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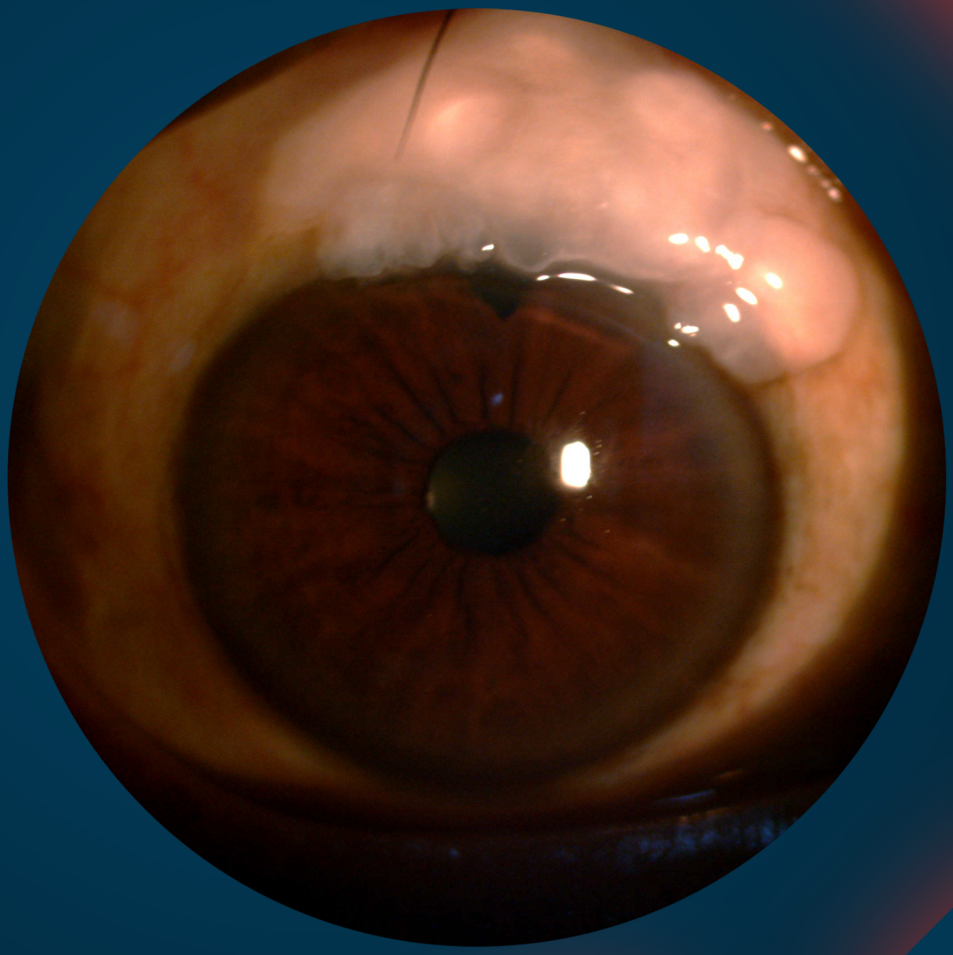

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MADHYA PRADESH STATE OPHTHALMIC SOCIETY

2022-2024

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MESSAGE FROM EDITOR IN CHIEF

Dr Vinita Ramnani
Editor in Chief, MPSOS Times

Respected seniors and dear colleagues

I feel delighted and extremely proud to present the first issue of scientific magazine of M.P. State Ophthalmic Society (MPSOS) as a Chairperson Scientific Committee. This is my second small effort for the MPSOS. First feather in cap is **PRECEPTORS OF FUTURE**, a series of competitive PG webinars from all the Divisional Societies of MPSOS.

With great help of dynamic editor Dr Mita Joshi along with three young and enthusiastic co-editors - Dr Ravi Chandil, Dr Chahveer Singh Bindra, and Dr Rahul Choubey along with all the advisors and section editors, first glaucoma issue is ready and will be released in midterm MPSOS conference at Jabalpur. Moving in future towards cornea, cataract, retina and other subspecialities in upcoming issues of MPSOS TIMES. which will have 10 diffrent subheads in each subspecialty issues. I request and expect active participation from all MPSOS members.

The present issue is dedicated to Glaucoma, hoping you all will like this issue, I am thankful to all the contributors. Special mention to my mentor and Gurudev who taught me glaucoma at L.V. Prasad Eye Institute Hyderabad - Dr G. Chandra Shekhar who generously accepted and blissfully gave guest editorial for MPSOS Times. I would like to thank our national faculties - Dr Manav Deep Singh, Secretary GSI, and Dr Manish Singh, Treasurer GSI for their unconditional contribution by giving expert opinion on Normotensive Glaucoma and Dr Suneeta Dubey, Medical Director, Shroff Hospital for her views on MIGS

Thanks and regards to all

Dr Vinita Ramnani

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MESSAGE FROM EDITOR

Dr Mita Joshi
Editor, MPSOS Times

Greetings from Editors Desk of MPSOS Times

MPSOS Times has traditionally been a newsletter keeping all members up to date with activities happening across all divisional societies of the MPSOS. This time we have decided to give it a more scientific bent and invited articles from all members for publication in their very own state society. We plan to shape it into a peer reviewed journal down the line.

March hosting the Glaucoma Awareness Week, this issue of MPSOS Times focusses on the silent thief of sight that is Glaucoma. We have a variety of articles covering a part of the vast spectrum of diseases that is glaucoma.

I take this opportunity to thank editor in chief Dr Vinita Ramnani for her constant guidance and encouragement and coeditors Dr Ravi Chandil, Dr Chahveer Singh Bindra and Dr Rahul Coubey for their enthusiastic support and section editors Dr Parag Sharma and Dr Tanuja Kate for their efforts.

Happy Reading !

Dr Mita Joshi

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MESSAGE FROM CO-EDITOR

Dr Ravi Chandil
Co-Editor, MPSOS Times

Respected seniors and Dear colleagues

It's great pleasure on my part as co-editor to bring out this issue of MPSOS Times on subspecialty “Glaucoma” which has an interesting mix of articles.

Glaucoma is the second leading cause of blindness worldwide. Since the disease runs an asymptomatic course, it is not very uncommon to see patients presenting for the first time in very advance stage. Early diagnosis and timely initiation of appropriate therapy is the key to successfully prevent it from progressing to an irreversible stage.

This issue of MPSOS TIMES on glaucoma include expert opinion on Normal Tension Glaucoma which is one of the difficult subtype to diagnose, treat and monitor. It have articles based on “recent advances in diagnosis and medical management of glaucoma”. This issue also consist of article on some basic yet important things like “calibration of applanation tonometer” and “cleaning of goniolenses”. It also covers articles on “medical management on glaucoma”, how to choose “drugs in pregnancy and lactation”. This issue also having good information on “lasers” (LPI, SLT, Diode CPC) as well as on surgical aspects of “MIGS”. This issue also contain wisdom pearl to “differentiate between glaucomatous and non-glaucomatous optic disc” and information on “impact of hypertension and hypotension on glaucoma”.

Our aim is to keep the readers well versed with basic things and recent developments in glaucoma fields.

I wish you all a pleasant reading

Dr Ravi Chandil

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MESSAGE FROM PRESIDENT

Dr Ashok Singhai
President, MPSOS

Dear friends

Happy world glaucoma week 2023

It is a matter of great pleasure that our dynamic Chairperson Scientific Committee - Dr Vinita Ramnani is coming up with the scientific magazine of MPSOS. Since the beginning, her efforts and dedication to our society has been evident and starting Preceptors of Future program for PGs is an appreciable initiative. I must congratulate the team for all new efforts put in for MPSOS, my full support is always there with team.

I am grateful to all the contributors of this issue and expect same enthusiasm and support for all upcoming volumes of MPSOS TIMES.

I am sure this issue of MPSOS TIMES is fruitful to you all and I will be happy to see you all at midterm MPSOS conference organised at Jabalpur on 9th April 2023.

Hope to enjoy reading and acknowledge efforts

With best wishes

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MESSAGE FROM HON. SECRETARY

Dr Rajeev Gupta
Hon. Secretary, MPSOS

Namaskar,

It gives me great pleasure to introduce first edition of our ophthalmic magazine dedicated to GLAUCOMA, “MPSOS TIMES”

Our team lead by dynamic scientific chairperson Dr.Vinita Ramnani and Editor Dr.Mita Joshi has worked very hard to bring it out.

Our magazine is dedicated to provide valuable information and updates to ophthalmologists, researchers in the field, we aim to explore the latest research, advancements, and treatments in GLAUCOMA, to help our readers stay up to date with the latest trends and innovations, our goal is to create a platform where experts can share their knowledge, experiences and perspectives on the topic.

We hope that our magazine will serve as a valuable resource for anyone interested in glaucoma and will help in the effort to prevent and treat this condition.

Thank you for your support and we look forward for your continued readership.

Dr Rajeev Gupta

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CONTENTS

EDITORIAL

Compassionate and Patient Centric Care

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Congratulations to the MP Sate Ophthalmic Society on its inaugural e-journal. It is said that "doctor is a student throughout his life". This is very valid in view of the rapid advances being made in the diagnosis and treatment of many eye diseases in the recent past. To be able to deliver the current and best treatment to our patients' we do need to update our knowledge and skills; and medical journals are an important aid towards this goal. Understandably, this issue of the journal is focussing on recent advances in many aspects of glaucoma diagnosis and care. I would like to share some thoughts on how patients' needs and perspectives might be divergent from our own and factoring these in our support for the patients would go a long way in compassionate and effective patient care.

Daniel Goleman in his seminal work on Emotional Intelligence writes "Medicine has defined its mission in terms of curing the disease or the medical disorder, overlooking the illness –the patient's experience of the disease."1 Illness is a combination of the physical symptoms of the disease and the psychological uncertainty and fear of disability and death. These fear and uncertainty lead the individual to a childlike dependence.[2] What is needed is for the medical profession is to meet the psychological needs of the patient along with the medical ones. Compassion is not merely hand holding, it is good medicine.[2]

In the interest of specialized and state of the art care in rare and complex diseases, super specialization is a well-justified need. But the risk of the super specialization is that the super specialist is so focused on "reducing the IOP" or "settling the retinal detachment", that their relevance to the eye as a whole or perhaps the patient as an individual with a family and social life and associated commitments could be missed.

A typical medical consult and the ensuing communication is focused on the bio-medical aspects disease; focussing on the diagnosis and prognosis. If our care needs to be patient centric, we should also focus on understanding the impact of the disease on patients psychology. We need to know his/her concerns about the possible cost and duration of therapy, the recovery time, ability to sustain the current livelihood as well as the aspirations for future based on the treatment outcomes etc. These would comprise the psychosocial aspects of the illness as opposed to the bio-medical aspects focussing on diagnosis and prognosis.[3] While as doctors we will not be able to solve the social and economic constraints for the planned management plan, the fact that we are even attempting is a big step in the right direction. With this attempt in place, we may come up with equally efficacious plans that can accommodate some if not all of the constraints. This planning of the treatment along with the patient rather than on the patient is likely to increase the compliance to therapy and improve the outcomes.

Informed consent is a double edged sword. Sharing the worst case scenario in disease progression, possible side effects of medications and complications of surgery could be counterproductive in some patients, but is needed in a litigant health care set up. May be these are two ends of the spectrum and in a given situation, we need to modify the details of the informed consent based on the patient's psychology (a glaucoma suspect very worried because someone in the family has gone blind with glaucoma versus a busy executive who might neglect the moderate to advanced glaucoma). It is reported that 4-26% patients who are randomly assigned to placebos in trials discontinue their use because of perceived adverse effects. These perceived side effects anticipated by the information has been called "nocebo".[4] The psychological impact of fear of blindness in a patient of glaucoma suspect might be more devastating. Finding a way to balance on how much disclosure of potential adverse effects therapy as well as the natural course of the disease is a challenge. While information may be crucial, taking away hope and a positive attitude towards the interventions is not the best possible holistic care.
Even with all the above thought process and intention of the physician in the right direction, effective communication is anther ball game altogether. EMPATHY a hall mark of patient centric care, has been used as an acronym to achieve better patient communication.[5]

Eye contact: Talking to the patient directly and with an eye contact is more reassuring than ignoring the patient and talking only to the attendant or quickly giving the advice while completing "notes".
Muscle tone: If we observe the patient facial expression closely, we will know if the patient is callous about the disease or very scared. Our approach in these situations could be different as alluded to above.
Position: Even if we spend a limited time with the patient, if we are seated at the same level of the patient, rather than standing or facing the computer, we communicate that we have enough time for them and not in a hurry.
Affect: is same as muscle tone (facial expression / emotional state) of the patient
Tone: The tone of our voice while talking to the patient can convey leisure and support rather than hurry and authority.
Hearing the whole patient: It is important to hear the "whole patient" by contextualising the no-verbal communication signals with the patients' remaining narrative and social world and avoid focussing on just symptoms or body parts in isolation.
Your response: We need to be aware of our own mental status and neither feel nor convey anger, frustration, and detachment.

In conclusion, the objective of compassionate and patient centric care is achieved, if we recognise the emotional state of our patients and endeavour to not only take care of the disease but the illness as experienced by them.

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EXPERT OPINION ON NTG

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Q1: What is NTG?

MDS: It is defined as typical changes in visual structure and/or function in the absence of numerically high IOP. To explain a bit further, structure means the disc and imaging findings; RNFL defect and/ or disc haemorrhage on fundus examination/ photography. The function means The visual field defects, especially if they have charateristic glaucomatous pattern and if the corrospond to the structural defects. In nut shell, all findings suggestive of glaucoma but with IOP <21 mmHg.

PV: NTG: Normal Tension Glaucoma. As the terminology itself suggest that, a patient having normal IOP ,but glaucomatous Optic Nerve Head and RNFL changes and co relating visual field defect. Here I want to make a point that normal IOP does not mean that single office hour reading on GAT but it should be diurnal variation recording.Second patient should not have very thin cornea(less than 460 micron)

VR: Normal tension glaucoma is an optic neuropathy with glaucomatous optic nerve head damage, progressive retinal nerve fiber layer thinning and corelating characteristic visual field defects, associated with open angles on gonioscopy and maximum intraocular pressure below 21 mmHg.

MP: Normal Tension Glaucoma (NTG) signifies the presence of characteristic glaucomatous optic neuropathy in the presence of "statistically" normal IOP with open angles. The "statistical" border between what a normal IOP and a higher IOP would be close to 21 to 22 mm Hg from population based studies. The IOP should ideally be measured in a diurnal testing and secondary causes for glaucoma such as old trauma, uveitis, steroid response among others should be excluded. In essence, it is a "diagnosis of exclusion" after a thorough history and clinical examination.

NSA: NTG is an optic neuropathy where glaucomatous changes or damage to the Optic nerve head occur with an intraocular pressure (IOP) within the statistically normal range i.e < 21mm Hg and open anterior chamber angles.

MS: Normal-tension glaucoma is a progressive glaucomatous optic neuropathy with intraocular pressures in the normal range. It is a diagnosis of exclusion. Hence one needs to exclude secondary ocular and systemic causes of optic nerve damage. Criteria for Diagnosis:
IOP < 21 mmHg (on Diurnal IOP measurement or multiple clinic visits)
Open angles on gonioscopy
Optic nerve and RNFL changes characteristic of Glaucoma
Changes on visual fields corroborating with disc changes

Q2: What are the causes responsible for NTG?

MDS: Classically it is believed that if an optic nerve head cannot tolerate a certain pressure and results in typical glaucomatous changes at a pressure <21, it may result in NTG. However, it has to be understood that numerical figures are man made. There are no absolute numerical figures in nature. Like one person at 6' 6' height may be absolutely normal, other may have gigantism with same height. Similarly, an IOP of 18 may be normal for one person but high for another. All this may be dependent on various factors e.g. CCT, trans lamina cribrosa pressure differential (depending on CSF pressure), viscoelastic properties of cornea (resulting in measurement errors) and un diagnosed diurnal variation. Apart from IOP related factos, vascular factors and sleep apnoea have also been implicated in NTG.

PV: We must consider following issues before labelling it as classicle NTG:
Intermittent angle closure disease, Thin CCT, Fluctuation of IOP, Steroid induced, Old Trauma, PDS(Burnt out), Recurrent Uveitis(e.g. Fuch,s,P S Syndrome), Neurological conditions like pituitary mass, Vascular conditions like old arterial occlusion, and Senile sclerotic disc
Classical NTG may have some systemic features and may not have.Systemic known causes are: Lower ocular perfusion pressure, Peripheral vasculopathy like Raynaud's phenomeon Pressure gradient across lamina cribrosa(Low Intra cranial pressure causes bowing posteriorly of lamina), Low pulse rate and low diastolic pressure, Nocturnal hypoxia, Migraine, Female gender, and Elderly population

VR: The pathogenesis of NTG remains unclear and it is believed that interaction of many systemic factor is associated with onset and progression of NTG like - (1) Impaired blood flow causes optic nerve susceptible to glaucomatous damage. (2) Reduced resistance of the optic nerve head to IOP in vascular insufficiency. (3) Reduced peripapillary retinal blood flow and structurally abnormal lamina cribrosa. (4) Vascular dysregulation causing repeated reperfusion injury to the optic nerve along with systemic hypotension specifically, a diastolic ocular perfusion pressure of less than 55mmHg has been associated with a 2-to-6-fold increase in the prevalence of glaucoma. (5) Autoimmune dysfunction and the possible role of intracranial and cerebrospinal fluid pressure and BMI.

MP: Clearly, as opposed to POAG, the optic nerve is showing signs of glaucomatous damage despite a recorded physiological range of IOP. Both POAG and NTG belong to the spectrum of open angle glaucomas. However they share distinct differences. Both may be affected by pressure dependent and pressure independent factors. At the higher IOP, the pressure dependent factors may be more predominant while at lower IOP, pressure independent factors may be predominant factors causing glaucomatous damage. In addition reduced blood blow in vessels supplying optic nerve are thought to be contributory

NSA: NTG has a multifactorial group of etiologies - mechanical, vascular or neurodegenerative. An IOP-dependent mechanism plays a role in the etiology in many eyes with NTG. The other causative factors independent to IOP include higher translaminar pressure gradient, impaired Cerebrospinal fluid (CSF) circulation, vascular dysregulation (systemic and local) and hematologic abnormalities. Genetic predisposition has also been suggested.

MS: Etiology of NTG is multi-factorial. Multiple heterogenous causes lead to retinal ganglion cell loss in NTG. Although IOP is normal, still IOP dependent cell loss plays a role in pathogenesis of NTG. Other Non IOP dependent factors like vascular insufficiency, oxidative stress, metabolic and neurodegenerative disorders, and abnormal biomechanics of lamina cribrosa are also important. Genetics also plays a significant role. There is strong association with family history and variation in prevalence in different ethnic groups.

Q3: What are ocular and systemic risk factors of NTG?

MDS: Low CCT, low CSF pressure, nocturnal and even day time systemic hypotension, beta blocker intake in evenings and vascular insufficiencty are the risk factors.

PV: Ocular risk factors are: Thin cornea, Weak Lamina cribrosa, Ischemic insult of Retina
Systemic risk factors are: 1) Peripheral vasculopathy, 2) Nocturnal hypoxia(sleep apnoea), 3) Bradycardia, 4) Low diastolic BP, 5) Brain hypoxic insult involving visual pathways

VR: Raynaud's phenomenon, migraine, systemic hypertension, systemic hypotension, nocturnal hypotension, cardiac arrhythmia, shockinduced neuropathy, chronic obstructive arterial disease and obstructive sleep apnea syndrome. Lifestyle factors such as smoking, high body mass index and impaired glucose tolerance may contribute to an increased risk of NTG.

MP: Even though IOP is within normal range, it still remains a risk factor for development and progression of glaucoma in NTG. Besides, studies have shown lowering of IOP reduces risk of progression in NTG. Systemic factors affecting vascular dysregulation such a migraine headaches, low blood pressure and cold hands and feet. A past history of hemodynamic crisis and sleep apnea should be determined. These conditions may lead to severe hypoxia and decreased ocular perfusion.

NSA: The ocular and systemic risk factors are thin central corneal thickness, Age, Family history, Race, Systemic hypertension, Nocturnal hypotension, Migraine, Raynaud phenomenon and obstructive sleep apnea.

MS: Some key risk factors for NTG are: Old Age, Family history of Glaucoma, Female Gender, Above-average IOP, Thin central corneal thickness, Systemic hypertension and Nocturnal hypotension, Migraine and Raynaud phenomenon, Obstructive sleep apnea, Carotid insufficiency and Hemodynamic crisis

Q4: How is NTG different from POAG?

MDS: NTG is only a part of continuous spectrum of OHT and POAG. In fact I do not believe that NTG is of something of common occurance. If we do 24 hour diurnal variation test and take CCT in measurements, many patients diagnosed with NTG will turn out to POAG. However, in true NTG, vascular insufficieny and low CSF pressure are most likely causes of glaucomatous structural and functional changes. Whereas angle of anterior chamber is open in both cases, sclerosis of trabecular meshwork is a more prominent feature of POAG. Another difference is in regard to response to treatment. Currently the only definitive treatment for Glaucoma is reduction of IOP. Since IOP is already low in NTG, drugs are not very effective. Even surgery, within the limits of safety, may not be able to achieve an IOP which is low enough to prevent further field damage. Another difference is whereas assymetry is the hallmark of POAG, NTG is more likely to be bilaterally symmetrical.

PV: NTG is a sub type of POAG with little differences 1) NTG either does not progresses (30%) or slow progresses as compare to POAG, 2) NTG patients never have IOP of more than 21 mm of Hg, 3) ONH has large cup,with prominently visible laminar dot sign,cup is shallower than POAG and prominent beta zone, 4)NTG has more often splinter haemorrhage at disc margin than in patients with POAG

VR: Normal-tension glaucoma is a subtype of primary open angle glaucoma where maximal intraocular pressure never exceeds 21 mmHg. According to Baltimore eye survey 50% of patients and Beaver Dam Eye Study nearly one-third of glaucoma have NTG. NTG patients tend to be older than those with primary open angle glaucoma, more frequent among females and Japanese people (Two-thirds of Japanese patients).

NTG patients have wider diurnal fluctuations of IOP with nocturnal IOP spikes. Central corneal thickness in patients with NTG is lower than POAG. ONH tilt, torsion and disc haemorrhages are features of NTG. Patients with high myopia are susceptible to NTG with frequent temporal crescent. Visual field defects are more likely to be deeper, steeper and closer to fixation often at low levels of total loss in NTG compared to POAG. The characteristics visual field changes of primary open-angle glaucoma are typical arcuate scotoma at Bjerrum's region and paracentral scotomas.

MP: NTG is typically characterised by increased frequency of presence of disc haemorrhages and visual field defects which are deeper and closer to fixation. NTG tends to cause more "focal' notches of the optic disc. Acquired optic disc pits seen as localised excavations of lamina cribrosa are more frequent.

NSA: NTG is a diagnosis of exclusion after we have ruled out other causes mimicking glaucomatous disc and visual field changes in an eye with a normal IOP. These patients are generally of greater age (± 60years) compared to POAG, although an entity known as NTG in the young does exist. Also, vascular dysregulation and systemic causes like Raynauds phenomenon, sleep apnea, nocturnal hypotention etc are believed to be more commonly associated with NTG than POAG. Localized, deep paracentral scotomas, are more common in NTG as compared to POAG. In NTG, the frequency of disc hemorrhages is also more common but the progression is generally slower than POAG in most cases.

