[2]

Pars plana vitrectomy and intravitreal injections are the commonly done procedures for various vitreoretinal pathologies.

Infants have smaller eyes and the surgical landmarks are different from adults. The pars plana is not fully formed by 8–9 months old, so the sclerotomy ports are placed at various distances from the limbus depending on the age.[3]

PARSPLANA VITRECTOMY (PPV)

This surgical technique involves a closed-system approach

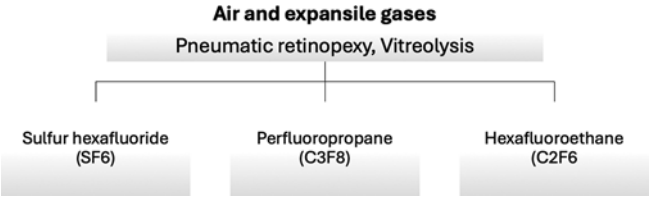


Figure 9: Intravitreal gases

in which 3 ports are placed 3-4 mm posterior to the surgical limbus, depending on the patient's lens status.

One port is dedicated to infusion into the vitreous cavity to achieve the desired intraocular pressure. Other ports are for visualization and various manipulations in the vitreous cavity.[4] A fourth port ,Chandelier endoillumination is used for bimanual vitrectomy. [Figures 2 and 3] Typical sclerotomy positions.

MCQ CHALLENGE

Identify the true statement:

a. Pars plana is wider on the nasal side than the temporal side.
b. Aqueous humor is produced by both pars plana and pars plicata.
c. Vitreous base extends only posterior to ora serrata.
d. Placement of sclerotomy ports in children is age-dependent.

PARS PLANA: THE ROAD TO INTRAVITREAL INJECTIONS

Antiangiogenic agents, steroid preparations, sustained delivery devices, antimicrobials, and various medications in clinical trials are delivered into the eye through the pars plana.

We can obtain vitreous fluid through a vitreous tap or biopsy which is typically done through pars plana for various microbiological and histopathological examinations which aids in the diagnosis and treatment of various conditions.[5]

Figures 4–9 the intravitreal drug deliveries.[6-9]

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Nil.

Conflicts of interest

Nil.

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Releasable sutures: For safe and titrated trabeculectomy

ABSTRACT

Trabeculectomy is considered the gold standard for glaucoma surgery, providing effective intraocular pressure control. However, complications such as over filtration leading to the hypotony and under filtration causing surgical failure can arise. Releasable sutures serve as an effective solution to mitigate these risks by allowing postoperative titration of aqueous outflow. These externalized interrupted scleral flap sutures regulate flow through the internal ostium and can be easily removed via a slit lamp. There are various techniques for using releasable sutures, and this video demonstrates two primary methods.

Keywords: Complications, releasable sutures, trabeculectomy

Video Available on: https://drive.google.com/file/d/1cz4XS6VLkZwNgZHTOTce1XV_P8SgRJSu/view?usp=drivesdk

FOCUS POINTS[1,2]

1. **Modification of Cohen's Technique**:
   - In this technique, the first suture bite is made at the scleral end, with the needle initially positioned away from the surgeon, then turned toward them. A second bite is taken at the limbus, creating a corneal loop.
   - A partial-thickness scleral bite is taken close to the base of the triangular flap, to get a posteriorly directed bleb.
   - At least four throws are made before tightening the loop of the suture. Once secured, cut the end of the suture.
   - The loop at the corneal end of the suture is where you hold and pull to release the suture in the postoperative period.
2. **2nd Method**
   - A 10-0 nylon suture is threaded through the clear cornea and passed through the limbus. A loop is formed between the corneal sutures, which is used to remove the suture later.
   - The scleral flap and bite placement techniques mirror the first method.
   - Disadvantage in this technique is that the corneal end is exposed easier forming a windshield wiper effect on cornea. When you are cutting the corneal end gently pull before you cut so that the cut end is retracted and does not get exposed.

CONCLUSION

Releasable sutures are a cost-effective alternative to laser suture lysis, with the lower risk of complications such as buttonholing and subconjunctival hemorrhage. Typically released within three weeks of surgery,[3] these sutures help maintain anterior chamber stability, particularly in angle-closure glaucoma, while minimizing over filtration. It provides an effective and simple way of titrating intraocular pressure postoperatively.

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Conflicts of interest

There are no conflicts of interest.