MS: NTG is often regarded as a variant of POAG where IOP is consistently normal. The role of Non-IOP dependent factors is more in etiopathogenesis of NTG compared to POAG where IOP is the predominant causative factor. Patients with NTG are generally a decade older at time of presentation than POAG patients and female preponderance is seen in NTG while no such gender difference is seen in POAG. Clinically flame shaped optic disc hemorrhages (Drance Hemorrhage), deep and focal nothing of the rim and peripapillary atrophy (Beta zone) are more frequently seen with NTG patients. Visual field defects noted in NTG are more focal and occur closer to fixation than POAG.

Q5: What is the battery of investigations performed in NTG?

MDS: Apart from standard investigations requied to establish the diagnosis viz. IOP measurement, Gonioscopy, Visual field examination, optic nerve examination/ photography, CCT measrement and imaging, we may need to do 24 hour DV for IOP with 24 hour BP monitoring, color doppler of carotid vesselas and detailed cardio-vascular assessment and even CSF pressure measurement may be desirable, especially if field defects keep progressing. Going into the details of personal history and discovering certain high risk practices may be helpful e.g. exercises involving total inversopn of body (sheerash aasan, sarvang aasan), wearing of tight tie, habbit of taking large quantity of water empty stomach, tobacco consuption and over dose of drugs for systemic hypertension.

PV: In many cases you would need help of: Neurophysician, Cardiologist, and pulmonologist, and good co ordination is needed between all stake holders, Investigations may involved, MRI brain(With MR angiography), Manometry for CSF pressure, Colour dopplar study for Carotid and Orbital vessels, Echo cardiography, Holter study for 24 hrs monitoring BP and Pulse, Sleep study for sleep apnoea (These all investigations are the discretion of respective specialist)

VR: (I) Detailed Clinical Examination - Visual acuity, Colour vision testing (to help differentiate from non-glaucomatous optic neuropathies) Goldmann applanation tonometry with Diurnal or supine measurement, Pachymetry, Afferent pupillary response testing. Complete slit lamp examination, gonioscopy, stereoscopic biomicroscopy of optic nerve head, optical coherence tomography and Humphrey field analyser.

(II) 24-hour blood pressure monitoring to exclude nocturnal systemic hypotension. Blood tests to rule out other causes of optic neuropathy such as vitamin B12 and folate levels, ESR/CRP and serum ACE. Nail fold capillaroscopy with cold provocation may detect blood flow abnormalities. ECG, ECHO and Doppler ultrasound may be used to monitor blood flow to the eye including the optic nerve.

(III) MRI & Neurological Evaluation is needed when - Age less than 50 years with atypical neurologic symptoms and unexplained visual acuity loss. Colour vision deficits or marked asymmetry or unilateral optic nerve involvement and pallor more than cupping. Visual field defects not corresponding or out of proportion to optic nerve damage and vertically aligned visual field defects.

MP: Clearly I would reassess the fluctuation of IOP (diurnal or office diurnal every 1-2 hourly) to determine whether the single recorded IOP is actually a snapshot of larger fluctuation of IOP. For example the IOP may be ranging from 18 to 23 mm Hg in a diurnal and the time of the day I checked IOP, it was noted to be 18 mm Hg. This will also help determine my target IOP and range. I would also rule out other causes of secondary glaucomas ex steroid use in past, past trauma or inflammatory eye diseases. Measuring blood pressure and getting requisite investigations such as visual fields (24-2) and central corneal thickness. I would also be keen to have a 10-2 program in these cases as defects tend to affect the central zones. The newer programs like 24-2C on the HFA3 would also help. Stereoscopic Optic disc photographs and optic disc imaging (ex OCT) can be used to document baseline data and progression. Measurement of systemic factors such as blood pressure are pertinent.. In select cases ambulatory BP monitoring and polysomnography to detect sleep apnea in consultation with physician are options. Overtreatment of blood pressure should be avoided and nocturnal hypotension should be avoided to hamper blood supply to optic nerve. Usually NTG is a typically slowly progressive disease. Atypical cases and those with rapid progression may require further neuro-imaging.

NSA: Slit lamp examination and Gonioscopy to rule out any secondary causes and to check the anterior chamber angle; Pachymetry to measure corneal thickness; Phasing if possible but more commonly office hour IOP measurement; Optic disc photography for baseline and subsequent structural changes and progression; Automated perimetry- 24-2, 10-2 and/or macular threshold depending upon the

stage of disease; OCT for RNFL and Ganglion cell analysis and for monitoring progression; 24 hour blood pressure monitoring if facility available
Contrast-enhanced MRI is usually advised in patients, who do not have a family history of glaucoma and present with sudden onset of symptoms like diminution of vision, associated with neurological symptoms like severe headache, nausea and vomiting etc. They usually have reduced visual acuity or RAPD, unilateral disease, atypical disc findings, such as pallor more than cupping, marked asymmetric cupping or with field defects respecting the vertical midline or not corresponding with optic disc or rapid progression. Many of these patients are of young age having a clear media and no visible attributable cause for the above signs and symptoms.

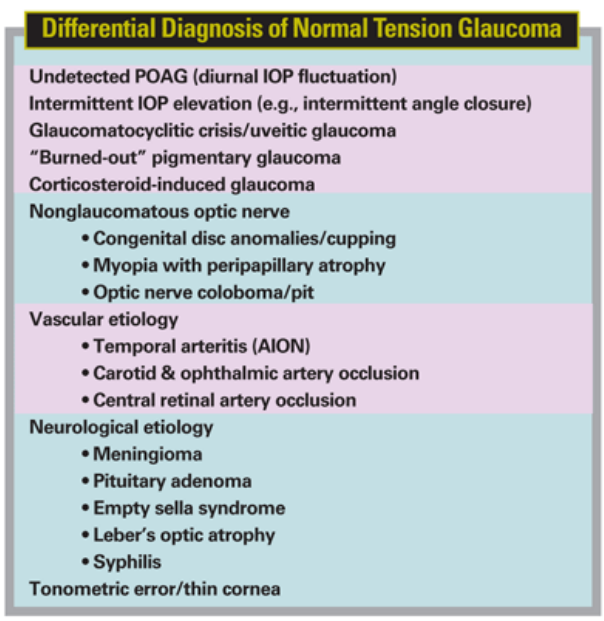
MS: **History:** Family history of Glaucoma; Any history of headache, syncope, dizziness, loss of consciousness etc (neurological symptoms); Use of steroids in any form; Any history of surgery, trauma, or inflammation; History of practices like headstand during Yoga, excessive water drinking and prolonged breath holding; Anemia, B12 or folate deficiency; Systemic diseases like migraine, sleep apnea, hypertension, hypotension, or acute blood loss;
Clinical examination: Visual acuity; Color vision (usually preserved till late stage); Slit lamp evaluation to look for secondary causes like pigment dispersion, pseudo-exfoliation, angle recession and sign of past inflammation; IOP should be normal by definition; Angles should be open on gonioscopy ;Pupillary response testing; Dilated Stereoscopic disc and fundus evaluation ; Diurnal IOP (if feasible) to exclude IOP fluctuations above 21 mmHg
Diagnostic procedures. Pachymetry; Automated Static Visual fields to look for defects corresponding to disc changes; Optic disc imaging for disc and RNFL assessment : Stereo Disc Photography or Optical coherence tomography
Systemic and Neurological workup: Patients with atypical visual field changes and those progressing on well controlled IOP should be evaluated for other vascular and neurological causes of optic nerve damage - 24 hour BP assessment; Anemia and Vit B 12 assessment; Carotid doppler study; Cardiological evaluation; Diabetes work up; Sleep apnea study; CT Scan or MRI
Patients with optic pallor more than cupping, younger age (<50 yrs.) and vertically aligned visual field defects should undergo neuroimaging preferably MRI.

Q6: How is differential diagnosis of NTG performed?

MDS: Most important DD are died down steroid induced glaucoma and anterior ischemic optic neuropathy. However, come retinal probems like BRVO, branch arterial occlusion, sectorial argon laser photocoagulation, atypical retinitis pigmentosa, sectorial mass lesion of retina and things as simple as myelinated nerve fibres may lead to arcuate field defects giving false impression of NTG. The confrimation is possible only by demonstarting progression over a period of time. Although NTG is a bilateral problem, AION is generally unilateral.

PV: Intermittent Angle Closure Disease(When IOP is normal at the time of measuring during office hours; Thin CCT(In very thin corneas GAT erroneously record low IOP); Fluctuation of IOP(Normal circadian rhythm ,it low point IOP is measured in office); Steroid induced(History of long term use or abuse of steroid in responders,which has caused glaucomatous damage now during visit patients is off the steroid and IOP is normal)d; Old Trauma (Traumatic optic neuropathy with normal IOP) PDS(Burnt out); Recurrent Uveitis(e.g. Fuch,s,P S Syndrome); Neurological conditions(Pitutary mass,lesion along with optic treck pathways); Vascular conditions (Peripheral vasculopathies, Old Retinal artery occlusion; Senile sclerotic disc(Classical papilla in old age withpout glaucomaous damage)

VR:



MP: I would consider the following as confounders: POAG: If IOP noted is normal either as a single reading in a diurnal curve and/or inaccurately measured by an inappropriate tonometer for accurate IOP (ex non-contact/ Schiotz etc); Past Glaucoma: Cases on chronic steroid use in past (topical/ systemic) or conditions such as uveitis/ trauma causing raised IOP in past ("burnt out glaucoma"); Congenital anomalies of optic disc or physiological cupping mimicking glaucoma

NSA: The DD includes - POAG with thin central cornea or a POAG patient on oral beta blockers; Chronic angle closure glaucoma; Previous episode of a raised IOP, including a history of ocular trauma, hyphema, uveitis, use of steroids or burned out glaucoma like previous pigmentary glaucoma, etc; Optic disc abnormalities like Large physiological cupping, tilted discs, large myopic disc, disc drusen, or congenital anomalies like optic disc pit, optic disc coloboma, segmental optic nerve hypoplasia are also a part of DD but are non- progressive; Previous vascular injuries like arteritic or non-arteritic anterior ischaemic optic neuropathy, retinal vascular occlusions, and systemic hypotension e.g., after surgery, trauma, etc; Optic neuropathies including inflammatory optic neuropathies like optic neuritis, hereditary optic neuropathies like Leber's hereditary optic neuropathy (LHON), toxic optic neuropathies like methanol poisoning, or traumatic optic neuropathy (TON); Intracranial tumors like pituitary adenomas etc or intraorbital tumors are also a part of DD.

MS: The common conditions which we need to differentiate are -
POAG with thin CCT (Diurnal IOP may help to differentiate); Burnt out pigmentary glaucoma; Resolved steroid induced, uveitic or traumatic glaucoma; Optic nerve coloboma or pit; Myopia with peripapillary atrophy; Glaucomatocyclitic crisis (Posner-Schlossman syndrome); Congenital optic disc anomalies like morning glory syndrome; Compressive, metabolic, toxic, inflammatory or infectious optic neuropathy

Q7: How and when to treat NTG?

MDS: Whenever you are sure of diagnosis. When in doubt, follow up the patient till you can demonstrate progession. If you are able to find a cause, like vascular factor, treat the cause. Target pressure in these eyes is generally kept lower than POAG with equavalent severity of functional loss. Treatment protocol is same as that of POAG. Treat with drugs. If target pressure is not achieved, do surgery. In surgery, aim at lower pressure by making a thinner scleral flap and by using MMC of stronger strength.

PV: Every patient of NTG should be treated to prevent further damage; Goal of treatment to reduce IOP by 30% from it.s trough at baseline; Preffered Drug is PG analog; CAI may have role in enhancing vascular supply to ocular tissue; Alfa 2 Agonist may have some neuroprotective ability; But as there is not enough data available to support this hypothesis hence both drugs if used than they are used as IOP lowering drugs only, Trabeculectomy is good option 1) In advance cases, 2) Cases where you do not want any fluctuation, 3) IOP lowering drugs are not preventing progression, 4) Poor compliance for any reason

VR: If a case looks likely a non-progressive type (history of cardiovascular incident, history of past use of corticosteroid eye drops, evidence of past pigmentary glaucoma) then to monitor the patient's condition till rate of progression is determined. Many NTG patients have mild or moderate disease that progresses very slowly and may not have an impact on a person's vision or quality of life such patients keep a watch on blood pressure, ensure that it is not over-treated avoid nocturnal Dip. According to the Collaborative Normal Tension Glaucoma Study Group an IOP reduction of 30% slowed the progression of normal-tension glaucoma. If a case seems typical and likely progressive, then start to lower IOP substantially to 10-12 mm Hg and follow-up further with respect to both the disease and adverse results from treatment. Choices for medical treatment in progressive NTG - Brimonidine significantly improved retinal vascular autoregulation in NTG patients. Dorzolamide is a safe and effective IOP lowering agent in patients with NTG. Prostaglandin derivatives tend to have greater IOP lowering effect. Betaxolol eye drops have beneficial effect on optic nerve blood flow in addition to IOP reduction. Other beta blockers and adrenergic drugs (such as dipivefrine) should better be avoided because of the probability of nocturnal systemic hypotension and optic nerve hypoperfusion.

A single session of selective laser trabeculoplasty (SLT) for NTG can achieve IOP reduction of 20% to 30% from baseline IOP. Deep sclerectomy or MIGS seems to be effective and safe in reducing IOP in patients with NTG. Trabeculectomy with or without MMC is considered if progression occurs despite medications. Treatment of anaemia, hypotension, congestive heart failure, transient ischemic attacks, and cardiac arrhythmias to increase optic nerve head perfusion. Memantine, Ginkgo biloba, Brovincamine ,Nilvadipine selective a calcium channel blocker are under trial and practically not being used much.Further research is required to improve understanding of the pathogenesis, diagnosis and treatment of this challenging disease.

MP: All cases of NTG do not progress. Besides this, studies have shown certain NTG cases to progress despite treatment. Some studies have reported inter eye asymmetric IOP (within normal range) to show increased progression in higher mean IOP eye in NTG. Other studies contradict these reports. Clearly, therapy is not required in all cases and a balanced approach on a case to case basis in mandatory.

For a "non progressive" patient, no treatment may be required. For detection of progression, close follow ups and serial visual fields/ optic disc imaging. Nonetheless, most studies do report that lowering of IOP either by medicines or surgery does reduce risk of progression. A desired lowering of IOP by 25-30% is preferable. For desired IOP lowering, prostaglandin analogues are considered drug of choice. In addition newer RhoKinase Inhibitors can be used and drugs such as brimonidine and carbonic anhydrase inhibitors can be used for their presumed beneficial effects on retinal circulation/neuroprective effects. Progressive patients on medications may need surgery (especially Trabeculectomy) to achieve desired IOP reduction to low teens/ single digits. Patients with nocturnal BP drop on systemic hypotensives should have a physician consult to avoid systemic medications especially during night. This is especially important in those progressing despite adequate IOP control.

NSA: For the non- IOP dependant component, stopping smoking, treating sleep apnea, chronic anaemia, blood loss etc are a part of the treatment. Also overtreating hypertention (HT) with anti HT medications and nocturnal hypotention (specially Beta blockers at night) should be avoided. For the IOP dependant component, a minimum of 25-30-% reduction of IOP should be achieved. For this, topical anti-glaucoma drugs like prostaglandins (First choice), brimonidine, betaxolol, etc are used. Laser trabeculoplasty (LT) is also another treatment option in patients having drug issues like allergies etc. Minimally invasive glaucoma surgery (MIGS) procedure combined with phacoemulsification is another option but less available in India. Trabeculectomy with or without cataract surgery is the preferred surgery of choice. Treatment is initiated in patients with moderate or advanced disease at presentation with threatened macular involvement, in patients with documented structural progression like disc haemorrhage or in visual field progression especially in fast progressors, or in those requiring low IOP in single digits.

MS: More than 50 % patients with NTG don't progress. Patients with early non progressive field defects can be kept under regular monitoring, but it should be discussed with the patient. Optic disc haemorrhages are also often considered as marker of disease progression. IOP reduction is the only known modifiable risk factor that can alter disease progression even for NTG patients. Collaborative Normal Tension Glaucoma Study (CNTGS) has shown that IOP reduction reduces progression in NTG patients. They have demonstrated that 30 % reduction of IOP from baseline should be aimed in NTG. Prostaglandins are the drug of choice. LoGTS study has shown that Brimonidine 0.5% might be better alternative to Timolol 0.5% in NTG patients. Topical Carbonic anhydrase inhibitors (increase vascular flow) and Rho-kinase inhibitors (neuroprotective and increased vascular flow) are also useful as adjuncts to prostaglandins for treating NTG. Selective laser trabeculoplasty (SLT) or drainage surgery may be considered for patients progressing on maximum medications. Mitomycin C (MMC) should be used in patients undergoing trabeculectomy as it helps in achieving lower target IOP.

MIGS (Minimally Invasive glaucoma surgeries) has limited role in NTG.

Patients should be advised to maintain healthy life style, monitor blood pressure , blood sugar and cardiac status, avoid anti-hypertensives a night, stop smoking, exercise regularly and practice mindfulness meditation.

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For a "non progressive" patient, no treatment may be required. For detection of progression, close follow ups and serial visual fields/ optic disc imaging. Nonetheless, most studies do report that lowering of IOP either by medicines or surgery does reduce risk of progression. A desired lowering of IOP by 25-30% is preferable. For desired IOP lowering, prostaglandin analogues are considered drug of cho`ice. In addition newer RhoKinase Inhibitors can be used and drugs such as brimonidine and carbonic anhydrase inhibitors can be used for their presumed beneficial effects on retinal circulation/neuroprective effects. Progressive patients on medications may need surgery (especially Trabeculectomy) to achieve desired IOP reduction to low teens/ single digits. Patients with nocturnal BP drop on systemic hypotensives should have a physician consult to avoid systemic medications especially during night. This is especially important in those progressing despite adequate IOP control.

RECENT ADVANCES

Recent Advances in Diagnosis of Glaucoma

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Glaucoma is second leading cause of blindness, affecting over 60 million people worldwide[1]. Due to an ageing population the prevalence of glaucoma is expected to increase substantially[2]. Because the condition is often asymptomatic in the early stages more than 50% of the patients with glaucoma are undiagnosed and nearly 14% blind in one eye at first presentation [3].

For the diagnosis of glaucoma, apart from comprehensive eye examination (slit lamp examination, gonioscopy, and dilated disc evaluation) one have to do IOP measurement, visual field assessment, and RNFL assessment by imaging. Due to advancement in technology we have newer technique for IOP measurement, visual field assessment, RNFL imaging in our armamentarium. The goal of management is to prevent vision loss from glaucoma in a patient's lifetime and to maintain or enhance quality of life. New diagnostic technologies helping us to accurately detect and monitor this disease so that we can appropriately treat our patients. This article gives an overview of the new diagnostic modalities.

A. Recent advances in IOP Measurement
B. Recent advances in visual field assessment
X. Recent advances in imaging

A. Recent advances in IOP Measurement

Although we don't diagnose our glaucoma patients solely on the basis of IOP but still IOP is the main modifiable factor. So IOP measurement play important role in glaucoma management.

Home Tonometry

The largest area of innovation in tonometry is home-based IOP measurements. As of now, we are only able to get a snapshot of a patient's IOP while they are in the clinic, which does not account for the natural diurnal curve. These diurnal fluctuations tend to be higher in patients with glaucoma which raises their risk of progression[4]. Not only diurnal fluctuation, Glaucoma patients don't have similar IOP in all the days of week. New devices such as the iCare Home tonometer, the eyemate system, and the Sensimed Triggerfish, allow out-of-office measurements that reflect the peaks and troughs of a patient's pressures and can be used for customized treatment plans.The Icare Home tonometer (Fig. 1) is designed to allow patients to measure their IOP at home. A significant advantage of this technology is that it uses rebound tonometry and thus does not require the administration of a topical anesthetic. All the data are wirelessly saved to a cloud database that can be seen by treating doctor only, not by patients so self-medication is avoided. Glaucoma treatment is currently based on a few seconds of in-office measurement per year, at-home tonometry could significantly alter how a patient's glaucoma is managed even in paediatric or postoperative patients especially after tube surgery. Limitations remain, including an inability to measure IOP during sleep. Nearly 10% of patients are not able to operate properly[5]. Disparity with GAT was greater when central corneal thickness was <500 µm or >600 µm[6].

Figure 1. icare home tonometer

(Photo courtesy of Icare)

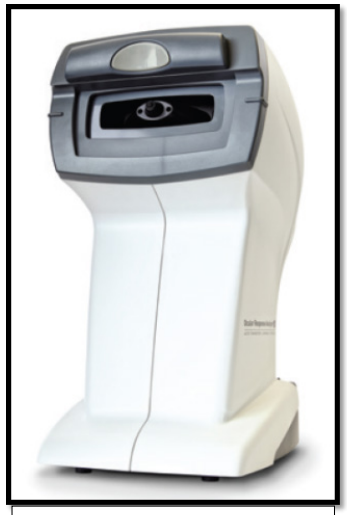

Figure 2. Ocular Response Analyser

(Photo courtesy of Reichert Technologies)

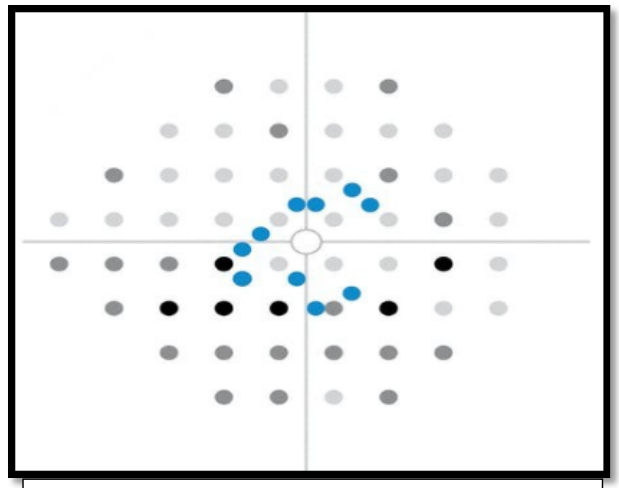

Figure 3. Arrangement of Central 10 New points24-2c

(Photo courtesy of Zeiss HVF3 manual)

Ocular Response Analyzer

Ocular Response Analyzer (Fig. 2) measures corneal hysteresis (CH), which is the difference between the pressure at which the cornea bends inward during air jet applanation and the pressure at which the cornea bends out again. It is the measure of the viscoelasticity of the cornea that corresponds to its ability to dampen forces imposed upon it. In different Studies it have been found that higher CH is linked to a lower probability of developing glaucoma and a higher maintained visual field index over time. It is suggesting that CH mimics the flexibility within the lamina cribrosa and indicates the eye's capacity for shock absorption, in turn which would suggest an ability to withstand deformation and stress from IOP fluctuations[7].

B. Recent advances in visual field assessment

Visual field testing is mandatory tool in the detection and monitoring of glaucoma. New advancement in testing Point Pattern, thresholding algorithms and new portable perimetry devices seems to have potential to improve visual field testing in clinical practice.

Recent advances in Testing Point Pattern

Traditionally, 24-2 visual fields are the most commonly used method for visual field assessment in glaucoma which screen few points within the macular area. Despite the fact that central vision affects last in glaucoma, there is an increasing appreciation that damage at the macula can be detected in even early stages of glaucoma[8]. In a recent study of patients with early glaucoma, nearly 61% eyes classified as normal on 24-2 tests were classified as abnormal on 10-2 visual fields[9]. In patients with ocular hypertension, nearly 35% eyes classified as normal on 24-2 tests were classified as abnormal on 10-2 visual fields[13]. It is therefore apparent that central visual field damage on the 10-2 test may be missed with the 24-2 strategy alone. The 24-2c (Fig. 3) test was developed based on these new data. The test includes 10 additional points within the macular area that follow the asymmetric pattern of the bundle, testing the most vulnerable points[8].

Recent advances in Thresholding Algorithms

Novel thresholding strategies are being investigated that incorporate spatial and structural information to improve the speed and precision of visual field testing. Some of the new perimetric algorithm are Gradient-Oriented Automated Natural Neighbour Approach (GOANNA), Spatially Weighted Likelihoods in Zippy Estimation by Sequential Testing (SWeLZ) and Structure Estimation of Minimum Uncertainty (SEMU). GOANNA uses spatial information regarding the location of a field defect to improve the characterization of field loss without increasing testing times[10]. SWeLZ uses spatial information on every presentation to alter visual field estimates, to reduce test times without affecting output precision or accuracy [11]. SEMU uses structural information to drive stimulus

choices and predicts sensitivity at a location based on OCT data and make an estimate of sensitivity at a location before any stimuli are shown, and then carefully test around this estimate[12]. These strategies have the potential for significant time savings in clinical settings but require validation in larger scale.

Virtual Reality Perimetry

Virtual reality (VR) (Fig. 4) headsets is another breakthrough in visual field assessment in glaucoma. These tests correlate to Humphrey fields, are available at a lower price, and can be more easily used by patients who are wheelchair-bound, bedridden, or have neck or back problems. Out-of-office examination can be done, which provides an opportunity to perform serial testing at home and can be used as tele -health approach[13].

C. Recent advances in imaging

Optical coherence tomography (OCT) is now the most widely adopted imaging modality for glaucoma. OCT has revolutionized the management of glaucoma by allowing us to quantitatively measure the peripapillary retinal nerve fiber layer (RNFL) and macular ganglion cell-inner plexiform layer (GC-IPL) thicknesses in vivo. Especially in early to moderate cases these measurements improve the detection of glaucoma. After disease diagnosis, these measurements can be used to monitor progression .New parameters, imaging protocols, and modalities are further enhancing the ability to diagnose and monitor glaucoma. Major recent advances include the introduction of three-dimensional OCT scanning, swept-source OCT, OCT angiography, and adaptive optics

Three-dimensional OCT scanning

OCT provide us highly-accurate quantitative assessment of the optic nerve head and surrounding retinal structures to assist in the diagnosis and monitoring of glaucoma. Traditional parameters include assessment of optic disc area, rim area, cup-to-disc ratio, and two-dimensional measurement of the thickness of the peripapillary retinal nerve fiber layer (RNFL) and ganglion cell-inner plexiform layer (GC-IPL). RNFL damage detection is helpful in the early detection of glaucoma, frequently preceding the development of visual field loss. However, two-dimensional parameters are susceptible to artefacts that may adversely affect their diagnostic ability specially where there are variations in optic disc size, optic nerve head tilt, peripapillary atrophy, and myopia[14]. Introduction of three-dimensional volume scans enable high-density sampling of nerve tissue and 3D reconstruction of the neuroretinal rim anatomy which may assist in the diagnosis and monitoring of glaucoma.

New parameter is the minimum distance band (MDB) (Fig. 5) due to three-dimensional scanning of RNFL and ganglion cell layer (GCL) volumes .The MDB is the shortest distance between the internal limiting membrane (ILM) and the optic disc margin, defined as the termination of the retinal pigment epithelium (RPE)/Bruch's membrane (BM)[15]. The MDB has multiple advantages over traditional parameter in which optic disc margin define based on the clinical optic disc margin while in 3D OCT, RPE/BM termination is an objective, consistent, and easily-identifiable anatomic landmark. Secondly MDB measurements are perpendicular to the course of retinal ganglion cell (RGC) axons, so they take into account the variable orientation of RGC axons as they approach the optic nerve head. MDB has been validated as a marker for glaucoma and has been shown to have good diagnostic performance compared to two-dimensional RNFL measurements. Shieh et al. showed that 3D MDB neuroretinal

Figure 4. VisuALL virtual reality visual field perimeter headset

(Photo courtesy of Olleyes)

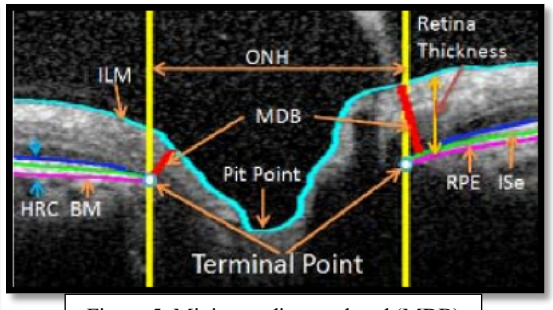

Figure 5. Minimum distance band (MDB)

(Photo courtesy of DICTA)

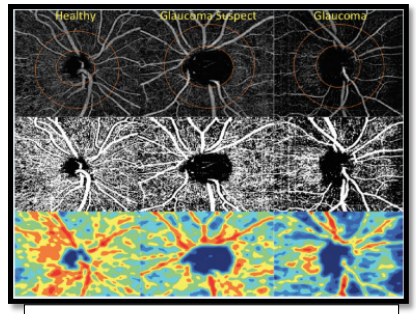

Figure 6. RNFL vessel density map in healthy, glaucoma suspects and in POAG eyes

(Photo courtesy of Glaucoma Today)

rim thickness measurements had uniformly equal or better diagnostic performance for glaucoma in all quadrants and was significantly better in the nasal region compared to 2D RNFL thickness measurements[16].

Wide-Field Swept-Source Optical Coherence Tomography

Recently introduced technology called swept-source OCT (SS-OCT) uses a swept laser to capture wide-angle, high-quality images of the optic nerve and macula in a single scan so omit the need of multiple scan[17]. SS-OCT provide better image quality, less affected by media opacities and less susceptible to artifacts and centering errors. Wide-field scanning has been shown to be effective at discriminating between healthy and glaucomatous eyes with a diagnostic accuracy comparable to spectral-domain OCT (SD-OCT) [18]. SS-OCT enables imaging of lamina cribrosa (presumed to be the primary site of axonal injury) and may assist in understanding of the pathogenesis of glaucoma.

Optical coherence tomography angiography

There is a possible role of reduced optic nerve head perfusion and vascular dysregulation in glaucoma[19]. Until recently, it was difficult to study the micro vascular structure of the retina and optic nerve head (ONH) due to the resolution limitations of fluorescein angiography. Optical coherence tomography angiography (OCT-A) is a new technology that allows non-invasive visualization of the microvasculature of the eye without the use of contrast dye. The technique takes advantage of improvements in OCT image resolution and scanning speed. OCT-A is able to detect change which are attributed to erythrocyte movement in perfused vessels, by comparing sequential scans at the same location. The technique have multiple advantages over traditional angiography. It includes the ability to simultaneously assess the retinal and choroidal circulations, quantitative assessment of the microcirculation, and three-dimensional assessment of both microvasculature structure and function while avoiding the need for invasive dye injections. So OCT-A is seems to be a good tool for glaucoma detection and monitoring.

OCT – A provides quantitative information on both blood vessel structure and microvascular function. Blood vessel structure reported as blood vessel density (Fig. 6) and foveal avascular zone area. Microvasculature function reported using flow index, a dimensionless parameter between 0 and 1. Differences between healthy and glaucomatous eyes have been observed with respect to vessel density, foveal avascular area, and reduced blood flow index. In patients with ocular hypertension, normal tension glaucoma, and primary open angle glaucoma reductions in vessel density and size of the foveal avascular zone have been reported[20]. These changes are associated with a reduction in optic disc flow and correlate with the degree of visual field defect. The measurements have been shown to have high repeatability and reproducibility. Studies have found the reduction in optic disc flow between healthy and glaucomatous eyes and this reduction correlated strongly with visual field pattern standard deviation (PSD) [21]. OCT – A finding shows association not only with the degree of visual field defect but also the location of the field defect. Changes in OCT-A parameters also correlate with the location of RNFL thinning.

OCT – A is not without limitations. Currently, there is a lack of comparability between machines and studies due to an absence of standardized measurement protocols. Also, image quality is highly dependent on fixation and patient co-operation. Further longitudinal studies are needed to determine whether OCT-A findings can predict or detect glaucoma progression. Nonetheless, OCT-A remains a promising technology for elucidating the physiology of glaucoma and evaluating structure and function in this disease.

Although RNFL thickness measurements tend to detect glaucoma better than OCT-A measurements, visual field defects correlate better with OCT-A measurements. In one study, the improved correlation of OCT-A and perimetry was especially apparent in advanced glaucoma, where RNFL thickness measurements reach a floor effect earlier than OCT-A.

Artificial intelligence

Artificial intelligence (AI) is revolutionizing almost every field and glaucoma not remain untouched.AI based tool is being developed to analyse and categorize data from testing, including fundus photos, perimetry and OCT. Although this technology is in its primitive stage but seems to have clinical role in future[22].

Conclusion

Glaucoma remains a common and important cause of irreversible visual impairment. Quality of life is grossly affected of glaucoma patients. Since the loss due to glaucoma is irreversible, so early detection and timely diagnosing the disease is utmost importance. This we can achieve by using newer and advance diagnostic technologies (of course with clinical sense) that will ensure continued improvements in glaucoma detection and hence management.

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RECENT ADVANCES

Recent Advances in Medical Management of Glaucoma

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The burden of irreversible vision loss from Glaucoma continues to rise. While the disease pathogenesis not well understood, intraocular pressure (IOP) is the only modifiable risk factor identified to prevent glaucomatous vision loss. Medical management remains the first-line of treatment in most adult glaucomas and the evolution of medical therapy for glaucoma has followed an exponential curve.So many drugs for the management of glaucoma the newer medicine are ..

Rho Kinase Inhibitors

Rho kinase inhibitors increase aqueous outflow and decrease outflow resistance by increasing the ability of the Schlemm's canal endothelial cells to form pores. Another hypothesis is that Rho kinase inhibitors cause relaxation of the smooth muscle fibers in the trabecular meshwork and there by increase outflow .Experimental evidence also supports changes in Schlemm's canal cytoskeleton causing decrease infocal adhesions in the juxtacanalicular meshwork. Ripasudil and netarsudil are the two commercially available formulations of Rho kinase inhibitors, both of which work on ROCK1 and ROCK 2 receptors. The most commonly reported adverse events included conjunctival hyperemia (76%), blepharitis (21%), Netarsudil is a Rho kinase inhibitor and nor-epinephrine transporter inhibitor which decreases IOP by decreasing the outflow resistance. Usually Netarsudil used once in night. The value of Rho kinase inhibitors as an adjunctive therapy is significant because the mechanism of action is different from that of the currently used medications. The side-effect profile with a relatively high incidence of conjunctival hyperemia and subconjunctival hemorrhages, however, may prove to be a deterrent to long-term compliance with these medications. The safety and efficacy of Rhokinase inhibitors in individuals While most studies have reported on the role of Rho kinase inhibitors in treating patients with OHT or POAG, the role of Rho kinase inhibitors in the treatment of different types of glaucoma needs further investigation. A prospective observational study of ripasudil found statistically significant drop in IOP in patients with POAG, uveitic glaucoma, and steroid induced glaucoma but not neovasular glaucoma. Other potential areas of investigation include the role of Rho kinase in neuroprotection via increased blood flow to the optic nerve and its proposed role in preventing postsurgical scarring by inhibiting TGF-β-mediated activation of fibroblasts.

Latanoprostene Bunod

Latanoprostene bunod 0.024% (LBN) is a uniquenitric oxide (NO) donating Prostaglandin F2 alpha analogue. BN metabolizes into the prostaglandin analogue, latanoprost acid, and butanediol mononitrate; butanediol ononitrate further metabolizes into 1,4 butane diol and NO. Latanoprost acid and NO are the two active etabolites Latanoprost acid binds to the Prostaglandin F receptor and increases the uveoscleral outflow by matrix metalloproteinases-mediated remodelling of the extracellular matrix of the ciliary muscle.

In the eye, NO synthetases are present in the Schlemm's canal, trabecular meshwork, and ciliary body. NO causes

vasodilation and smooth muscle cell relaxation. It decreases cell contractility and volume, thereby increasing trabecular outflow. LBN is thus a single molecule that provides two active metabolites that work through two different pathways for reducing intra ocular pressure.

More evidence is awaited on the role of LBN as adjunctive therapy. A recent retrospective study on patients on netarsudil and LBN as adjuvant therapy concluded that both showed similar efficacy as when used in monotherapy. LBN is currently dosed once daily at bedtime. The safety profile of the drug in pregnancy and lactation has not yet been established.

Newer Drug Delivery Systems

Medication noncompliance is a significant challenge for glaucoma patients who commonly complain of difficulty while instilling drops and difficulty in adhering to complex eye drop administration schedules. In an attempt to ease chronic medication use, new sustained drug delivery systems have been developed in the past two decades. Ocusert was the first sustained pilocarpine implant introduced in 1975, but the product was soon taken off the market because of poor medication tolerability. The bimatoprost implant (Durysta™) is a sustained release, biodegradable implant that uses the NOVADUR drug delivery system for intracameral use. The implant is administered into anterior chamber using a 28 gauge, single-use, prefilled applicator. The drug delivery system is made of biodegradable polymers that disintegrate by hydrolysis into carbon dioxide]
Another sustained release application is the bimatoprost ocular ring (BIM ring) which is a silicone and polypropylene ring impregnated with bimatoprost, available in diameters ranging from 24 to 29 mm, designed for insertion between the upper and lower fornices. It continuously elutes bimatoprost for a period of 6 months, after which it needs to be replaced. The rate of drug elution decreases with time, ranging from 35 µg per day on the day of insertion to 6 µg per day at 6 months. IOP control over 6 months was found to be comparable to 0.03% bimatoprost topical drops with the main adverse effect being mucinous discharge from the eye in some patients.

Travoprost punctum plugs:- Travoprost impregnated in polyethelene glycol resorbable hydrogel rod is inserted into the upper or lower punctum. Within the hydrogel rod, travoprost particles are encapsulated in polyactic acid microparticles, which hydrolyze with time to provide a sustained delivery of travoprost over 90 days. The rod is also impregnated with fluorescein to aid visualization.

Investigational Glaucoma Medications

Cannabinoids

Cannabinoids are derived from the cannabis plant (phytocannabinoids) or are artificially produced (synthetic cannabinoids). They interact with cannabinoid receptors 1 and 2 in the human body (CB1 and CB2), which are the natural receptors for endocannabinoids which modulate pain, memory, and appetite. CB1 and CB2 are expressed in the human retina, ciliary body, iris, Schlemm's canal, trabecular meshwork, and the retinal pigment epithelium. The neuroprotective effect of cannabinoids is linked to the inhibition of glutamate release reported a Palmitoyl ethanolamide (PEA) is a congener of the endogenous cannabinoid, anandamide (AEA) that is cosynthesized with AEA in many human cells. It prolongs the action of AEA by competing with fatty acid amide hydrolase involved in the hydrolysis of AEA. The use of PEA in glaucoma was first reported by Gagliano in a cross-over study with a reduction in IOP of 6.2% after 2 months of treatment. Oral PEA was also effective in reducing the IOP spike post yttrium aluminum garnet laser iridotomy. Inhalational cannabinoids reportedly caused a 2.1 mm of Hg drop in IOP from baseline 80 min after administration of cigarettes containing 12 mg Delta 9 THC, but the IOP lowering effect was found to be linked with tolerance. Inhalational administration of Delta 9 THC led to higher IOP reduction compared to oral administration. However, the IOP reduction was noted to be short term with a significant decrease in IOP (4.1 ± 1.5 mmHg) at 30 min that peaked at 90 min (6.6 ± 1.5 mmHg). The most common side effect was a significant decrease in systolic and diastolic blood pressures resulting in postural hypotension. Topical cannabinoids have failed to demonstrate a significant effect on IOP in clinical trials. The

challenge with topical administration is the lipophilic nature of cannabinoids. Mineral oil, needed as vehicle for topical formulations,leads to poor penetration of the drug, lid inflammation, and conjunctival hyperemia. Topical formulations of THC with cyclodextrins, which are cyclic oligosaccharides with a central cavity that is hydrophobic to hold the drug molecule and an outer surface that is hydrophilic so as to allow water solubility, are undergoing animal studies. Albumin solubilized, intravenous Delta 9 THC caused a dose-dependent peak IOP lowering of 60%, but the effect was short lived. Hypotension and presyncopal episodes were the most commonly reported side effects. Despite extensive research, the role of cannabinoids in medical management of glaucoma remains equivocal. The relatively short-term effect on IOP, the risks of developing tachyphylaxis, and serious side effects impacting patients' general and neurocognitive health greatly outweigh the potential benefit at this time. Future research may provide stronger evidence for their use in neuroprotection with tolerable side effects

Adenosine receptor agonists

Adenosine is a nucleoside that activates the G protein linked to adenosine receptors, A1, A2A, A2B, and A23. It increases the conventional outflow facility by shrinkage of cell volume and remodeling of the extracellular matrix in human trabecular meshwork cells. A1, A2A, and A3 agonists are currently undergoing Phase 1 and 2 trials. Phase 2 trials of trabodenoson, a selective A1 agonist, showed clinically and statistically significant IOP reduction with no serious adverse events.

Prostanoid receptor agonist

Omidenepag isopropyl (OMDI) is a nonprostaglandin, selective, prostanoid EP2 receptor agonist, known to decrease IOP by increasing the conventional and uveoscleral outflow.. This drug is currently approved for use in Japan.

Small interference RNA

RNA interference is the cutting-edge technology of specific gene silencing, using small bits of RNA called small interference RNA (siRNA). SYL040012 is a siRNA developed to specifically silence the Beta 2 adrenergic receptor (ADRB2) at the ciliary body, thereby reducing the aqueous humor production.

Neuroprotection

Neuroprotection is the holy grail of glaucoma care. Glaucoma is known to be a neurodegenerative disease which causes chronic progressive RGC death, and glaucoma treatment remains restricted to reduction in IOP at this time. Lowering IOP removes a stressor for neuropathy and arguably is a form of neuroprotection. The search for non-IOP-dependent neuroprotection is ongoing. Though a consensus on the actual cause of glaucomatous optic neuropathy is awaited, the cellular processes that cause RGC death include exposure to neurotoxic substances like NO and glutamate, deprivation of internal trophic factors, loss of cellular self-repair process, and intracellular destructive process.

Memantine

Elevated levels of glutamate are toxic to retinal ganglion cells and the resulting cell death is mediated by excitotoxicity of the N-methyl-D-aspartate (NMDA) receptor, by causing an excess of intracellular calcium and cell death.Memantine is an NMDA receptor antagonist and can prevent cell death by calcium influx

Neurotrophins

Neurotrophic factors play a key role in cell survival. Brain-derived neurotrophic factor (BDNF), ciliary neurotrophic factor, glial cell-line-derived neurotrophic factor and nerve growth factor (NGF) are potential candidates in neuroprotection undergoing preclinical studies. Valproic acid, traditionally used to treat epilepsy, has been demonstrated to induce neuroprotection by stimulating the BDNF–TrkB pathway. Topical NGF drops have also been shown to demonstrate improvement in visual fields, contrast sensitivity, and electrofunctional tests in a few patients with advanced glaucoma.Obstacles in the safe administration of these molecules at the intended site of action, poor understanding of pharmacokinetics, and lack of clarity on the long-term effects of these agents

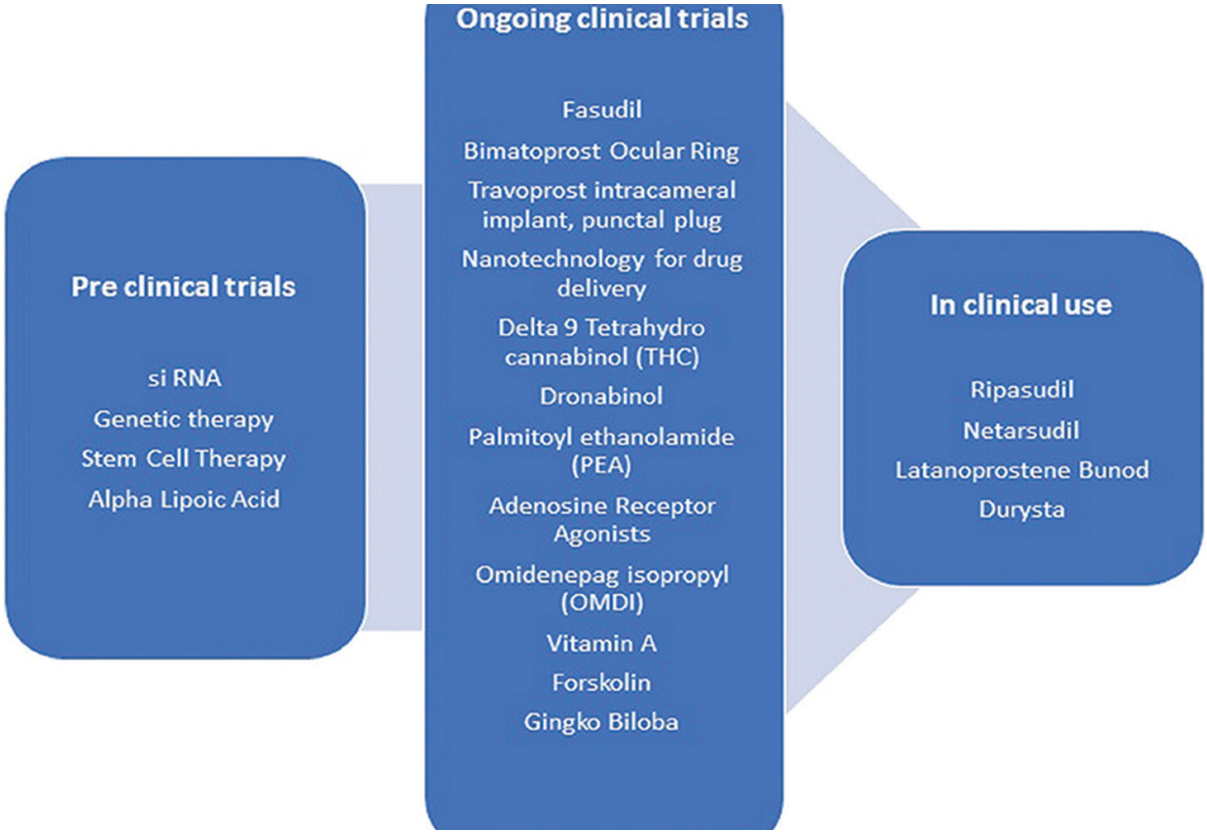
remain challenges in the translation to human trial.

Gene therapy

Gene therapy for glaucoma is still in the early stages of research. The large number of chromosome loci responsible for POAG, challenges in gene transfer with final binding at the intended site, and the possibility of mutagenesis have all dampened progress of this mode of treatment. Aquaporin 1 is a protein in the ciliary body involved in aqueous production by facilitating the transmembrane transport of water. Disruption of Aquaporin 1 by gene therapy with CRISPR-Cas9 RNA has been reported to reduce IOP in animal models.The treatment which targets a gene involved in a physiologic process rather than a specific gene mutation has the potential to be universally applicable. Intravitreal injection of AAV2 vectors increased the production of BDNF and increased duration of action of BDNF by upregulating tropomyosin-related receptor kinase B.