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Reply to comments on the case report of "Fleeting diplopia and wobbly feet: Miller-Fisher syndrome – Presenting as bilateral abducent nerve palsy"

Dear Editor,

We sincerely thank Chaurasiya and Suraj Kumar *et al*. for taking their valuable time to review our article, 'Fleeting diplopia and wobbly feet: Miller-Fischer syndrome presenting as bilateral abducent nerve palsy.' Your valuable observations and insightful comments are appreciated.

Regarding the limitations encountered during the evaluation and treatment, we work hand in hand with neurologists when dealing with neurological/neuro-ophthalmic conditions. We do not view this as a limitation; rather, we consider it our responsibility to refer the patient to the neurologist promptly for their expert opinion. We use their inputs along with our clinical diagnosis to define the future course of management. The same approach was followed here as well.

Regarding the follow-up, we agree that we did not mention a long follow-up in the case report. However, we have follow-up data for 6 months post intravenous immunoglobulin (IVIG) therapy. The patient has recovered with full eye movements and has no ataxia. The patient however continues to have a small esotropia persisting in primary gaze with diplopia, for which he was prescribed Fresnel prism with which he is managing well.

In our article, we aimed to sensitize the general ophthalmologist to the salient features of a rare neurological syndrome as well as to highlight how it could present in a regular outpatient department. We did not dive into the depth of various manifestations and presentations in detail due to the constraints of case reporting. The core clinical presentations to an ophthalmologist are diplopia with symmetrical ophthalmoparesis and lateral rectus (100%) being most commonly involved, followed by superior rectus (60%), inferior (52%), and medial (48%) rectus muscle.[1] Most of the patients present 10–15 days following an antecedent event. Eye manifestations vary from complete ophthalmoplegia, external ophthalmoplegia, internuclear ophthalmoplegia, acute ptosis, and so on.[1,2] Involvement of other cranial nerves (other than 6,3,4) and paresis of the extremities have been reported.[1]

Detailed attention to the subtle neurological findings in any patient presenting with diplopia or ophthalmoparesis, along with features of ataxia, with proper neurological evaluations can avoid delay in diagnosis, facilitate early management, and hasten recovery.

We respect the points of view expressed by our esteemed colleagues and hope this case gives more insights when a similar presentation comes in the future.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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Comment on comparing the prevalence of lattice degeneration and requirement of barrage laser in different grades of myopia

Dear Editor,

The published study by Bhat S., titled "Comparing the prevalence of lattice degeneration and requirement of barrage laser in different grades of myopia" caught our attention.[1] Although the study provides insightful information on the frequency of lattice degeneration in myopic individuals, I would like to draw attention to a few points that should be discussed and given more consideration to support the findings:

1. **Sample Selection and Representation**: The patient group for the study was selected from a single ophthalmology center, which may have introduced bias into the screening process. This restriction might limit the applicability of the results to a larger myopic population, as the sample may not accurately reflect a range of demographic and geographic origins.[2]
2. **Exclusion Criteria**: While it is methodologically sound to isolate the variable of interest, excluding patients with a history of systemic ailments, surgeries, other ocular problems, or specific medications may limit the study's applicability. This approach could unintentionally exclude a significant portion of the population that might provide additional information about the connection between lattice degeneration and myopia.
3. **Study Design - Cross-sectional Nature**: Although long-term data are needed to monitor the development of lattice degeneration and the long-term efficacy of treatments such as barrage lasers, the cross-sectional approach only offers a glimpse of prevalence at a given moment. Longitudinal research is recommended to gain a deeper understanding of these processes.[3]
4. **Assessment Methods**: Variability in diagnostic accuracy is a concern when subjective evaluation methods, such as slit-lamp examination and scleral indentation, are used. The necessity for standardized evaluation techniques to guarantee consistency in identifying lattice degeneration is highlighted by the possibility of inter-examiner variability.
5. **Statistical Analysis and Confounding Factors**: The Chi-square test used in the study's analysis does not appear to account for potential confounding variables, such as sex, age, or other demographic characteristics. Addressing these factors is essential to ensure the validity and robustness of the results.
6. **Generalizability of Findings**: The study's findings may not be fully applicable to other groups, particularly those from diverse ethnic and geographic backgrounds, where the frequency and risk factors for myopia and lattice degeneration can vary.
7. **Variability in Treatment Indications**: The decision to use barrage laser therapy is based on specialist judgement, which may result in inconsistent treatment methods. Developing established policies or procedures for deploying lasers could reduce this variability, ensuring more uniform and equitable patient treatment.[4]
8. **Ethical Considerations and Informed Consent**: Although the study mentions obtaining informed consent, it would be beneficial to provide more detail on the ethical considerations, particularly regarding the disclosure of potential risks and benefits associated with participation and treatment.