Stem cell therapy

Traditional glaucoma treatment modalities aim to delay or arrest the progression of glaucoma. Stem cell therapy provides the captivating possibility of regenerating and repopulating RGCs and possibly restoring vision lost from glaucoma. Preclinical studies have validated that mesenchymal stem cells secrete neurotrophins which promote cell survival and can repopulate RGCs in the retina. Stem cell therapy may also play a role in cell-based The past few decades have opened up multiple new horizons in glaucoma treatment. With the pace and scale of ongoing research, we have reason to look forward to newer medications, delivery systems, and novel therapeutic modalities being available for patient care.



CASE REPORT

Bilateral sequential manual small incision cataract surgery as the management of phacomorphic glaucoma in a patient with Werner's sydrome.

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Abstract
Werner's syndrome is a rare progressive hereditary disorder characterized by the accelerated aging. Bilateral cataracts are one of the cardinal signs of Werner syndrome. The clinical characteristics of our patient met the diagnostic criteria of "Probable WS" according to the International Registry of Werner Syndrome.Our patient presented with bilateral phacomorphic glaucoma. In previous Case reports of WS cataract surgery has been done by clear corneal phacoemulsification. Our patient underwent bilateral sequential manual small incision cataract surgery with intraocular lens implantation. We highlight the fact that bilateral cataract surgery with MSICS for phacomorphic glaucoma is a safe alternative for patients with Werner's syndrome.
Key-words: Phacomorphic glaucoma, Werner's syndrome, MSICS

Introduction
Werner syndrome (WS) is a rare progressive Genetic disorder. Unusually accelerated aging (progeria) Characterise Werner Syndrome that is typically recognized by the third or fourth decades of life.[1] Bilateral cataracts are one of the cardinal signs of Werner syndrome. We report the successful management of a Werner Syndrome patient who presented with bilateral phacomorphic glaucoma.

Case History
A 33-years old patient presented with complaints of diminution of vision in both eyes (BE) for the past 15 days which was insidious in onset and painful in nature .The patient had associated mental retardation. On general physical examination, the patient was 143 cms tall and weighed 30 kgs. He had a "old man look", with a bird-like face, pinched nose, alopecia, greying of beard, thin extremities with atrophied, hyperpigmented skin and decreased subcutaneous fat. He had a high pitched voice and with loss of dentition. (Figure1)
On ocular examination his vision had light perception with inaccurate projection of light in both eyes. The anterior segment examination showed bilateral circumciliary congestion with corneal edema and a shallow anterior chamber. The pupils were fixed, mid-dilated and sluggishly reacting to light. In addition the right eye also showed iris had atrophic patches with posterior synechiae. Both eyes had intumescent mature cataracts with no view to the fundus. IOP by Goldmann Applanation Tonometer in Right Eye was 44 mmHg and left eye was 32 mmHg.No angle structures were visible on gonioscopy with four mirror Sussman gonioprism on indentation. (Figure 2).

The characteristics of our patient (as described above) met the diagnostic criteria of "Probable WS" according

to the International Registry of Werner Syndrome. (Table 1) Based on the ocular examination a provisional diagnosis of bilateral phacomorphic glaucoma was made. The Patient was admitted for treatment with a plan for cataract surgery with iol implantation once the cornea oedema resolves.
Preoperative preparation with Intravenous injection of mannitol 20% w/v 100ml was given over 45 minutes on admission and one hour before surgery to control the IOP. Tablet Acetazolamide 125 mg QID, Tablet Ranitidine 75 mg BID. Topical Timolol eye drops continued BID. We were unable to perform specular microscopy due to the presence of nystagmus.

The patient underwent immediate bilateral sequential cataract surgery. Manual small incision cataract surgery with intraocular lens implantation was done. Superior scleral tunnel of 6 mm length was made with frown incision. High molecular weight OVD (Sodium hyaluronate 1.4%) was used to protect the corneal endothelium during surgery. Continuous curvilinear capsulorhexis was done. Nucleus was delivered by irrigating wire vectis. Irrigation & aspiration was done using 21 G simcoe cannula through the side port. Rigid single piece PMMA PCIOL was put in the bag. No sutures were used for the tunnel or the side port. Injection dexamethasone (4 mg/ml) 0.5 ml was given subconjunctivally at the end of surgery.

Post-operatively the patient was put on Moxifloxacin 0.5% eye drops 6 times per day for 2 weeks, Prednisolone 1% eye drops 1 hourly for first day and then tapering doses for one month and Homatropine 2 % eye drops TDS for 1 week.

On the first post-op day the patient had a UCVA of 6/60 in his right eye and 6/36 in his left eye. The scleral tunnel wounds were well opposed. Fundus of Both eyes showed 0.6:1 cup-disc ratio, healthy neuro-retinal rim, macula was normal in both eyes. IOP by Ocular response analyzer was 20 mmHg in the Right Eye and 16 mmHg in the left eye.

Category	Werner syndrome signs and symptoms
Cardinal signs	Cataract (bilateral)
	Bird like facies
	Tight / atrophic /hyperpigmented skin
	Short stature
	Parental consanguinity
	Premature greying / thinning of scalp hair
Additional signs	Diabetes mellitus
	Hypogonadism
	Osteoporosis
	Osteosclerosis of distal phalanges
	Soft tissue calcification
	Premature atherosclerosis
	Mesenchymal neoplasms
	Voice changes
	Flat feet
Diagnostic criteria	
Definite	All cardinal signs +two additional signs
	Conformed mutation of both allele of WRN gene
Probable	First three cardinal sighs + any two additional signs
Possible	Either cataract or dermatological changes+ any four additional signs

Table 1 Adapted from The International Registry of WS (www.wernersyndrome.org)

Discussion

Werner syndrome is a rare progressive hereditary disorder characterized by the appearance of unusual and accelerated aging. In most cases the inheritance is autosomal recessive but sporadic cases have also been reported. Although the disorder is typically recognized by the third or fourth decades of life, certain characteristic findings are present during adolescence and early adulthood.[1] Affected individuals have short stature and low weight relative to height. By age 25, individuals with the disorder typically experience early greying and alopecia also cataract develops at a relatively younger age in these patients.

It has a global incidence rate of less than 1 in 100,000 live births; only a handful of cases of Werner Syndrome have been reported till date. To the best of our knowledge there is no existing literature on the safety of M-SICS in eyes with werner's syndrome . We report the outcomes of M-SICS in eyes with werner syndrome presenting with phacomorphic glaucoma

Case reports of cataract surgery by clear corneal phacoemulsification in WS. The corneal tunnel was sutured with nylon suture in the reported cases.[5, 6] Kusumesh *et al* has reported a case of hypermature cataract successfully managed with clear corneal phacoemulsification.[6] In our patient the presence of intumescent lens made phacoemulsification challenging considering the associated risk of rhexis run away and intra operative endothelial damage.

In our case, M-SICS permitted atraumatic extraction of the nucleus with good postoperative with well apposed scleral tunnels and stable wound postoperatively, also there was no corneal edema suggestive of minimal intraoperative endothelial damage.

We highlight the fact that immediate sequential bilateral cataract surgery with MSICS for phacomorphic glaucoma is a safe alternative for patients with werner syndrome.

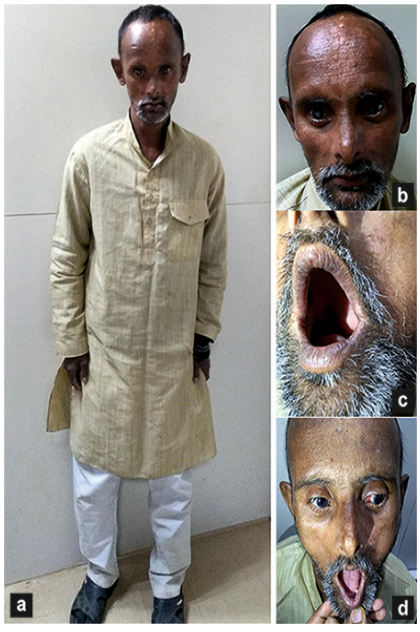

Figure 1

Figure 1: (1a) Physical appearance of the patienacial appearance of the patient. (1b) Note the bird like face, pinched nose, greying of eyebrows, moustache and beard, alopecia. (1c) and (1d) Showing loss of teeth except one pre-molar of mandible. (Picture taken post cataract surgery)

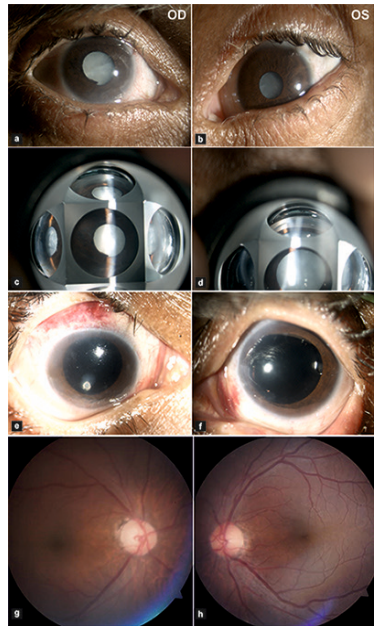

Figure 2: Pre-operative and Post-operative image (a) Right eye showing corneal haze, posterior synechiae inferiorly and mature cataract. (b) Left eye showing mature cataract, (c) Gonioscopy of right eye and (d) left eye showing closed angle (e) Right eye and (f) left eye on post op day one showing well apposed scleral tunnel wound, clear cornea and PCIOL in bag, (g) and (h) Post-operative fundus photos showing Glaucomatous changes

Figure 2

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CASE REPORT

Klippel - Trenaunay syndrome (KTS) - A rare case report

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Introduction -

Klippel-Trenaunay Syndrome (KTS) is a rare mesodermal phakomatosis with a triad of irregular and asymmetrical capillary port-wine stain and cavernous hemangioma on the trunk or limbs, venous varicosities, and asymmetrical hypertrophy of bone or soft tissue . Sturge-Weber syndrome (SWS) and KTS have overlap in many of the diagnostic criteria ,but there are important differences among these diseases that carry important prognostic and therapeutic implications.

Case Report -

We present a case of KTS in 10 year female child who presented with complains of ocular pain in the both eyes and headache since birth. Physical examination revealed macrocephaly , reddish discolouration on whole face but more on left side (naevus flammus) and , hypertrophied right limb with non healing ulcer, since 1 year . On detailed ophthalmological examination-BCVA OU was 6/18 , episcleral bluish hue with megalocornea (13.5 mm and 14 mm) , IOP (GAT) – OD 22 mm Hg , OS 24 mm Hg with open iridocorneal angle on gonioscopy , and fundus examination - OD with in normal and OS showed tortuous blood vessels in posterior pole On MRI Brain with angiogram shows multiple tram track calcification in left frontal and occipitoparietal cortex with brain atrophy, ipsilateral choroid plexus enlargement with calcification and calvarial thickening.

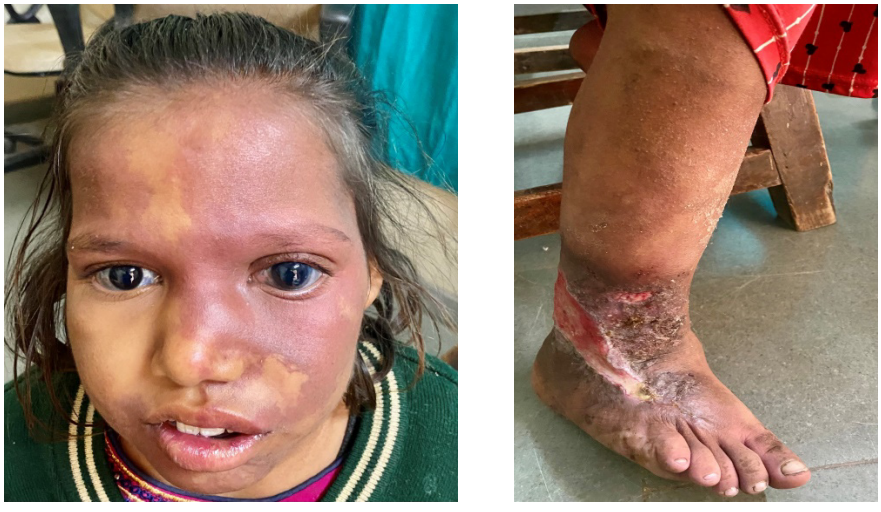

Figure - showing Facial, Ocular and Limb findings

Patient had history of convulsions since birth and was on regular anticonvulsant treatment. Developmental milestones were achived . On USG A scan ,axial length of OD and OS were 22.31 mm and 26.98 mm respectively .

Patient was started on E/d Timolol 0.5% in twice daily doses . At 3 week s followup the IOP was OU 16 mm hg with relief of ocular pain . For leg ulcer and hypertrophy Local wound care, compression dressings, special orthopaedic footwear (heel inserts) and lifestyle modification was suggested in association with pediatrician and orthopedician.

Discussion -

Klippel-Tranuanay Syndrome is a rare sporadic disease characterized by clinical triad of capillary malformation; soft tissue and bony hypertrophy; and atypical varicosity. Although this syndrome was first described more than hundred years ago, exact incidence has not been estimated yet as it is many times wrongly diagnosed as Sturge Weber Syndrome. Diagnosis of this embryological malformation is purely clinical, which plays a pivotal role in its management.

A multidisciplinary approach to treatment and prevention of possible complications of KTS including paediatrician, internist, phlebologist, paediatric, orthopaedic, plastic and vascular surgeons, an interventional radiologist, cardiologist or vascular internist and a physical therapy physician provides optimal care for the patient.

Prevention of venous thrombo-embolism with anticoagulation or inferior vena cava filter is as important as prevention of repeated episodes of cellulitis and lymphangitis in those with associated lymphoedema or as treatment of the symptomatic vascular anomalies.The absolute indications of treatment are haemorrhage, infections, acute thromboembolism or refractory ulcers.

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CASE REPORT

Bilateral Posterior Scleritis Presenting as Angle Closure Glaucoma

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Abstract:
A-52-year-old female patient presented with 1-week history of ocular pain and redness in both the eyes. On examination, intra-ocular pressure was 22mmHg and 24mmHg in the right and left eye respectively. Gonioscopy revealed closed angles in both the eyes. There was no anterior chamber inflammation. Undilated fundus examination revealed normal fundus in right eye while glaucomatous changes were seen in left eye. Accordingly, right eye was diagnosed as primary angle closure (PAC) and left eye was diagnosed as primary angle closure glaucoma (PACG). Both laser peripheral iridotomy (LPI) was done and ocular hypotensive drugs were prescribed. Further imaging examinations (i.e., ultrasonography and fundus fluorescein angiography, FFA) indicated a diagnosis of bilateral posterior scleritis. Oral prednisolone along with intraocular pressure-lowering agents were prescribed. She was Anti ds DNA positive and advised to take rheumatologist opinion for the same.

Introduction:

Posterior scleritis is inflammation of the sclera, posterior to the insertion of the rectus muscle. [1,2] It is the rarest form of scleritis, and diagnosis is often difficult due to its rarity and varied clinical presentation. It should be considered in the differential diagnosis of exudative retinal detachment, choroidal detachment, circumscribed fundus mass, choroidal folds, optic disc edema and angle closure glaucoma.[1] The diagnosis is usually made on detecting thickened sclera (≥ 2mm) on B-scan ultrasonography.[3]

The incidence of raised intra-ocular pressure (IOP) in posterior scleritis ranges from 7 to 12.0%. [3,4] Previously, posterior scleritis has been reported to present as angle closure, open angle glaucoma or ocular hypertension. [5-10] We report a patient of posterior scleritis who was initially diagnosed as primary angle closure glaucoma, and discuss the difficulties in the diagnosis of such vexing presentations.

Case report:

A 52-year-old female presented with chief complaints of pain and redness in both the eyes for the past 1 week, which was sudden in onset and progressive in nature. She had consulted elsewhere, and was diagnosed with glaucoma due to raised intra-ocular pressure, and was treated with a combination of Brimonidine tartrate 0.2 % and Timolol maleate 0.5 % eye drops twice daily. Her symptoms had reduced with the use of the eye drops. She had no significant personal or family history of glaucoma. She did not report any other systemic illnesses or symptoms.

On ocular examination, her best corrected visual acuity was 20/20p which improved to 20/20 with pinhole. Slit lamp examination showed mild conjunctival congestion and shallow anterior chamber (Grade I Van Herick) with no flare or cells in both eyes. Both the pupils were round, regular but sluggishly reacting to light, and the central part of the lens was clear. The intra-ocular pressure was 22 mmHg for the right eye and 24 mmHg for the left eye. On gonioscopy, no angle structures were visible in both the eyes. On indentation the angle in the right eye opened till scleral spur with no visible peripheral anterior synechiae, but did not open in

the left eye. Un-dilated fundus examination showed a normal optic disc in the right eye but there appeared to be glaucomatous changes in the left eye. The posterior pole was normal. The peripheral fundus was not seen.

On the basis of the above clinical examination, a diagnosis of primary angle closure in the right eye and primary angle closure glaucoma (PACG) in the left eye was made. Bilateral laser peripheral iridotomy was performed in both the eyes, and she was advised Brimonidine tartrate 0.2 % and Timolol maleate 0.5 % eye drop twice daily along with Prednisolone acetate 1% eye drop in tapering doses in both the eyes.

On her follow up visit after 2 weeks, the iridotomies were patent, and the IOP was 16mmHg in both the eyes. A dilated fundus examination revealed a cup disc ratio of 0.4:1 in both the eyes without any glaucomatous changes. However, there were there were multiple retinal folds seen in the peripheral fundus (Figure 1 and 2) in both the eyes. B-scan ultrasonography of the eyes revealed thickening of the posterior wall (2.20 mm in the right eye and 2.06 mm in the left eye), fluid under the Tenon's capsule, and pockets of fluids in suprachoroidal spaces (Figure 4). Axial length by optical biometry in the right eye was 20.37mm and 20.57mm in the left eye. Fundus fluorescein angiography of both the eyes revealed multiple pinpoint leaks in late phase with prominent choroidal folds in late phase in both the eyes (Figure 5). A revised diagnosis of posterior scleritis with secondary angle closure glaucoma was made. She was treated with topical and oral corticosteroids. Her blood reports that included ESR, ANA, c-ANCA, p-ANCA, and RA factor were normal except for anti-ds DNA, which was elevated. She was advised to take a rheumatologist opinion.

Discussion:

Several mechanisms have been postulated to cause glaucoma in scleritis.[1]These are ciliochoroidal effusion that rotates the iris-lens diaphragm forward closing the trabecular meshwork; clogging of the trabecular meshwork with inflammatory cells in the anterior chamber; extension of inflammatory cells into the ciliary body and trabecular meshwork; inflammatory cells cuffing of the episcleral vessels that increases the episcleral venous pressure; inflammatory cells cuffing of the trabecular meshwork; and secondary to use of topical steroids in treatment of scleritis. In our patient, the raised IOP and narrowing of the angle was probably due to all these reasons with the exception of inflammation in the anterior segment and steroid-response. Ultrasound biomicroscopic examination could have aided in determining the exact cause, however we did not have access to this diagnostic tool.

Our patient had presented with typical signs of primary angle closure suspect/glaucoma: bilateral presentation with shallow anterior chamber and closed angles of the anterior chamber. The optic disc in the left eye viewed through the un-dilated pupil appeared to be glaucomatous. There was no systemic complaints of joint pain or other symptoms suggestive of rheumatic disease, no anterior chamber inflammation, and the retinal folds were in the periphery, and hence were not detected through the un-dilated pupil. This led to the initial diagnosis of PACS/PACG and the laser iridotomy was done, which could have been avoided had we dilated the patient to examine the fundus. However, the risk of precipitating an acute angle closure attack, was higher than the perceived need for a dilated fundus examination. Our experience highlights the limitation of un-dilated fundus examination, and the need to consider rarer causes of secondary angle closure like posterior scleritis.

The symptoms in posterior scleritis are peri-ocular pain, blurred vision, headache, photophobia and floaters.[1,3,4,10] Pain may not be present in all patients and if present can range from mild to excruciating, referred to the brow, temple or zygoma, [1] and may be aggravated on eye movements. Ocular signs that may be present are conjunctival chemosis or congestion, choroidal folds, serous retinal detachment, anterior chamber cells and flare, macular edema, associated anterior scleritis, optic disc edema, vitreous cells, proptosis, sub-retinal mass and retinal vasculitis.[1-4,10] Sometimes no abnormalities may be present.[3] B-scan ultrasonography is a very useful tool for the diagnosis, and signs present include increased eye wall thickness (>2.0mm), scleral nodules, fluid in Tenon's capsule, swelling of optic disc, distended optic nerve sheath, and retinal detachment. [3] Although various systemic diseases like rheumatoid arthritis, systemic vasculitis, Wegener's granulomatosis, sarcoidosis, ocular tuberculosis, etc. may be present, often times, the cause may be idiopathic.[3,4,10] Sometimes antinuclear antibody may be the only positive finding, [10]as in our patient.

Conclusion:

Bilateral posterior scleritis should be among the differential diagnoses of acute primary angle closure. A high degree of suspicion is required to make the diagnosis of posterior scleritis. B-scan ultrasonography is very helpful in making the diagnosis.

Figure 1 Right eye fundus photo indicating retinal folds (yellow arrow)
Figure 2 Left eye fundus photo indicating retinal folds (yellow arrow)

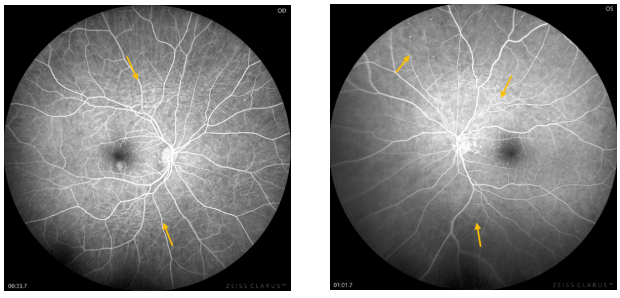

Figure 3 and 4 FFA image: late phase – indicating prominent retinal folds with multiple pinpoint leakage in the periphery

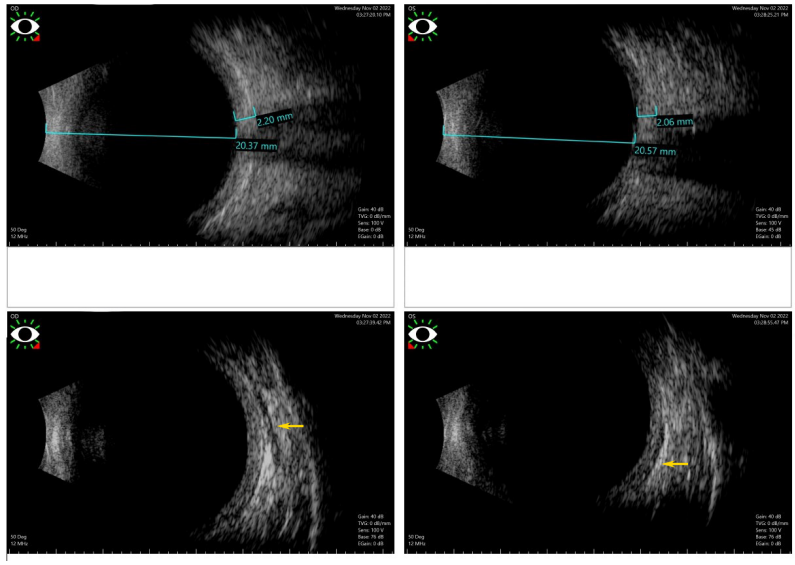

Figure 5 Both eye B-scan image showing pockets of fluids in subtenon's space (yellow arrow) with increased thickness of retina- choroidal complex (blue bracket)

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ONE MINUTE TIP IN GLAUCOMA

Calibration of Applanation Tonometry

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Goldman Tonometry is considered to be gold standard, most widely accepted and accurate method to measure IOP.

Calibration error of GAT is not uncommon. One can check the instrument for calibration error. This is done at the dial position 0, 2 and 6, (0, 20 and 60 mmHg equivalents) exact alignment of the marking is crucial. An error of 1 mmHg is acceptable at position 0 and 2, and of 2 mmHg at position 6.

Calibration needs to be checked every 6 months for new equipments and every 3 months for old equipments

1. Before you start
   - Insert the prism in the holder and place the tonometer on the slitlamp
2. Calibration at dial position 0
   - At setting 0, if the dial position is moved to -0.05 the feeler arm should fall towards the examiner; if the drum is moved the position +0.05 the arm should fall towards the patient.
3. Preparing to calibrate at dial position 2 and 6.
   - To check setting 2 and 6, the calibration error check weight bar provided by the manufacture is used .
   - The calibration error check weight bar has 5 marking on it. The central marking corresponds to level 0. Two on either side of it represent level 2 and the two outer most marking represent level 6. These marking correspond to 0, 20 and 60 mmHg of IOP respectively
   - The calibration error check weight bar and holder are fitted into the slot provided on the side of the applanation tonometer.
   - After setting the mark on the weight bar corresponding to position 2 or 6 on the index mark of the weight holder, the measuring drum is rotated forwards, the reading at which the feeler arm with the prism in place moves forward freely is recorded.
   - The difference of this reading from the respective test position is the positive error at that level of testing. Similarly, on rotating the revolving knob in the reverse direction, the reading at which the feeler arm moves backward is noted. The difference between the latter and the testing position is the negative error at the level of testing

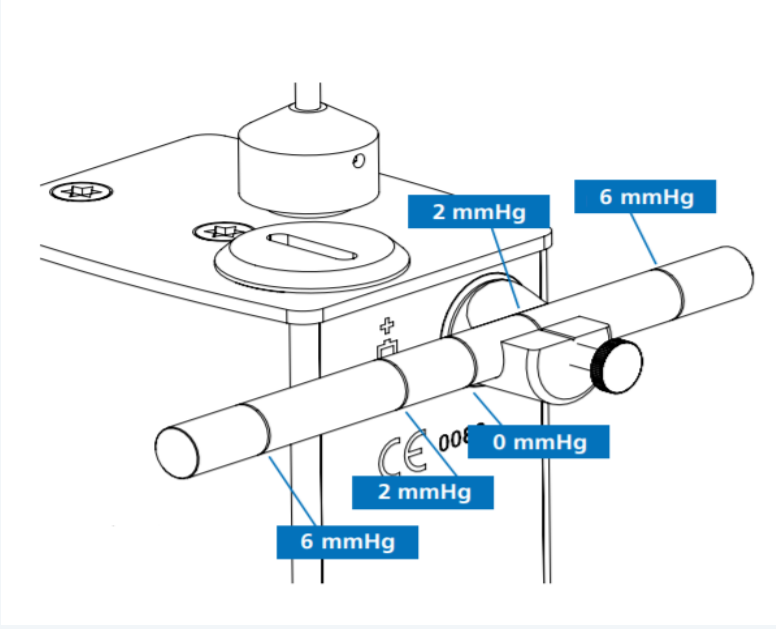
The manufacturers of Haag-streit GAT accept calibration errors within ± 0.5 mmHgat all levels of testing (0, 20 and 60 mmHg). On the other hand, the South East Asia Glaucoma Interest Group (SEAGIG) guidelines is less stringent and recommends that the acceptable range of calibration error should

progressively widen at the higher levels of error testing. By this guideline, the acceptable error could be within ± 2 mmHg at 0 mmHg, ± 3 mmHg at 20 mmHg and ± 4mmHg at 60 mmHg testing levels.

A simple way to test calibration,which should be used at the start of each clinic,is to check that the arm rocks around zero by moving the dial 0. 5 mmHg either side of zero with prism in place

If a GAT has unacceptable calibration error, the instruments should be sent to the manufacturer for rectification of the error.

One should avoid estimating true IOP from faulty GAT by applying the correction factor as the calibration error of GAT has high variability.



ONE MINUTE TIP IN GLAUCOMA

STERILIZATION OF GONIOLENS AND APPLANATION TONOPRISMS

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BACKGROUND
As an ophthalmologist, its our duty to keep up the trust of our attending patients by ensuring safe procedures and adequate treatment. Any procedure that involves exposure with the patient's ocular surface should be done and followed by utmost care to prevent iatrogenic spread of infection. The risk of iatrogenic infections can be minimised by following a routine quick sterilisation proceure of reusable medical equipments.
Gonioscopes and tonoprisms are vital tools for diagnosis and management of cases of glaucoma but are also potential tools for spread of infections. They have also been classified as semicritical instruments, devices that come in contact with intact mucous membranes or nonintact skin, per Spaulding, CDC[1].

What is the risk?
Nosocomial outbreaks are most commonly linked to adenovirus 8 and herpes simplex virus (HSV)
Other potential infectious agents:

- HIV
- Hepatitis C Virus (HCV)
- Enterovirus 70
- Pseudomonas Aeruginosa
- Methicillin-Resistant Staphylococcus Aureus (MRSA)
- Acanthamoeba
- Prions (Creutzfeldt-Jakob Disease)

IDEAL STERILISING AGENT SHOULD

- cover a broad antimicrobial spectrum
- act rapidly
- not damage the instrument
- be nontoxic to the user, patient & environment

COMMON CHEMICALS USED IN STERILIZATION

- 70% isopropyl alcohol & 70% ethyl alcohol germicidal
- 3% hydrogen peroxide germicidal effect is attributed to destructive hydroxyl free radicals
- Sodium hypochlorite (Household Bleach) used in 1:10 and 1:20 concentrations biocidal does not leave toxic residues is not affected by water hardness acts fast

METHOD OF STERILISATION
After examining each patient the gonioscope or tonoprism should be cleaned with mild soap and running water or using a wet wipe.

Followed by exposure to the sterilising agents as follows:

S.No.	Agent	Method	Effective against
1.	70% Isopropyl Alcohol Wipe	Just Clean With The Wipe	Adenovirus, HIV, HSV
2.	70% Isopropyl Alcohol Soak	Soak In The Solution For 15 Mins	Adenovirus, HIV, HSV, HCV
3.	3% Hydrogen Peroxide Wipe	Just Clean With The Wipe	Adenovirus, HIV
4.	3% Hydrogen Peroxide Soak	Soak In The Solution For 15 Mins	Adenovirus, HIV, HCV
5.	Sodium Hypochlorite Soak (1:10 Dilute Bleach) {Household Bleach}	15 Mins	Adenovirus, HSV
6.	Sodium Hypochlorite Soak (1:10 Dilute Bleach) {Household Bleach}	5 Mins 1 Min	Adenovirus

NOTE:

- After the soaks the instruments should be washed in 5ml sterile water and air dried thereafter.
- Keep the instrument in a dry, clean container afterwards
- It's important to always use a fresh solution for every cleaning procedure.
- With the goniolens positioned on its side, the entire lens should be immersed
- Avoid immersion in any fluid for more than 1 hour and to avoid temperatures of more than 60°C to prevent damage

DISCUSSION

Pepose et al[2] studied the effectiveness on HIV1, HSV1 and HSV2, of dry wipes, 70% isopropyl alcohol wipes or 3% hydrogen peroxide. It concluded that Sterile gauze and tissue wipes were not effective whereas both 70% isopropyl alcohol wipes and 3% hydrogen peroxide wipes were effective disinfectants for HIV-1, HSV 1 & HSV 2.

Nagington et al[3] tested efficacy of 0.05% sodium hypochlorite (1:20 dilute bleach), phenyl mercuric borate & isopropyl alcohol for elimination of adenovirus 8, enterovirus 70 and HSV 1 from tonometer tips.
Two minutes in 0.05% sodium hypochlorite rendered adenovirus 8, enterovirus
70, and HSV 1 undetectable. Isopropyl alcohol was found only effective against HSV 1.

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ONE MINUTE TIP IN GLAUCOMA

An Overview of Medical Management of Glaucoma

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Glaucoma is the second leading cause of blindness is India and worldwide next to cataract. Blindness due to glaucoma is irreversible, so it is important to diagnose it early and manage the disease properly. Most patients are asymptomatic till advanced stage is reached. In addition, there is a significant issue of under and over diagnosis up to 50%. Lack of medical facility, ignorance, illiteracy and poor socio-economic support complicate the gravity of situation in Indian scenario. Medical management is the standard of care in glaucoma. Before initiating treatment, a target IOP range should be established for each patient. Medical treatment of glaucoma should start with the simplest, most effective drug regimen and modify it upon patient's response to treatment.

Considerations on first-choice of treatments:

First choice of treatment is different from first line of treatment. It is the best treatment which is suitable for a given patient. It needs to be individualized for every patient keeping in mind the following.

Safety:

1.Systemic: Ask for specific diseases (e.g., asthma and arrhythmia), exclude allergies, and note concomitant medications.

2.Local: OSD and other ocular diseases & then choose the medication.
Choosing the most appropriate medication

- Greatest chance of reaching target IOP.
- Best safety and tolerability profiles.
- Minimal inconvenience.
- Affordable.
- Maximal likelihood of adherence.

Patient and family education must include information about the disease, but also about medication regimen and the cost. It is advisable to teach patients and their relatives the technique for eye drop instillation by demonstrating the preferred method, including punctal occlusion and eyelid closure for at least three minutes Double DOT technique. "don't open the eyelid" and "digital occlusion of the tear duct .”Educational materials must be given for reading at home.

Least complex regimen: Start low and slow, at the minimal concentration and minimal frequency. Least disruption to lifestyle. Establishing reminder systems (e.g., cell phone-based alarms) significantly improve adherence.

Monotherapy

Monotherapy should reduce the IOP at least 10-15% from baseline, otherwise the patient can be labelled a non-responder to the medication. If IOP reduction is less than 15%, a switch to another class can be made. The most common first-choice monotherapy is PGAs, followed by beta blockers, CAIs, alpha agonists, and others. The table 1 summarizes the most common antiglaucoma drugs and their characteristics.

The characteristics of an ideal IOP-lowering eye drop include:

- Proven efficacy in reducing IOP consistently over a 24-hour period.
- Minimal local and systemic adverse effects.
- No tachyphylaxis and good tolerance over time.
- Minimal frequency of dosage.
- Applicability in diverse patient populations.

Adjunctive Therapy:

If the target IOP is not achieved another drug which has a different mode of action can be added. The disadvantages of adjunct therapy is reduced compliance, added cost , increase in side effect and increase in dryness of eyes

Fixed –dose combinations

Given the issues presented by adjunctive therapy, fixed-dose combinations are thus preferable when available. The efficacy is usually clinically equivalent to unfixed combinations. Although combination drugs provide many advantages. They reduce the cost, increase the compliance and adherence, less load of preservative so reduces the chances of OSD.

KEY POINTS

1. PGAs are recommended as first-choice agents for most eyes with glaucoma. IOP reduction with initial monotherapy should be at least 20% from baseline.

2. IOP reduction of less than 10% should be considered as non-response. When using PGAs, switching drugs within the PGA class may, upon occasion, provide greater IOP lowering.

3. Adjunctive therapy is indicated when existing therapy fails to reach the target IOP. Adjunctive therapy should be limited to one drug from each class.

4. Fixed combinations are preferred, when possible, over the use of two separate bottles due to convenience, reduced amount of preservative instillation, and possible improved adherence.

5. Surgery is indicated when medical therapy fails to adequately lower IOP or prevent progression, the risk of progression remains too high despite the use of medical therapy, or is not possible due to allergy, intolerance, poor adherence or lack of availability.

6. PGAs are the first choice for monotherapy in PXFG, PXF syndrome, and pigmentary glaucoma as well as in OHT when treatment is required. They are also useful in angle closure glaucoma management after laser PG is done.

7. Poor adherence and perseverance are major problems in glaucoma, Patient self-reporting of adherence is often overestimated.

8. The most common risk factors for lower adherence rates includes younger and older age, financial problems, inadequate under standing of disease and its consequences, alternative medicines, inability to put drops due to Parkinsonism and lack of family /social support and nonavailability of medication.

9.During pregnancy, PGAS may be associated with uterine contraction. Beta blockers and alpha agonist in cause serious toxicity (respiratory and central nervous system depression) When possible, these agents should be withdrawn during the last few weeks of pregnancy. Topical CAIs are generally well tolerated.

10.Spending sufficient time with patient, family and making them understand the disease and its consequences will significantly improve the outcome of disease.

11. Surgery should be the choice of treatment in patients who have moderate to advance glaucoma, patients in younger age group with moderate to high presenting IOP, fast progressing disease, high diurnal variation in IOP, patients who have poor access to health care and in patients, who are less likely to adhere to the treatment.

Table 1

Drug class	Mechanism of action	Duration of action	Systemic side effects	Local side effects	Peak effect and wash-out period
Beta Blockers	Decrease aqueous production	12 hours	Bradycardia Hypotension Asthma Bronchospasm Dyspnea Impotence Insomnia Hypoglycaemia	Allergic blepharoconjunctivitis Dry eye Corneal anesthesia	PE: 4-6 weeks Wash off : 4-6 weeks
Miotics	Increase Trabecular outflow by constricting longitudinal ciliary body muscle and opening TM	6-8 hours	Increased sweating Salivation Bradycardia	Miosis ,accommodative Spasm, Iris cysts Anterior subcapsular lens opacities Lacrimation	PE: Within 3 hours Wash off : 1week
Adrenergic (Adrenaline and dipivefrin)	Decrease aqueous production and increase outflow facility	8 hours	Headache Nervousness Tachycardia Arrythmia Hypertension Dry mouth Drowsiness	Mydriasis Lid retraction Adreanochrome deposits Allergic follicular conjunctivitis CME in aphakia	PE: 2 weeks (stabilizes at 6 weeks)
Alpha -2 agonists	Decrease aqueous production	8 hours	Drowsiness Headache, Dry Mouth, High Levels of fatigue, Crosses blood – brain barrier Absolutely contraindicated in patient using MAO inhibitor & children below 10 yrs of age	Allergic conjunctivitis	PE : 2weeks(stabilizes at 6 weeks) Wash off : 4-6 weeks
CAIs	Decrease aqueous production	6-8 hours	Fatigue Malaise Paresthesia of fingers and toes cramps Diarrhea Nephrolithiasis (calcium oxalate and calcium phosphate), Renal failure Acute leucopenia Agranulocytosis Aplastic anemia Hypokalemia Metabolic acidosis Stevens – Johnson syndrome	Conjunctival hyperemia .Allergic reactions, Blepharitis Burning/ stinging sensation, corneal edema in patients with compromised endothelium (e.g., subclinical Fuchs' dystrophy, post-surgical changes)	PE: 24-48 hours Wash off: 1-2 weeks
PGAs	Increase uveoscleral outflow	24 hours	Skin rash Skin Pigmentation Iris ,hyperchromia and eye lash growth and thickening	Herpetic keratitis reactivation CME in psedophakics and aphakics	PE : 1-2 weeks, Wash off : 2-3 weeks
Hyperosmotic agents (mannitol and oral glycerol)	Increase blood osmolality, dehydrates the vitreous	8 hours	Caution in patients with cardiac,renal, and hepatic disease. Nausea and vomiting Circulatory overload, CCF Pulmonary Oedema Hyponatremia Dehydration Metabolic acidosis with poor renal function		Mannitol PE : Within 1 hour
Rhokinase Inhibitors	Decreases aqueous production, increases outflow and decreases episcleral venous pressure.	8 to 12 hrs	Conj hyperemia, subconj haemorrhage		PE : 1-2 weeks, Wash off : 2-3 weeks

ONE MINUTE TIP IN GLAUCOMA

Glaucoma Medication In Pregnancy and Lactation

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INTRODUCTION

Glaucoma is more common among patients older than 40 years, but we may come across younger female patients with pre-existing glaucoma such as congenital, juvenile or secondary glaucoma. Also, primary open-angle glaucoma might be present in women who have a delayed pregnancy into their 40s. These female patients pose a complex challenge since the benefits of therapy need to be weighed against the potential risks to the foetus and the neonate. The issue is further complicated by lack of evidence-based recommendations due to obvious ethical considerations.

EPIDEMIOLOGY

Data regarding glaucoma prevalence in subjects in women of childbearing age is scant. One Japanese study reported the prevalence of open angle glaucoma as 0.48%, 0.42%, and 0.73% among women aged 15-24, 25-34 and 35-44, respectively.[1]

GLAUCOMA IN PREGNANCY

Intraocular pressure (IOP) typically decreases during pregnancy and mechanisms postulated for this include greater aqueous outflow facility due to hormonal changes, decreased episcleral venous pressure, and metabolic acidosis resulting from gestation.[2,3] The hyper oestrogenemic state of pregnancy is associated with a 10 % IOP reduction [4] which is more marked in the second and third trimesters. However, despite this theoretical hormonal protecting factor, glaucoma evolution during pregnancy remains variable, thus necessitating the need for close monitoring of these patients.

MANAGEMENT

Glaucoma management in pregnancy should be considered within the frameworks of preconception, pregnancy, and lactation.

Before conception: The potentially serious side effects of anti-glaucoma medications on the foetus, depend upon the stage of embryogenesis during which it is exposed. Hence, it becomes important to ask about pregnancy or plans to become pregnant when evaluating glaucoma in any female patient of childbearing age. If the patient is already on glaucoma treatment, she needs to be informed that her medication regimen may change once conception occurs.

Pregnancy: Treatment options for glaucoma during pregnancy depend upon disease severity and progression. In nonurgent cases, observation may be the protocol, since young pregnant females usually tolerate mild IOP rise without problem and so treatment may be deferred until after delivery.[5] In case of disease progression, medical treatment should be initiated or altered.

Drugs used during pregnancy have been classified by FDA into risk categories, viz. A, B, C, D, and X ranging from safest to contraindicated, as under[6] -

Category A- deemed safe
Category B- possibly safe to use in pregnancy
Category C - adverse effects reported in animal studies
Category D- definite risks but possible benefits
Category X- drugs with known risks to the foetus that cannot be outweighed by possible benefits

No anti glaucoma medication falls in category A. Prostaglandin analogues (PGA), carbonic anhydrase inhibitors (CAI), para-sympathomimetics, and osmotic agents are classified as category C drugs. Oral beta blockers are categorized as class C medications, but no such categorization is available for topical beta blockers. Brimonidine is the only glaucoma drug which is a category B medication and considered safe in pregnancy. The newer category of Rho kinase inhibitors has not yet been classified, due to lack of clinical studies.
Thus, the choice of ocular hypotensives for pregnancy and post-partum would be as follows-

1st trimester- Brimonidine (category B) may be the safest option. Other groups of antiglaucoma drugs, such as PGAs, beta blockers, and CAIs should be avoided to reduce teratogenic effects or premature abortion.
2nd trimester- Other than brimonidine, beta blockers can be used with regular monitoring of foetal heart rate and growth. PGAs, CAIs and pilocarpine may also be used as second line with caution and frequent monitoring.
3rd trimester- Brimonidine should be stopped in late third trimester as it can cause CNS depression in new born. PGAs should be avoided as they carry the risk of inducing uterine contraction and labour. Use of topical CAIs may be optimal in this period.
Post partum- Glaucoma medications can appear in human breast milk. Brimonidine is contraindicated in lactating mothers. CAIs and beta blockers have been certified as safe during lactation by the American Academy of Paediatrics.[7] Whatever the choice of drug used, its potential systemic side effects should be avoided or minimized by decreasing its systemic absorption. Digital occlusion of the naso lacrimal duct and eyelid closure are simple techniques that not only reduce the systemic adverse effects, but also increase eye-drug contact time resulting in higher intraocular drug concentrations.

Apart from medications, laser trabeculoplasty may be an alternative modality. Both argon laser trabeculoplasty (ALT) and selective laser trabeculoplasty (SLT) are equally safe and can be used in all trimesters. Although IOP control achieved by trabeculoplasty is short lived, the effect will generally last till the end of pregnancy and lactation. Surgery is best avoided during pregnancy, as it is potentially hazardous for both the mother and foetus. Additional challenges include problems with anaesthesia, positioning for surgery, and management of postoperative complications. However, if required, it is best performed either before conception or if needed during pregnancy, then the second trimester is the safest.

CONCLUSION

All female glaucoma patients of childbearing-age should be educated about the importance of communicating any plan to become pregnant so that a tentative glaucoma treatment plan can be formulated before conception. Ideally, the glaucomatous disease should be treated/controlled by laser or surgery before pregnancy occurs, to reduce the need for medications later. When medication is necessary, the safest drug in minimal effective dosage should be used, with steps to minimize systemic absorption. Laser trabeculoplasty may also be considered as a safe and effective alternative for IOP lowering. Surgery is best avoided during pregnancy.

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ONE MINUTE TIP IN GLAUCOMA

Tips & Tricks to Perform Safe LPI

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Laser Peripheral Iridotomy (LPI) is an OPD laser procedure performed to relieve pupillary block and to facilitate movement of aqueous humour from posterior chamber to anterior chamber.

Indications

1. Angle Closure Glaucoma
    - α. Primary Angle Closure (PAC)
    - β. Early stages of Primary Angle Closure Glaucoma (PACG) with limited ITC
    - χ. Fellow Eye of patient with PAC/PACG
    - δ. Potentially Occludable Angles with Poor Compliance / Follow-ups or those requiring frequent eye dilation for some retinal pathology (Diabetic Retinopathy)
    - ε. Patients with Symptoms of Angle Closure Attacks
2. Secondary Angle Closure with Pupillary Block
    - α. Ectopia lentis, Subluxated lens, Marfans Syndrome, Microspherophakia
    - β. Uveitic Glaucoma (Early Stages)

Procedure

1. Explain the procedure to patient and take a signed consent
2. Instil single drop of 0.2% Brimonidine eye drops 1 hour before & just after the procedure to lower IOP spike.
3. Instil 2% pilocarpine eye drops 3 times 15 minutes apart to reduce iris thickness. This facilitates iridotomy creation.
4. Seat the patient comfortably on the laser slit lamp. Do proper adjustments to the chin rest & table height. Apply iridotomy lens (Abraham's 66D) after putting topical anaesthetic eye drops in the eye.
5. Laser Focus - Set focus on iris stroma
6. Iridotomy site - Peripheral crypt between 11 & 1 O'clock (as it is covered by upperlid). Avoid 12 O'clock position as air-bubbles may occlude the iridotomy. Avoid visible iris vessels. In silicone oil or gas filled eyes perform an inferior LPI. Use minimal amount of energy to avoid any damage to lens capsule
7. Laser parameters (Nd:YAG Laser)
    - ι) Power: 1 - 6 mJ
    - ιι) Spot Size: 50 - 70 microns
    - ιιι) Number of Pulses Per Burst: 1 - 3 (constant for each laser model)
8. Endpoint: A small (150 to 200 micron), peripheral and completely patent iridotomy is the ideal result. A full thickness iridotomy creation is indicated by a gush of aqueous with iris pigments from posterior chamber to anterior chamber. Once an opening is created, it can be enlarged horizontally to achieve adequate size.