In summary, while the study makes a significant contribution to the understanding of lattice degeneration in myopic patients, addressing these methodological concerns could enhance the reliability and applicability of the findings. I commend the authors for their work and suggest these areas as potential avenues for future research and refinement.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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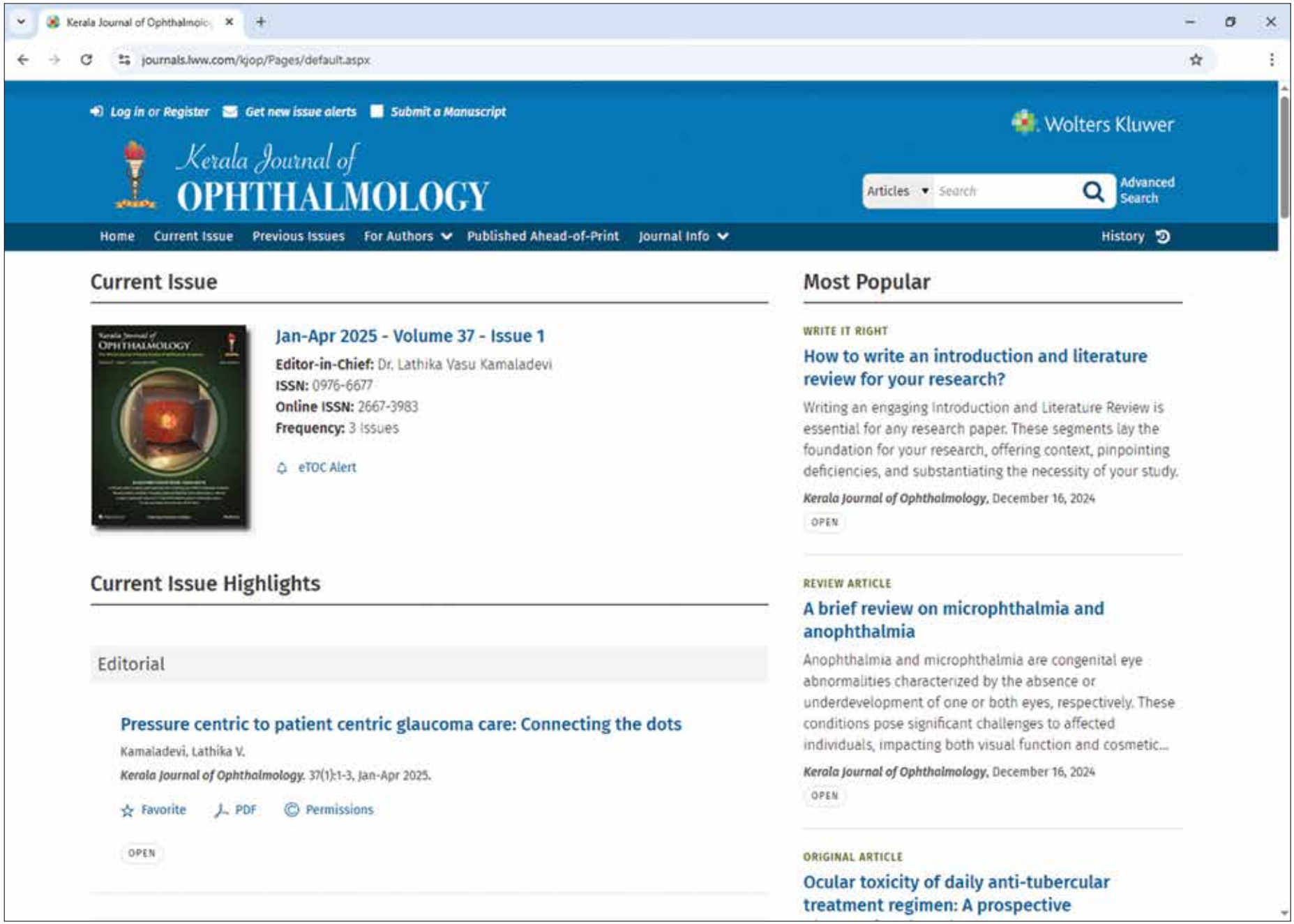
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