An ideal iridotomy should be patent even after iris oedema, pigment proliferation & pupillary dilation. Positive transillumination through iridotomy site is not a relaible indicator of patency.

9. Post procedural medications - Give topical aqueous suppressant drugs (betablockers) & topical steroids for a period of 7-15 days to lower IOP & control inflammation

Follow-up Checkup

Evaluate IOP, inflammation, patency of iridotomy 1 hour & 7 days post procedure. If patency is uncertain, repeat goniscopy to confirm angle opening. Repeat iridotomy procedure if required.

Complications

1. Transient Rise in IOP:
    - Most common complication
    - Transient, occurs most frequently within first 4 hours after procedure
    - Cause is transient obstruction of the trabecular meshwork by released iris pigments, debris & blood
    - Other causes include Plateau Iris Syndrome, Non Pupillary-block ACG, extensive inflammation, PAS & prolonged exposure to steroids
    - Treatment is with topical aqueous suppressants (Betablockers)
2. Hyphema
    - Mostly occurs through the iridotomy site
    - Minor bleed can be stopped by applying light pressure with gonioscopy/iridotomy lens
    - The incidence and severity of hyphema has been reported to be similar whether the patient was on or off antithrombotic therapy (i.e. aspirin, clopidogrel, warfarin) and between groups taking different antithrombotic medications. Therefore, antiplatelets and anticoagulants need not be discontinued before LPI.
3. Visual Dysphotopsias
    - Few patients report blurring, glares, halos, lines, spots and shadows (2% - 16%)
    - Majority of symptoms resolve within first 6 months of follow-up
4. Cataract Progression
    - LPI increases risk of cataract progression ranging from 12 months to 6 years
    - Posterior subcapsular cataract is the most common form
5. Iridotomy Closure & Need for repeat LPI
    - Closure rate of PI has been reported around 1% at 2 weeks to 20% at 6 months, but majority of PI remain patent years after the procedure
    - Closure is due to accumulation of debris & pigments at the site, although closure of PI is rare with Nd:YAG laser.
    - Repeat LPI can be performed at the same site (immediately after the procedure) or at other site (on next sitting)
6. Aqueous Misdirection Syndrome
    - Rare complication
    - There is forward movement of lens iris diaphragm causing 2^0 angle closure & raised IOP
    - It can occur in very small eyes (axial length <21 mm) with higher hypermetropic refraction (> +6D)
    - Possible mechanism is contact of ciliary processes with lens equator, and/or a firm zonule/ posterior capsule diaphragm, causing misdirection of aqueous into the vitreous
    - It should be considered in eyes with progressive anterior chamber shallowing and myopia despite normal IOP
7. Other rare complications
    - Reduced endothelial cell count

- Posterior Synechiae formation due to persistent inflammation
- Sterile Hypopyon
- Cystoid macular oedema
- Macular Hole
- Retinal Hemorrhages

Outcomes

1. IOP Control
    - Better in earlier stages of Angle Closure Spectrum (PAC>PACG)
    - Several studies regarding the spectrum of PAC report that 7.1%-28.0% of PACS eyes, 42.4%-80.0% PAC eyes and 83.3%-100% of PACG eyes required additional medical and/or surgical intervention after LPI
2. Anatomical Improvement
    - LPI Increases angle width, anterior chamber depth
3. Prevention of Angle Closure Attacks

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ONE MINUTE TIP IN GLAUCOMA

Diode CPC – How & When

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Lee and Pomerantzeff in 1971 introduced the use of the laser for CPC. The effect of laser energy on target and surrounding tissue depends on the amount and duration of energy absorbed, which are dependent on tissue pigmentation, wavelength, amount of energy delivered, exposure time, and size of the laser spot.

WHEN:

Laser CPC is a destructive procedure. It is a therapeutic modality of last resort, useful in advanced glaucoma, in which the IOP is uncontrolled despite maximum tolerated medical treatment, in eyes that have failed filtering surgery or are likely to fail future filtering surgery. This includes eyes in which filtering surgery has a high risk, such as aphakic glaucoma, neovascular glaucoma, glaucoma after penetrating keratoplasty, or in eyes with very low visual potential. CPC destroys tissue, lowering IOP by the destruction of the ciliary body, decreasing the amount of aqueous produced. A patient to undergo CPC should have IOP that is uncontrolled despite maximum tolerated treatment and any one of the following:

- Failed prior filtration surgery or the expectation that further glaucoma filtering surgery will fail
- Glaucoma that is likely to fail filtering surgery (neovascular, inflammatory, post-penetrating keratoplasty, post-scleral buckling) or is a high risk for complications of filtering surgery (vitrectomized aphakic eye)
- Poor visual acuity
- The patient is not a surgical candidate for filtering surgery for general medical reasons

HOW:

The laser energy may be delivered with a slit-lamp (non-contact) delivery system or with a fiber-optic probe placed directly on the conjunctiva (contact). It destroys the ciliary epithelium, stroma, and vascular supply. A specialized contact lens may be useful to help control eye movement, compress the conjunctiva, and assist in placing laser applications with the non-contact variety. The angle of laser incidence of the beam should roughly parallel the visual axis and strike the globe approximately 1–2mm posterior to the limbus in non-contact technique.

In laser trans-scleral destruction of ciliary processes, treating 270° with 16–18 applications may decrease the incidence of complications and is recommended. The probe should be placed 0.5–1.0mm from the limbus and held as perpendicular to the sclera as possible. Probes made especially for cyclophotocoagulation such as the G-Probe™ of Gaasterland make placement easier and more uniform. The 3 and 9 o'clock meridians are avoided to prevent damage to the long posterior ciliary arteries.

Pre-operative: Continue all preoperative glaucoma medications prior to treatment.

Topical vasoconstrictors are recommended by some to reduce the sub conjunctival hemorrhage.

Informed consent

Anaesthesia: Retrobulbar or peribulbar anaesthesia. Long-acting anaesthetics such as bupivacaine help with immediate postoperative pain control.

Positioning: Patient recumbent for contact and sitting for noncontact treatment.

The patient's eyelids may be separated manually or, more conveniently, with a speculum.

Laser settings: Diode laser: 3 watts, 1.3 s (if pops are heard with 3 consecutive spots, decrease power by 0.5 watts) 18- 20 spots over 270 degrees, sparing inferonasal quadrant Spot location.

Application of G-Probe, place edge of handpiece at limbus, which centres fibreoptic 1.2-mm posterior to limbus. With each subsequent spot, the radial edge of the handpiece overlaps the previous spot, producing 18 spots over 270 degrees. Press gently with probe during treatment throughout exposure, keeping the handpiece perpendicular to the sclera. Maintain contact throughout energy delivery.

Post operative care: The treated eye is patched for 6 – 24 hours

Prednisolone acetate 1% QID and Atropine sulphate 1% BID. To be tapered when inflammation subsides.

All preoperative glaucoma medications are continued except for miotics.

IOP check at Day 1, 1 week and thereafter depending on the patient's clinical response. Maintaining IOPs of 22mmHg or lower is the desirable response. Oral carbonic anhydrase inhibitor in cares of initial IOP spikes in cases treatment is insufficient retreatment to be considered.

Retreatment: if required, 1 month postoperatively. Each retreatment increases the possibility of phthisis.

Complications: are less as compared to cyclocryotherapy includes, reduced visual acuity, uveitis, pain, sub conjunctival haemorrhage, hypotony and phthisis bulbi.

Endocyclophotocoagulation procedures may be most useful for aphakic and pseudophakic eyes or in combination with phacoemulsification procedures. This allows the surgeon to directly visualize and destroy the ciliary processes, is gaining in popularity. To target the ciliary epithelium more accurately endocyclophotocoagulation has been increasingly popular. It is required to be done under stritct a sterile environment. Here an endoscopic probe and laser delivery fibre introduced via a limbal incision and is invasive technique.

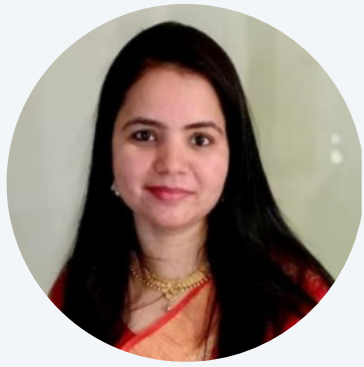
When compared to ND YAG laser, the diode laser has the advantage of greater absorption by melanin and nearly equivalent scleral transmission, greater convenience and economical. Anecdotally, there is less pain and inflammation with Contact technique as compared to non-contact one.

CONCLUSION:

Diode CPC is a relatively safe and effective procedure for patients with poor vision or poor visual prognosis if all other therapies fail or are contraindicated to control IOP. It is possible to reduce aqueous production and lower intraocular pressure (IOP) by destroying elements of the ciliary body by Diode laser.

ONE MINUTE TIP IN GLAUCOMA

SELECTIVE LASER TRABECULOPLASTY



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Glaucoma is a disease of progressive optic neuropathy and irreversible field loss. Raised intraocular pressure (IOP) is the key cause for these changes. The various modalities of treatment for glaucoma like medical, laser and surgical, attempts to lower the IOP.[1]

Laser trabeculoplasty is a safe and effective treatment modality to reduce the IOP in patients with glaucoma. The foremost advantage is its ability to lower IOP without relying on patient's compliance. Argon laser trabeculoplasty (ALT) was introduced 40 years back by Wise and Witter to lower the IOP.[2] Diode laser and krypton laser were also tried. Selective laser trabeculoplasty (SLT) which uses frequency doubled (532 nm) Q- switched Nd:YAG laser. SLT was FDA approved in 2002.

Mechanism of action:

SLT is based on the concept of selective photo thermolysis of trabecular meshwork without damaging the neighboring cells unlike argon laser trabeculoplasty. It lowers the IOP by stimulating the inflammatory response following the laser and remodeling the extracellular matrix by activated macrophages to increase the aqueous outflow.

Indications

- Newly detected open angle glaucoma (OAG)
- OAG patients not responding to medications
- OAG patients with poor compliance or poor tolerance to medical management
- Patients with pseudo exfoliation and pigmentary glaucoma.
- In secondary glaucoma following Penetrating keratoplasty.

It is contraindicated in patients with poor visualization of trabecular meshwork, inflammatory glaucoma, and congenital glaucoma.

Procedure

SLT uses frequency doubled Q-switched Nd:YAG laser (532nm) coupled to slit-lamp delivery system. The parameters are:

- Spot size – 400 microns
- Power- 0.8-1.4 millijoules
- Duration of exposure- 3 nanoseconds

The procedure is performed under topical anesthesia and 1% Apraclonidine is instilled in eye 1 hour prior to procedure to prevent post-procedure IOP spikes. Most accepted protocol is to treat 360 degrees of trabecular meshwork with non-overlapping 100 laser spots. A single mirror gonio-lens is used to focus trabecular meshwork. The energy levels are set in a way to visualize minimal bubbles, referred as "champagne bubbles" from meshwork on exposure to laser. Following the procedure anti-inflammatory drops are prescribed for 7 days.

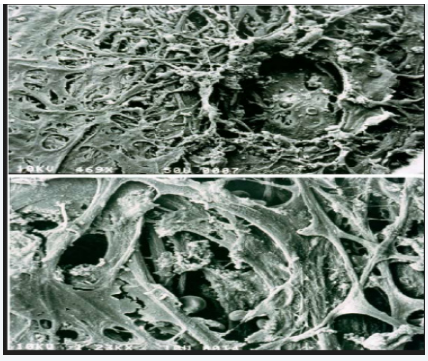

Figure 1: Electron microscopy of trabecular meshwork following ALT above shows disruption of trabecular meshwork and crater formation (above). SLT shows intact trabecular beams (below)

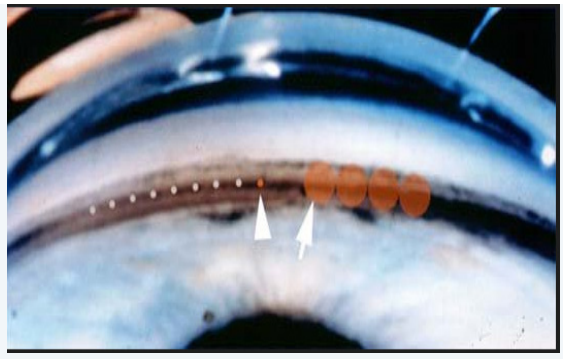

Figure 2: Difference in spot size of ALT (left arrow) and SLT (right arrow)

Repeatability

Hong et al reported that, eyes that underwent 360-degree SLT lost their efficacy to control IOP after 6 months and with the second sitting of SLT there was better control of pressure. Other studies also show SLT is safe and effective to repeat.[3]

Advantages

In glaucoma patients there is myriad of adherence barrier like high cost of medication, poor eye drops instillation technique, non-compliance of patients on multiple drugs. This in turn leads to in-consistent control of IOP. By opting for SLT as an adjunct or first line of treatment one can decrease or eliminate the need for topical medication. And can also reduce the systemic and local adverse events associated with it. SLT can be repeated it is cost-effective and delay the need of filtration surgeries and complications associated with it.

Adverse events

Few adverse events like transient rise in IOP, anterior chamber reaction , redness , pain , blurring of vision and bleeding are reported but these are infrequent and can be easily managed.

SLT v/s other management

Melamed et al studied the role of SLT as initial line of management in patients with OAG and reported 30% reduction in IOP from the baseline which was comparable to prostaglandin efficacy.[4] Nagar et al compared the pressure controlling effectiveness of SLT and latanoprost and found it to be on par.[5] But, the IOP fluctuations were better managed by latanoprost and SLT had an advantage of one-time intervention. Francis et al have reported that a combination of SLT in medically managed patients of glaucoma had additional IOP reduction.[6]

SLT has become a popular alternative to ALT mostly due to lesser collateral damage to surrounding tissue. It is better tolerated by patients with lesser discomfort and post-operative complications.

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SURGICAL SECRETS

Secrets to Perform MIGS – Guide to Beginners

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Glaucoma is a chronic progressive eye disease that damages the optic nerve and can lead to irreversible vision loss if left untreated. The condition affects more than 76 million people globally, making it one of the leading causes of blindness worldwide. Fortunately, advancements in glaucoma surgery have made it possible to manage the condition effectively.

Microinvasive glaucoma surgery (MIGS) is a relatively new approach to managing glaucoma. MIGS techniques involve minimally invasive procedures that target the trabecular meshwork or the drainage system of the eye, which is responsible for regulating the flow of aqueous humor, the fluid that nourishes the eye. Unlike traditional glaucoma surgeries, MIGS procedures typically have fewer risks and complications, faster recovery times, and less invasive techniques.

- One of the most popular MIGS procedures is the Kahook Dual Blade (KDB), which was introduced in 2015. The KDB is a single-use instrument that is designed to excise the trabecular meshwork in a controlled and precise manner. The device is inserted into the eye through a clear corneal incision and advanced towards the trabecular meshwork. The tip of the KDB Glide can enter the trabecular meshwork at a 10° angle (angled up) and then the footplate is leveled gently against the anterior wall of the canal of Schlemm. KDB Glide is advanced while ensuring the two blades are engaging TM on either side to create parallel incisions. Surgeons should target treating ~3-4 clock hours. The procedure is typically performed under local anesthesia and can be completed in less than 10 minutes.
- Another MIGS procedure that has gained popularity in recent years is the Bent Angle Needle Goniectomy (BANG). This procedure involves using a specialized bent needle to create a small incision in the trabecular meshwork and excise it, allowing the aqueous humor to drain more freely.
- The Trabectome is another MIGS procedure that has been in use since 2004. The Trabectome is a small, handheld device that is used to remove a portion of the trabecular meshwork. The device is inserted through a clear corneal incision and advanced to the trabecular meshwork, where it is used to remove a small strip of tissue.
- The iStent is a tiny, FDA-approved device that is implanted in the trabecular meshwork to improve the outflow of aqueous humor. The iStent is inserted through a clear corneal incision and advanced to the trabecular meshwork, where it is implanted.
- Finally, the Gonio-assisted Transluminal Trabeculotomy (GATT) is a newer MIGS procedure that involves using a microcatheter/ prolene suture to access and remove the trabecular meshwork.

In conclusion, microinvasive glaucoma surgery has revolutionized the management of glaucoma. MIGS procedures are typically less invasive, have fewer risks and complications, and faster recovery times than traditional glaucoma surgeries , can reduce the medication burden and improve quality of life. With several MIGS procedures available, ophthalmologists can choose the best procedure to fit each patient's individual needs.

Tips for success with MIGS

1. The first step towards angle surgery is thoroughly understanding the anatomy of the angle and to practice gonioscopy in OPD as much as possible. Make sure you do a detailed pre operative work up and examine the angle – document all your findings, including the pigmentation of Trabecular meshwork.

2. To master intra operate gonioscopy and to be able to achieve enface view of the angle (Figure 1) is of paramount importance. Tilt the head of the patient 35-40 degree away from surgeon and tilt the microscope 30 degree towards the surgeon. Gently place the goniolens over the cornea with your non dominant hand and perform angle surgery with your dominant hand. Care must be taken to not compress the cornea to avoid creating corneal folds, the gonio lens should float over a bed of viscoelastic over the cornea. Use higher magnification to observe details of the angle anatomy (so the gonio lens takes up most of the view).

3. With practice, one can become familiar with how much OVD to use when filling the anterior chamber. Using too much can cause compression of Schlemm's canal, making it challenging to access or open during a trabecular bypass operation. On the other hand, using too little OVD can result in the iris protruding forward, which can obscure a clear view of the iridocorneal angle

4. Preferably give a clear corneal incision to avoid limbal bleeding. Nicking these vessels can lead to bleeding and cloudiness of the corneal tear film, which can make it difficult to see through the gonioprism. A 1.8 mm incision or larger will allow the surgeon to maneuver easily from side to side. The incision can be “flared” posteriorly to allow for greater excisional range.

5. In suture GATT, the tip of the suture shouldn’t be a big mushroom tip causing damage to the schlemm’s canal.

6. When combined with cataract extraction, goniotomy can be done before or after completing phacoemulsification with intraocular lens (IOL) implantation. Many surgeons new to goniotomy prefer to perform the procedure after phaco and IOL implantation due to a deeper chamber enhancing angle visualization. However , if doing goniotomy prior to cataract extraction – the big advantage is that the eye is NOT hypotonous . Hypotony will lead to blood in schlemm’s canal and when starting to excise trabecular meshwork , blood reflux comes and obscures the surgeon’s view .

7. Intraoperative blood reflux is expected in angle surgery. Reflux indicates distal outflow patency. If the view is compromised, STOP, and displace blood with OVD. Do not treat what you cannot see. At the end of the case, keep IOP somewhat firm (~25 mmHg) and ensure watertight incision. 10%-20% fill of dispersive OVD to encourage flow of fluid out of the eye.

8. Use of Pilocarpine 1-2% 1drop q5m x 3 preoperatively can provide for a clearer view of the angle at the time of KDB if used in standalone cases. In cases combined with cataract extraction, miotic can be used intra-operatively to enhance the view of the angle if done after IOL implantation.

9. The TM strip can be removed with intraocular forceps (e.g., rhexis forceps, micro forceps) or irrigated/aspirated out of the eye (e.g., with I/A tip). Tethered TM (Inside-Out technique) may be amputated by grasping and pulling radially and in the direction of the untethered point or multiple passes with KDB can be completed if needed to amputate tethered tissue. Avoid pulling the tethered TM centrally.

10. Inflate the anterior chamber to a pressure of 20-25mmHg to help direct flow towards the collector channels. Hydrate the wounds to ensure they are watertight. You will notice blanching of episcleral vessels of the treated angle (nasally) while hydrating the wounds. This blanching proves that the drainage system is patent and communication has been established between the anterior chamber and collector channels.

11. Post operatively, apart from antibiotic and steroid drops, pilocarpine drops can be used for initial 2 weeks to prevent peripheral anterior synechiae formation and also to take care of any IOP spike that may happen in the initial post op period.

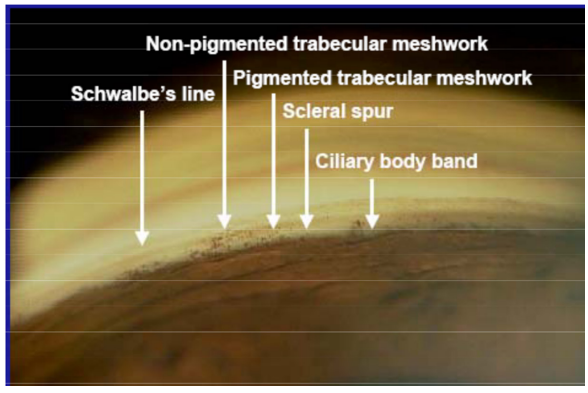

Figure 1 – En face view of angle

PEARLS OF WISDOM

How To Differentiate Glaucomatous And Nonglaucomatous Disc?

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- The evaluation of the appearance of the optic disc is central to the diagnosis and management of Glaucoma.
- Optic Disc Evaluation:
  - Why..?
  - How..?
  - What to look for..?

The 4 goals of optic disc evaluation

- Distinguishing between the healthy and the sick =Diagnosing.
- Quantifying the amount of damage: Healthy,Mild,Moderate,Advanced Disease
- Monitoring Change,for better or for worse
- Quantitating the rate of change

Optic Disc Evaluation

- Slit lamp biomicroscopy : Ideal
  - Stereoscopic View –Cupping
  - Measuring the optic disc size
- Direct Ophthalmoscopy
  - Good Magnification
- Indirect Ophthalmoscopy
  - Overall View
- Optic disc Photoghraphy.
  - Documentation,Monitoring for progression

The 7 parameters to look for…

1) Disc: Size and Shape
2) Neuroretinal Rim (NRR):
   - Size,Shape,Pallor
   - ISNT rule
3) Cup: Size and Shape in relation to the optic disc size,
   - Vertical C/ D Ratio, Cup depth / Excavation
4) Optic Disc Hemorrhage: Presence & Location
5) Nerve Fibre Layer Defect:
   - focal & diffuse
6) Para Papillary Atrophy;
   - Size,location & Configuration
7) Retinal Arterial Attenuation:
   - focal & diffuse

All these variables can be measured semi quantitatively by ophthalmoscopy without applying sophisticated techniques

1) Optic Disc: Size & Shape

- Determining the size of the disc =Crucial
  - Helps to differentiate Physiological cupping from Pathological.
    - Large discs have big physiological cups.
    - Small Discs have small cups or no cups
- Measurement of Vertical Disc diameter :
  - Length of the vertical beam of slit lamp light
  - Multiplied by correction factor of the condensing lens
  - Volk 60 D= X 1
    Volk 90D= X 1.5

2. Cup: Size, Shape, location in relation to the disc size (Figure 1)

- Optic Cup= Excavation in the optic nerve head
  - Stereoscopic evaluation
- In normal eyes= Areas of optic disc & Optic cup are corelated
- Large optic discs=Large cup
- Small optic disc =Small cup or no cup
- Early & moderate glaucomatous damage in small disc may be missed because of the erroneously low cup disc ratios
-

Vertical Cup Disc Ratio (Figure 2)

- Vertically oval optic disc
- Horizontally oval optic cup
- In normal eyes: Horizontal CD ratio > than vertical CD ratio
- In Glaucomatous eyes: Vertical CD ratio > than the horizontal CD ratio

3) The Neuroretinal Rim (Figure 3)

- Size, Shape, Pallor.
- The ISNT rule:
  - Thinning of the NRR
  - Pallor of NRR
  - Notching:
    - A notch is a localized defect in the Neuroretinal rim on the cup side of the rim

The Neurretinal rim loss in Glaucoma (Figure 4)

- Usual sequence of NRR loss in Glaucoma:
  - Inferotemporal
  - Superotemporal
  - Horizontal temporal
  - Inferonasal
  - Superonasal
- In contrast, in the non glaucomatous optic nerve damage, the NRR is not always affected and hence contour of NRR is maintained.

4) Optic Disc Hemorrhage (Figure 5)

- Splinter or Flame shaped hemorrhages
- At the margin of the disc
- Hallmark of Glaucomatous optic nerve damage
- 4 to 7 % of eyes with galucoma

- Found in early & moderately advanced Glaucoma and rare in very advanced stage
- Located usually in the inferotemporal & superotemporal disc margins
- Associated with localized RNFL defect and neuroretinal rim notches .
- Suggests Progression.
- More common in NTG

5) Retinal Nerve Fibre Layer Defect (Figure 6)

- RNFL contains retinal ganglion cells axons covered by astrocytes and bundled by processes of muller cells
- Seen as bright fine striations fanning off from the disc to the periphery.
- Dilated pupil, green light, clear optical media aids the evaluation of RNFL
- Localized RNFL defects:
    - Can be detected before visual field defect has developed
    - Focal type of NTG
    - Early to medium advanced Glaucomatous damage
- Diffuse loss of RNFL:
    - More difficult to detect
    - Peripapillary retinal vessels appear bare
    - Underlying Choroidal vessels more clearly seen

6) Parapapillary Chorioretinal atrophy (Figure 7)

- 2 zones
    - Central Beta zone
    - Peripheral alpha zone
- Beta zone occurs more often in glaucomatous eyes than in normal eyes.
- Helps to differentiate various subtypes of POAG
- Helps to differentiate from nonglaucomatous optic nerve damage

7) Retinal Artery attenuation (Figure 8)

- Diffuse narrowing:
    - Decreasing NRR
    - Increased RNFL loss
    - Increased Visual field defects
- Focal Attenuation
    - More common in NTG
    - Degree of narrowing increases with amount of damage.

Pre Perimetric Diagnosis of Glaucomatous Optic Nerve damage (Figure 9 & 10)

- Most important Variables
    - Shape of the NRR
    - Size of the cup in relation to the optic disc
    - Diffuse or focal RNFL defects
    - Disc Hemorrhages

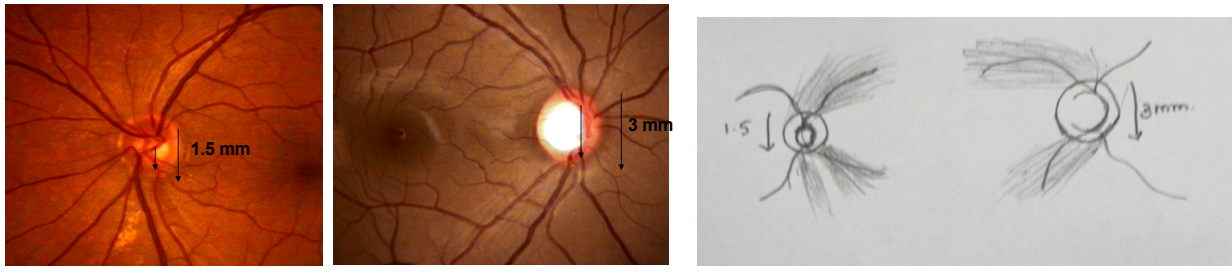


Figure 1

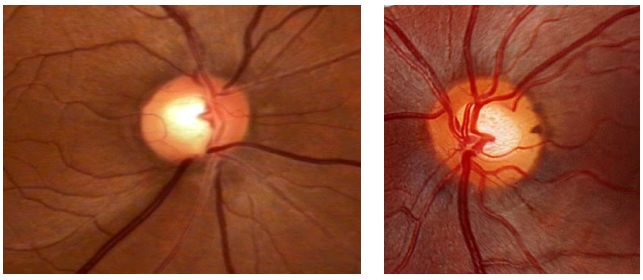


Figure 2

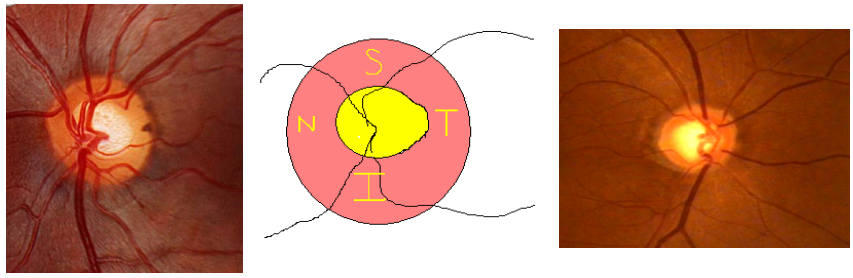
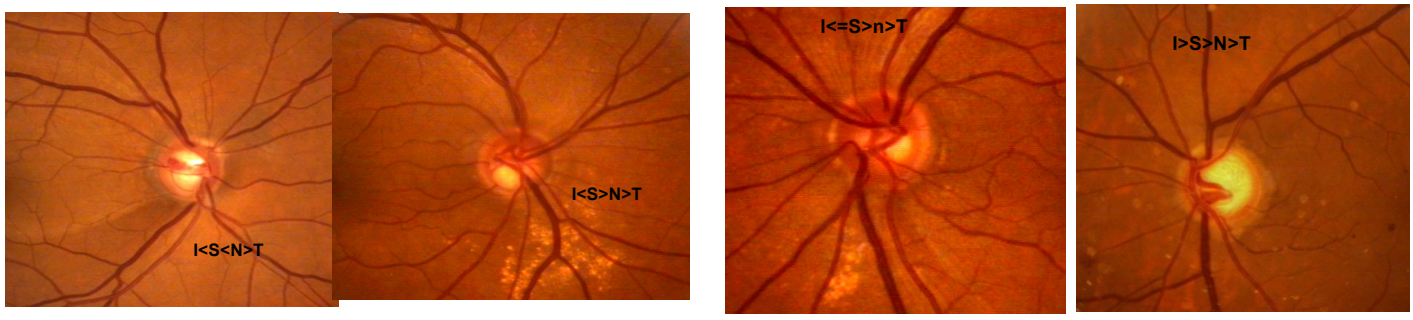


Figure 3



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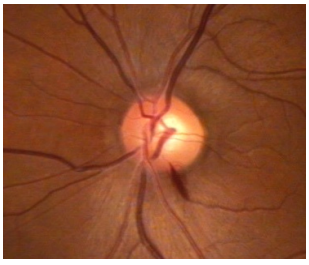

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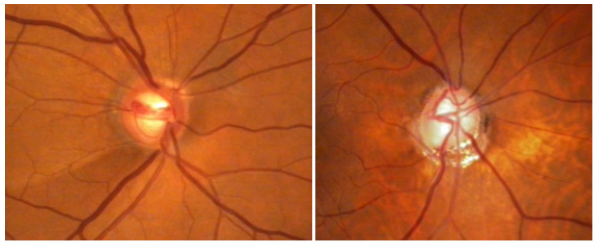

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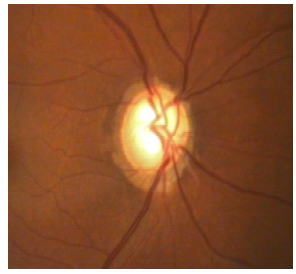


Figure 7

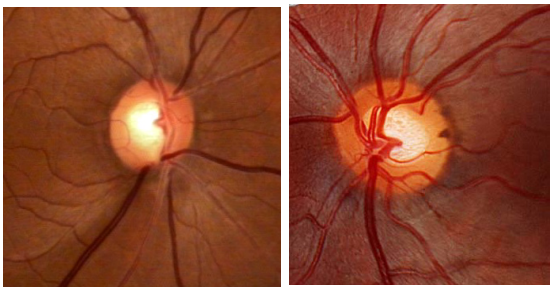


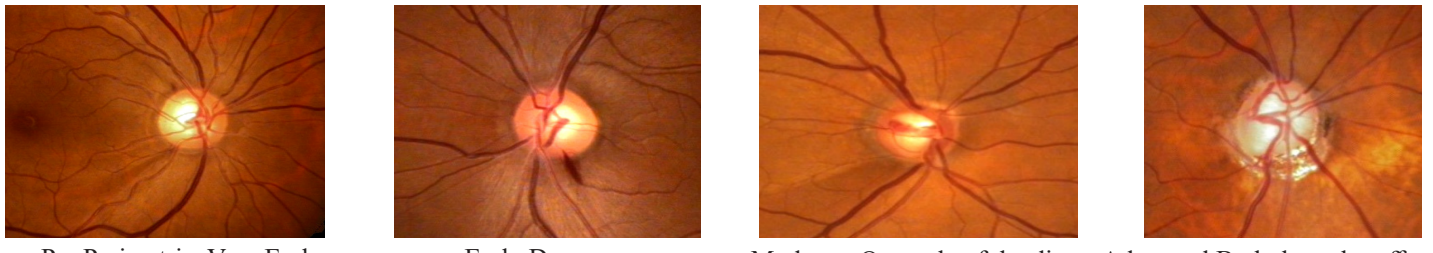
Figure 8

Figure 10

Disc Damage Likelihood Scale

THE DISC DAMAGE LIKELIHOOD SCALE

DDLS Stage	Narrowest width of rim (rim/disc ratio): For Small Disc <1.50 mm	For Average Size Disc 1.50-2.00 mm	For Large Disc >2.00 mm	DDLS Stage	Examples: 1.25 mm optic nerve	1.75 mm optic nerve	2.25 mm optic nerve
1	.5 or more	.4 or more	.3 or more	0a			
2	.4 to .49	.3 to .39	.2 to .29	0b			
3	.3 to .39	.2 to .29	.1 to .19	1			
4	.2 to .29	.1 to .19	less than .1	2			
5	.1 to .19	less than .1	0 for less than 45°	3			
6	less than .1	0 for less than 45°	0 for 46° to 90°	4			
7	0 for less than 45°	0 for 46° to 90°	0 for 91° to 180°	5			
8	0 for 46° to 90°	0 for 91° to 180°	0 for 181° to 270°	6			
9	0 for 91° to 180°	0 for 181° to 270°	0 for more than 270°	7a			
10	0 for more than 180°	0 for more than 270°		7b			



Pre Perimetric, Very Early

Early Damage

Moderate One pole of the disc is damaged

Advanced Both the poles affected

Figure 9

PEARLS OF WISDOM

Is it Glaucoma or High Myopia?

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Many of us face the dilemma differentiating high myopia and glaucoma in our clinical practice.

Previous epidemiological studies across different ethnic groups have shown an increased prevalence of glaucoma in myopic patients, including the Blue Mountain Eye study, Beaver Dam Eye Study, Singapore Malay Eye Study and Beijing Eye Study.

Why myopes are at increased risk of glaucoma?

Exact eitology is unknown, but some hypotheses suggest that the tilting of the optic nerve often seen in high myopia may lead to shearing effect to the ganglion cell axons. Some studies suggest that the effects of IOP can be more pronounced in myopes due to thinning of the retinal nerve fiber layer (RNFL), lamina cribrosa and sclera. (1)

Increased Axial length in high myopia also causes stretching and thinning of the lamina cribrosa. Thus making retinal ganglion cell axons more susceptible to increased IOP.

In glaucoma as well, the primary site for axonal insult is the lamina cribrosa. Superior-temporal and inferior-temporal quadrants of optic nerve head are most vulnerable to glaucomatous damage. (2)

Traditional methods to diagnose glaucoma ,include direct observation of the optic nerve head to notice neuroretinal rim thinning, IOP measurement, RNFL thickness measurement using optical coherence tomography (OCT) and a visual field test to determine any functional vision loss as a result of glaucoma.

Tilting of the optic nerve head seen in degenerative myopia makes the assessment of neuroretinal rim (NRR) thinning challenging thus making it difficult to rule out glaucoma.

FUNDUS PHOTOGRAPHY- Red free fundus photograph should be captured to determine the extent of NRR thinning. It helps in better visualisation and delineation of the neuroretinal rim & optic cup.

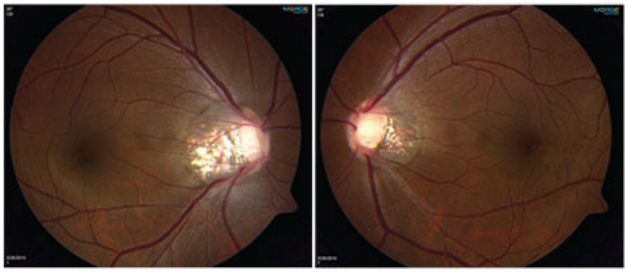

Figure 1:A young myope with abnormal-looking discs. Both ONH are oval and tilted, with significant temporal peripapillary atrophy.

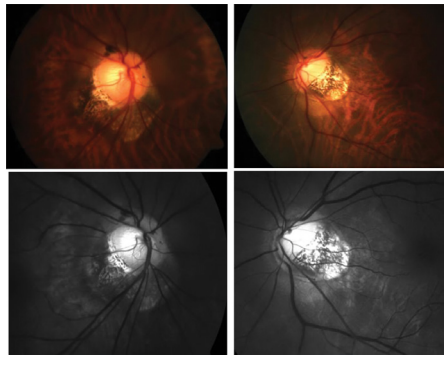

Figure 2: colored fundus photograph shows anomalous ONH insertion, tilting and torsion in a high myopic patient (top). Below is the same image of the ONH using red-free fundus photography to visualize the extent of thinning better

Any disc haemorrhage to be documented suggesting of glaucoma. Periodic disc photographs to be documented to observe any disc changes.

IOP MONITORING- Look for diurnal variation in IOP. Schedule for IOP measurement visits at different times of the day.

Optical coherence tomography (OCT) -Both glaucoma and high myopes have reduction in RNFL thickness. In high myopes, large optic nerve head size & structural variations like peripapillary atrophy can give inconclusive RNFL thickness measurement.

Some studies suggest that measurement of macular ganglion cell- inner plexiform layer thickness have good glaucoma detection ability comparable to that of peripapillary RNFL thickness and optic nerve head (ONH) parameters.(3)

VISUAL FIELDS (VF) - In pathological myopia, optic nerve thinning & tilting can create visual field defects similar to those seen in glaucoma. Progress of VF loss in follow up visits is a clear sign of glaucoma.

In myopes, trial lenses are used to correct refractive error, causing significant minification of the stimulus. While assessing the VF in high myopia with vision <6/60, a size V stimulus may be beneficial to assess any progression more easily.

Clinical pearls:

*Consider patient's age, family history and visual status.

*One eyed or patient with poor vision to be considered for treatment in case of doubt.

*Serial disc photographs to be taken in follow up visits examination & any change points towards glaucoma progression.

*Notice any progression in follow up VF indicating glaucoma progression.

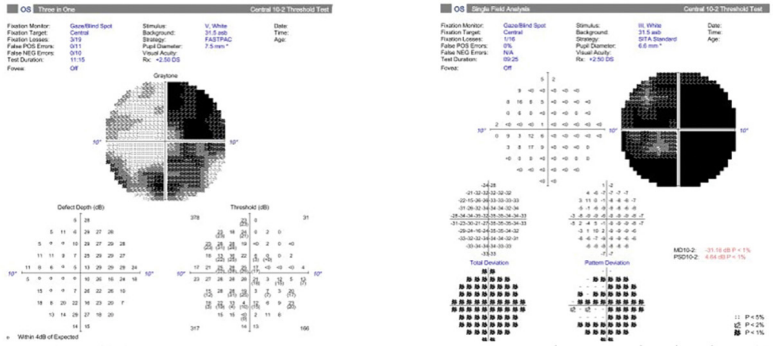

Figure 3: A 10-2 VF with size III target showing a generalized depression (right). 10-2 of the same patient with a size V target showing a better visualization of the pattern of VF loss (left). The bigger stimulus size may allow for easier determination of glaucoma progression

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EVIDENCE BASED APPROACH

OCT Evaluation Of Peri Papillary RFNL Thickness And Macular Thickness to Evaluate and Monitor Glaucoma Patients

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1. Introduction

Glaucoma is leading cause of irreversible blindness in the world. A report estimated that there are 80 million people worldwide with glaucoma in 2020. POAG is the most common type of glaucoma. It is basically chronic progressive optic neuropathy accompanied by cupping and atrophy of Optic disc, visual field loss etc. Glaucomatous Optic Neuropathy causes progressive death of Retinal Ganglion Cells(RGC) and their axons which Can be seen by increased optic nerve cupping or peripaillary RNF losses on SD-OCT.

Macular is densely populated by RGC containing 30% of the total no. of these cell occupying only 2% retinal area while 50% located within 4.5mm of fovea with peak density occurring 750 to 1100 micron from centre of fovea. It has been hypothesized that in early glaucoma loss of RGC occur in macula and disease invariably affect macular thickness early in it's course and fundamental defining abnormality is located at RGC. Thus machular RGC and IPL (inner plexiform layer) thickness measurement are ideal parameters for detecting glaucoma its earliest progressive Profile.

Conventionally in glaucoma patient structural progression may be focal or diffuse and it may occur well before any visual deficits are apparent. Structural tests depends on clinicians eliciting changes on the optic disc and of retinal nerve fiber layer (RNFL) on clinical examination. Though nerve anatomy and topography demonstrate significant inter individual variability, we frequently find it cumbersome to distinguish physiological variants such as cupping from those caused by glaucoma (neuroretinal rim loss).

OCT does an objective and quantitative measurement of RNFL and IPL thickness. It is a noninvasive imaging modality uses low coherence light to obtain high resolution cross section of human structures and has drastically changed our perception of retina visualization. The technology has evolved leaps and bounds since its inception by Huang et al in 1991. The most significant growth occurred when the moving mirror was used

II. Material and method

This study was conducted during the session October 2015- November 22 2017 in upgraded department of ophthalmology NSCB medical college. Jabalpur. In this study overall 100 eyes of POAG and 100 eyes of normal age matched control were included. Informed consent was obtained from all subjects. The tenets of declaration of Helsinki were followed. All patients were subjected to detailed history taking regarding following points-

1. Detailed ocular exam – It includes diminution of vision pain, redness, watering photophobia, colouredhaloes, headache, vomiting etc.
2. History of surgery- like cataract surgery, filteringsurgery, post. Segment surgery
3. History of associated systemic illness like diabetes mellitus, hypertension, bleeding disorder or any other disease
4. History of trauma
5. Family history

6. Personal history

All patients underwent detailed clinical evaluation including BCVA by means of snellens chart, Anterior segment evaluation by slit lamp biomicroscopy, fundus examination, IOP measurement with NCT, gonioscopic examination with 4 mirror gonioscope and visual field testing including 30-2 SITA full threshold program with Humphrey's automated perimeter. All patients were scanned with the Zeiss Cirrus HD Spectral Domain OCT.

Exclusion criteria for all patients included –BCVA lkess than 20/40, refractive error exceeding± 5 diopter of sphere or 2 dioptre of cylinder, evidence of vitreous or retinal pathology apart from glaucoma unreliable AP or other pathological condition that could affect the visual field (pituitary lesion, demyelinating disease) and secondary causes of IOP rise (iridocyclitis, corticosteroid use) and prior incisional surgery or laser treatment.

Visual Field testing –Permetry was performed with Humphrey field analyser using the Swedish Interactive Threshold Algorithm (SITA) standard strategy 30-2 full threshold test procedure and size 3 stimulus. The analysis of data was carried out by program STATPAC2 included in the software of the permeter. Perimetry was performed at the same time with OCT. Reliability criteria to accept visual field examination included Fixation loss less than 20% and maximum false positive and false negative rates of 25% Measurement of visual field deparession presented on Humphrey printout include the mean deviation(MD). And patterned standard deviation (PSD) MD is measure of overall field loss and it reflect generalized visual field loss while PSD is measure of focal loss or variability within the field taking into account any generalized depression in the hill of vision and it reflect small localized defects that appear in early stages of glaucoma. The MD and PSD were used for statistical analysis in order to evaluate correlation between macular GCIPL thickness, RNFL thickness and visual field global indices. Visual field global indices and GCIPL and RNFL thickness measurement were compared statistically in all groups

OCT measurements- The Zeiss Spectral Domain Optical Coherence Tomography (SD-OCT) is a noncontact and noninvasive technology that allows cross sectional imaging of human retina at histologic level of resolution. It is based on principle of low coherence interferometry. It is designed to provide real time objective, cross sectional measurement of various layers of retina based on reflectivity of its different layers. It was excluded that an image with a minimum strength 6/10 and below. One of the 3 scans with maximum signal strength was included, for this study we analysed the global average macular GCIPL (Ganglion cell-inner plexiform layer) and average peripapillary RNFL (Retinal nerve fiber layer) thickness in 2 groups of subjects. This results were analysed using the SPSS for windows software and relationship were considered significant if $P<0.05$. Data were reported as mean±standard deviation.

III. Results

Table No.1

S.No.	Age Group	Control		POAG		Total
		No.	%	No.	%	
1	40-50 year	37	37	15	15	52
2	51-60 year	35	35	41	41	76
3	61-70 year	22	22	37	37	59
4	-70 year	4	4	7	7	11

This table shows most of the patients were of 51-60 years age group

Table No. 2: Gender profile

Sr.No.	Gender	Control		POAG		Total
		No	%	No	%	
1	Male	53	53	54	54	107
2	Female	47	47	46	46	93
3	Total	100	100	100	100	200

This table shows that maximum no of patients were male in both group

Table No. 3: IOP Recording and Average CD Ratio

Sr. No.	Parameter	Control	POAG	Significance
1	IOP (mm of hg)	14.82±1.72	24.49±1.91	T=31.48 P<0.001
2	Average CD Ratio	0.38±0.06	0.75±0.09	T=26.9 P<0.001

Table No. 4: Visual Field Indices

Sr. No.	Parameter	Control	POAG	Significance
1	MD (in decibel)	01.71±0.87	.9.07±6.23	T=9.79 P=0.0001
2	PSD (in decibel)	1.84±0.30	6.34±3.36	T=11.19 P<0.0001

This Table shows MD and PSD in control and POAG group

Table No. 5: Co-relation of RNFL thickness with visual field indices

S. No	Status	MD	PSD
1	Average RNFL control (91.91±6.85)	0.03p=0.75	0.04 P=0.74
2	Average RNFL POAG (58.14±15.76)	0.57 P<0.0001	-0.45 P<0.0001

This table shows Average RNFL POAG is directly related to MD and inversely related to PSD

Table No. 6: Various OCT Parameter studied during this study

S.No.	OCT Parameter	Control	POAG	Significance
1	AVERAGE RNFL (in um)	91.91±6.85	58.14±15.76	(36.74%) T=16.44p<0.001
2	Average GCIPL(in um)	81.31±4.64	59.20±13.19	(27.19%) T=13.23P<0.001
3	Minimum GCIPL (in um)	77.99±4.95	42.93±16.92	(44.95%) T=16.64P<0.001

This table shows Average RNFL, GCIPL and minimum CGIPL in control group and POAG patients

Table no. 7: Correlation –Macular Thickness and Peripapillary RFNL

Group	MD	PSD	Average RNFL	Average CD ratio	Minimum GCIPL
Average GCDIPL in control (81.31+4.64um)	P=0.21	P=0.23	P=0.014	P=0.66	P<0.0001
Average GCDIPL in POAG (59.20±13.19um) (27.19%)	P=0.41	P=0.37	P=0.002 (36.74%)	P=0.12	P<0.0001 (44.95%)

This table shows correlation of Average GCIPL thickness in control group and POAG patients with MD, PSD, Average RNFL thickness, Average CD Ratio and Minimum GCIPL thickness

IV. Discussion

This main goal of glaucoma management is to diagnose disease when it is asymptomatic. Visual field testing is essential in diagnosis and monitoring of glaucoma but standard perimerycan not detect VF defects until 20-4% of ganglion cells have been lost. Nowadays RNFL defects have been objectively demonstrated earlier than Visual Field defects with new investigative technologies. Measuring macular RGC, GCIPL and RNFL thickness by OCT enables an objective and quantitative assessment of glaucomatous structural loss. Mwanza et al. showed that Cirrus OCT had an excellent intravisit and intervisit reproducibility of RNFL thickness and ONH parameters. Hong et all. also reported reproducibility of Cirrus HD-OCT to analyse peripapillary RNFL thickness was excellent

in healthy eyes.

V. Conclusion

SD-OCT of the macula and optic nerve are excellent adjuvant modality for evaluating glaucoma patient and can increase the detection of glaucoma disease in its earlier stage and monitoring the patient during the course of treatment aptly modifying the treatment while gauging the parameters of macula (average GCIPL and minimum GCIPL Thickness) and RNFL thickness. Early diagnosis of glaucoma and early initiation of treatment is very important so that further vision loss can be stopped or slowed down. The evaluation by SDOCT is not superior to ophthalmologist as data acquired from SD-OCT can only guide us. It should be evaluated with the clinical findings of glaucoma patients.

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EVIDENCE BASED APPROACH

Glaucoma and Hypertension

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Introduction -

Glaucoma d escribes a group of optic neuropathies that result in progressive loss of ganglion cells. It manifests as characteristic optic disc cupping, nerve fibre layer losses and visual field defects. Despite extensive clinical and experimental studies, the mechanisms underlying the development and progression of glaucoma remain unclear. An improved understanding of risk factors and how they interact to produce glaucoma can help us better to select potential treatment strategies . While IOP reduction remains the only effective treatment for glaucoma ,many patients with apparently 'adequate' IOP reduction still suffer vision loss.This inconsistent role of IOP control in glaucoma progression may be explained by the involvement of other risk factors, including older age, positive family history, ethnicity , thinner central corneal thickness, associated myopia, diabetes and blood pressure (BP) abnormalities. High IOP, low blood pressure and the presence of long-standing diabetes may all converge to manifest as abnormal blood flow to the optic nerve. Thus a better understanding of the role that blood flow plays in glaucoma might account for these apparent ‘clinical inconsistencies’.

The interplay between blood pressure and IOP determines the ocular perfusion pressure (OPP), which regulates blood flow to the optic nerve .OPP represents a gradient between blood pressure and IOP (in simplistic terms, OPP = BP – IOP. Evidence suggests that abnormal blood flow and changes in the eye's ability to buffer against changes in perfusion pressure (a process known as autoregulation) are central to glaucoma pathogenesis. As blood flow varies with OPP, the eye's ability to maintain blood flow becomes impaired (reduced oxygen and nutrients), thus promoting neuronal dysfunction.

Pathophysiology - High BP could increase IOP by

- increased production of aqueous humor by means of elevated ciliary blood flow and capillary pressure
- decrease aqueous outflow as a result of increased episcleral venous pressure

According to the mechanical theory , elevated IOP causes direct or indirect damage at the level of the optic nerve head. However, low BP, whether spontaneous or secondary to antihypertensive therapy, can reduce OPP, leading to ischemic damage of the optic nerve . The vascular theory emphasizes the importance of vascular perfusion of the optic nerve head and considers glaucomatous optic neuropathy to be the result of unstable blood supply of the optic nerve head due to vascular dysregulation.

Autoregulation - Autoregulation of blood flow is defined as the intrinsic ability of an organ to maintain constant blood flow despite changes in perfusion pressure. When there is a change in OPP , the retina tends to maintain its blood flow . In normal human subjects, autoregulation at the optic nerve is effective for IOP below 27–30 mmHg. This represents an OPP reduction of some 40–50 per cent from baseline for a mean arterial pressure of 100 mmHg (2/3*100–IOP).At the other extreme, blood flow remains unchanged until the OPP is elevated by more than 30 ± 8 per cent above baseline , which in human studies is usually achieved by blood pressure elevation via isometric exercise.

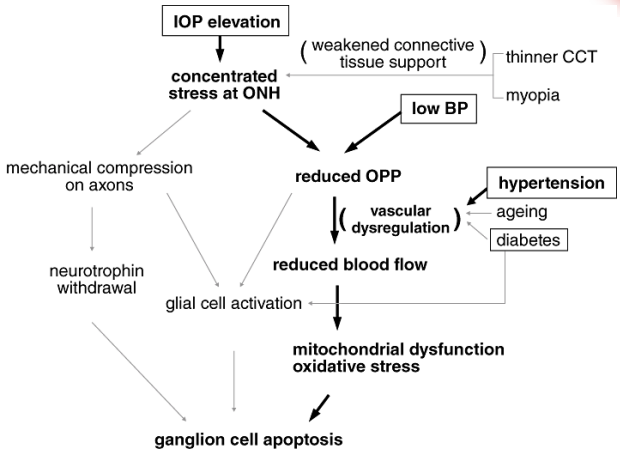

Figure 1, BP = blood pressure, CCT = central corneal thickness, IOP = intraocular pressure, ONH = optic nerve head, OPP = ocular perfusion pressure.

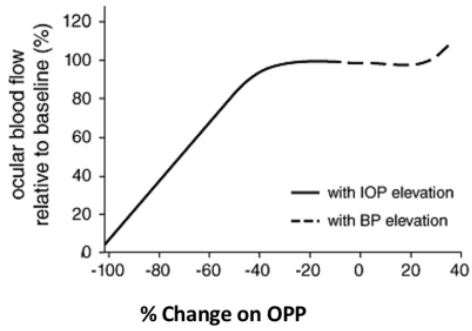

Figure 2. : Ocular blood flow in healthy human subjects remains stable over a range of ocular perfusion pressure levels. Ocular perfusion pressure changes are induced either by blood pressure elevation (dashed curve) or by IOP elevation (solid curve).

Ocular autoregulation involves both myogenic and metabolic mechanisms, through the action of endothelium-derived vasoactive factors that modulate smooth muscle tone and pericytes. In the eye, the role of hormonal components (epinephrine and norepinephrine) in autoregulation is relatively minor as there is no autonomic innervation of retinal and ONH blood vessels; however, it is worth noting that the choroidal circulation has strong autonomic input. In addition, alpha- and beta-adrenergic as well as cholinergic receptors are present on ocular blood vessels.Thus, higher systemic concentrations of catecholamines, as seen in hypertension or higher local concentration due to the use of anti-glaucoma agents (beta-blocker and alpha agonists), may impact local blood flow.

Estimation of ocular perfusion pressure -

Perfusion pressure usually represents the difference between arterial and venous pressure. In the eye, venous pressure approximates the IOP, so that mean OPP can be taken as the difference between the mean ophthalmic artery pressure ($MAP_{ophthalmic}$) and IOP. $MAP_{ophthalmic}$ is not readily measured in clinical practice. Instead, the brachial arterial pressure ($MAP_{brachial}$) is most commonly measured in clinical settings using an arm cuff sphygmomanometer in an upright position. This is a useful estimate of $MAP_{ophthalmic}$ as the blood pressures in both the ophthalmic and brachial arteries are related in the absence of vascular pathology. Measured by ophthalmodynamometry, systolic ophthalmic arterial pressure is approximately three-quarters the systolic brachial arterial pressure, whereas diastolic ophthalmic arterial pressure is approximately two-thirds of the diastolic brachial arterial pressure.

$$OPP = \frac{2}{3} \cdot MAP_{brachial} - IOP \qquad MAP = DBP + \frac{1}{3}(SBP - DBP)$$

It has been hypothesized that both elevated IOP and blood pressure might be driven by a common extrinsic factor such as an age-related increase in sympathetic tone.[1] The Baltimore Eye Survey [6] found that the association between blood pressure and glaucoma is age dependent. In particular, systemic hypertension appears to be protective against glaucoma in younger patients, whereas it increases the risk of glaucoma in older patients. The authors speculate that the optic nerve benefits from high perfusion pressure (that is, high blood pressure) when blood vessels are normal early in life, but as the vessels undergo atherosclerosis to become rigid and narrow with age there will be increased resistance to blood flow as well as compromised oxygen and nutrient exchange at the capillary beds, such that high blood pressure is no longer beneficial [7] In general, each 10 mmHg rise in systolic blood pressure is associated with 0.27mm Hg increase in IOP and for 5 mm Hg increase in diastolic BP , iop increase is 0.17 mm Hg. [2-5]

The Los Angeles Latino Eye Study[8] showed that both low diastolic and high systolic blood pressure are associated with an increased prevalence of open-angle glaucoma. Figure 4, redrawn from that study, shows that the relationship between glaucoma prevalence and diastolic blood pressure is 'U' shaped, indicating that

patients at both extremes of the blood pressure spectrum are at greater risk of glaucoma. This apparent paradox at the extremes can be explained by two factors; one is that patients with hypotension suffer from low OPP at the ONH and the second is that those with chronic hypertension develop atherosclerosis over time leading to increased vascular resistance and compromised vascular autoregulation, as well as impaired nutrient exchange in the capillary beds at the ONH.

Fluctuations in OPP from high or low BP can lead to unstable ocular blood flow and oxygen supply and to oxidative stress which may be relevant in the pathogenesis of glaucoma. Some glaucoma patients have altered autoregulation of their ocular blood flow, but also present with features of a more generalized vascular dysfunction, such as cold extremities, a syndrome known as primary vascular dysregulation. The Flammer syndrome, which is associated with an increased prevalence of NTG, describes a phenotype of subjects with a variety of symptoms and signs and altered vascular response to stimuli such as cold, physical, chemical, or emotional stress.

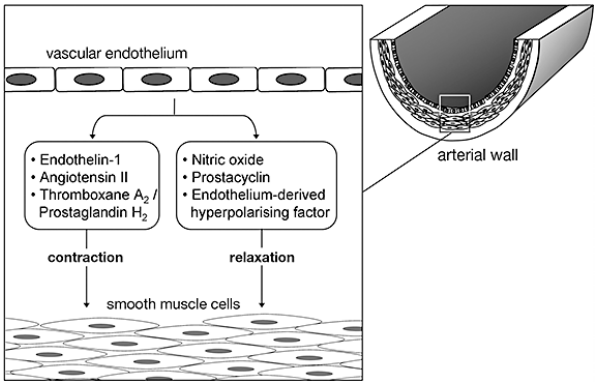

Figure3 : : Autoregulation of ocular blood flow

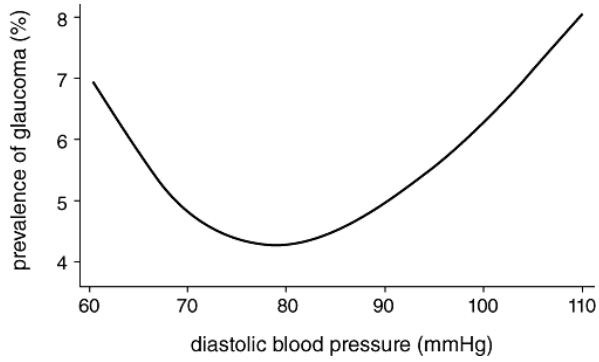

Figure 4 : Relationship between prevalence of open-angle glaucoma and diastolic blood pressure, redrawn from the Los Angeles Latino Eye Study [8]

Ambulatory BP monitoring provides the average of BP readings over a defined period, usually 24 hours. Compared with office BP, it is a better predictor of hypertension-mediated organ damage and of cardiovascular events, such as stroke or coronary artery disease. Normally, BP decreases during the night. Both nondippers (usually defined as a <10 % decrease in nighttime BP compared to daytime BP) and extreme dippers (usually defined as a ≥20 % nocturnal dip) have an increased cardiovascular risk, although data are less convincing for extreme dippers .There are also variations in the IOP, mainly driven by body position. An increase in IOP is observed in the supine position, which is the sleeping position during the night. Consequently, OPP decreases during sleep. In addition to the effect of the normal circadian variation for BP and of the body position for IOP, antihypertensive medications and antiglaucoma medications also can influence OPP.

As stated above, OPP decreases during sleep. Studies relating BP and glaucoma progression mainly focused on the 24-hour variations in BP and, especially, on nocturnal BP. A meta-analysis included 5 studies (286 patients) with well-described 24-hour ambulatory BP monitoring method, clear report of daytime BP, nighttime BP and nocturnal dipping, and assessment of VF over an observation period of at least 2 years. Patients with POAG and NTG, and with or without arterial hypertension, were included. Although systolic and diastolic diurnal and nocturnal BP between patients with or without progressive VF loss were not different, nocturnal dips over 10 % in systolic or diastolic BP were significantly associated with deterioration of the VF.[9]

In a cross-sectional study including 314 consecutive patients with POAG or NTG (202 hypertensive and 112 normotensive), extreme dippers with daytime systemic normotension had more VF loss than extreme dippers with daytime systemic hypertension. Based on these results, a Dresden safety range was defined as a mean nocturnal BP between 65 and 90 mm Hg, provided IOP is well controlled (12 mm Hg in this study). The authors suggest that patients within this range are expected not to progress or to have a slower glaucoma progression compared to patients outside this range. However, given the cross-sectional design of the study, no follow-up data are provided.[10]

The landmark SPRINT study (Systolic Blood Pressure Intervention Trial) shows that a systolic BP target of <120 mm Hg is more protective than the conservative systolic BP target of <140 mm Hg.In 2017, the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines recommended a target BP of <130 mm Hg (systolic) and <80 mm Hg (diastolic) for most hypertensive patients.Consequently, the number of over-dippers and of patients with glaucoma progression could potentially increase in the next decades in treated hypertensive patients. Unfortunately, to our knowledge, neither the SPRINT trial nor other studies comparing outcomes with different BP targets did include assessment of glaucoma incidence or progression.

Summary

- Both high BP and low BP are associated with an increased risk of glaucoma.
- There is mounting evidence that low nighttime BP or excessive dipping could adversely affect glaucoma progression.
- If any, systemic antihypertensive drugs have minimal effect on IOP.

Recommendations

- Patients with high BP should be screened for glaucoma, and patients with glaucoma should be screened for arterial hypertension.
- Patients with coexisting glaucoma and high BP should undergo closer ophthalmologic examinations.
- Ambulatory BP monitoring should be performed in glaucoma patients with unexplained deterioration of their VF, and perhaps in all patients with coexisting arterial hypertension and glaucoma.
- Identification of a low nocturnal BP or over-dipping should prompt discussion between glaucoma and hypertension specialists in charge of the patient. However, to date, no specific recommendation can be proposed to limit over-dipping. Importantly, the nocturnal BP fall has prognostic implications, reduced and reverse dipping being associated with a significantly higher rate of cardiovascular events
- In such an uncomfortable situation (glaucoma progression and over-dipping), there may be room for precision medicine and tailored management, always prioritizing BP control for optimal cardiovascular protection, taking into account that one size does not fit all.

Glaucoma and hypertension scientific societies should join their efforts to stimulate research and studies to determine the best treatment strategy for systemic hypertensive patients with concomitant glaucoma. In particular, the role of 24-hour ambulatory BP monitoring should further be studied, as it shows promise as a useful tool for identifying those patients at highest risk for glaucoma progression.

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EVIDENCE BASED APPROACH

Management of Ocular Surface Disease in Glaucoma

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Introduction

In glaucoma patients, Ocular Surface Disease (OSD) is exacerbated by multiple factors, including preservative-containing glaucoma drops, antimetabolite exposure, and disruption of tear film distribution related to bleb formation [3,4]. OSD spectrum in glaucoma patients include allergy, medicamentosa, punctate keratitis, corneal ulceration, unstable tear film, and cicatrizing conjunctivitis, limbal stem cell deficiency. Management of OSD in glaucoma patients can be quite challenging, especially while concomitantly managing glaucoma for which topical medications are usually firstline therapy.

The Importance Of Managing Ocular Surface Disease In Glaucoma Patients

OSD reduces the compliance to antiglaucoma medications & this may cause progression of glaucoma(5,6). Chronic use of preservative containing antiglaucoma medications cause subconjunctival inflammation and fibrosis which reduces the success of subsequent glaucoma surgery & also increases the rate of postoperative infections (7,8). A healthy ocular surface is also crucial for preoperative intraocular lens measurement accuracy and postoperative patient satisfaction. OSD also affects the quality of diagnostic tests (OCT & Perimetry) & this can make medical and surgical decision making more difficult (9,10). Most importantly, OSD significantly reduces the quality of life among glaucoma patients (11, 12). A specific management approach is needed to address OSD in glaucoma

Developments In The Diagnosis Of Ocular Surface Disease

In OSD signs & symptoms may not always correlate. A patient may have minimal surface staining but significant symptoms or may present with severe surface staining and MGD while being asymptomatic (13). Its important to identify those patients who require treatment. Patient surveys, such as the standardized patient evaluation of eye dryness questionnaire, are simple to implement and can identify patients with OSD who may otherwise go unnoticed (14). Recently, an ocular surface frailty index for predicting OSD symptom onset after cataract surgery has been described (15). This index is based upon tear break up time, osmolarity, surface staining and Schirmer's test. It would be valuable to apply a similar index to patients undergoing glaucoma intervention to identify those requiring active treatment.

Recent research has demonstrated increased levels of IL-6, TNF-a, and VEGF in tear samples of glaucoma patients on chronic topical prostaglandins when compared with patients with dry eye not receiving any glaucoma therapy. Of note, patients on preservative-free prostaglandins had lower expression of IL-1b (16). A 2019 study demonstrated elevated MMP-9 levels in 46.7% of patients on preservative-containing glaucoma medications versus 16.7% on preservative-free medications or untreated patients. These biomarkers can be potentially used to develop simple diagnostic tests to identify OSD in glaucoma patients

Chronic ocular surface inflammation in glaucoma patients may manifest as dysfunction of the meibomian glands,

leading to an evaporative dry eye state with resultant hyperosmolarity. Osmolarity testing can be easily performed in a busy clinic using a handheld osmometer to collect a tear sample, which is analyzed in several seconds (TearLab Corporation, Escondido, CA, USA). Hyperosmolarity indicates more rapid tear evaporation and has been demonstrated in eyes treated with topical glaucoma medications (18, 19)

MGD has been reported in up to 80% of patients on glaucoma drops (20). Infrared meibography has become a valuable tool to assess gland structure and is performed in approximately 5 min using one of multiple available noninvasive imaging systems, such as LipiView (Johnson&Johnson, New Brunswick, NJ, USA). Significant gland truncation or loss may indicate the need to modify the glaucoma regimen, add anti-inflammatory agents, start rigorous eyelid hygiene, or utilize in-office treatments such as thermal pulsation and intense pulsed light. Lipid layer thickness (LLT) was found to be significantly thinner in patients on glaucoma medications than in normal eyes, and a longer duration of medication use and greater number of drops were associated with a thinner LLT. Tear film extensional viscosity, a measure of tear film integrity, has also been recently described as a promising new biomarker for dry eye disease, and its role in glaucoma patients with OSD remains to be determined (22). These various tests can be performed at baseline and repeated at intervals to monitor treatment interventions.

Advances In The Medical Management Of Ocular Surface Disease In Glaucoma Patients

Preservative containing antiglaucoma medications exacerbate underlying MGD with decreased lipid layer thickness and hyperosmolarity. They also reduce the density of conjunctival goblet cells, thereby reducing the mucin layer of the tear film (23). Apart from this they also have neuroinflammatory effects which is demonstrated by increased nerve tortuosity and dendritic cell density (24). Prior studies have linked trabeculectomy failure to reduced goblet cell density, and a recent report on the development of OSD after trabeculectomy pointed to preoperative chronic glaucoma drop use as a risk factor for postoperative development of dry eye and limbal stem cell deficiency (25, 26).

Multiple strategies have emerged for reducing preservatives exposure in glaucoma patients, including consolidating medications to fixed-dose combination products or switching to preservative-free formulations (28). A recent systematic literature analysis found that prostaglandin analogs, especially preservative-free formulations, had a less detrimental effect on conjunctival goblet cells than other antiglaucoma medications (23). Another study demonstrated significant improvement in Ocular Surface Disease Index (OSDI) scores after 6 weeks of switching from a BAK-preserved prostaglandin to preservative free tafluprost (29). Xelpros (Sun Pharmaceutical Industries, Cranbury, NJ, USA) is a commercially available formulation of preservative free latanoprost 0.005% in a multidose bottle that has been recently introduced to the market. Compounding pharmacies are also releasing fixed-dose combination formulations of preservative free glaucoma drops. The expansion and availability of such formulations would help to reduce the excessive preservative burden and should be strongly considered.

Reducing bacterial load on the ocular surface and eyelid margins is also an important consideration in the management of blepharitis that contributes to OSD. High ocular surface bacterial loads and MGD increase the risk of postoperative infection after glaucoma surgery (8). Hypochlorous acid spray 0.1% twice daily application to the eyelid margin has been shown to reduce the load of Staphylococcus aureus, Staphylococcus Epidermidis and other bacteria with efficacy similar to that of betadine application (30).

Topical steroids are often implemented in the treatment of OSD. In glaucoma patients, steroid use should be closely monitored due to the potential risk of intraocular pressure elevation. Loteprednol etabonate is associated with lower rates of intraocular pressure elevation compared to prednisolone acetate or dexamethasone, and it may be particularly useful when steroids are required in these patients (31). Cequa (Sun Pharmaceutical Industries, Cranbury, NJ, USA) is an aqueous, nanomicellar ophthalmic solution of cyclosporine 0.09% that has been approved to increase tear production in patients with dry eye (32,33). Cyclosporine has been shown to increase the density of conjunctival goblet cells, which are reduced with the chronic use of topical glaucoma medications, and may be of particular benefit to glaucoma patients with OSD (34). Xiidra (Lifitegrast 5.0%, Novartis, Cambridge, MA, USA) is a lymphocyte function-associated antigen (LFA)-1 antagonist that has been recently approved as twice daily application to treat signs and symptoms of dry eye (35– 37). Trials have demonstrated improvement in inferior corneal staining and eye dryness score with use of Xiidra versus placebo, and symptomatic improvement

may begin as early as two weeks of use (37).

The role of Omega-3 fatty acids in managing OSD remains somewhat unclear. Recently, the Dry Eye Assessment and Management (DREAM) study did not find a significant benefit to the use of Omega-3 supplements over the course of 12 months compared to placebo (38). Interestingly, however, the benefit of Omega-3 fatty acids was demonstrated in a study of glaucoma patients with OSD [39]. This prospective, multicenter study of 1,255 patients on topical antiglaucoma drops found that supplementation of 1500mg of Omega-3 fatty acids per day over a period of 12 weeks reduced symptoms of scratching, stinging, grittiness, tired eyes and blurry vision, as well as improvement in Schirmer's test and tear break up time scores.

Advances In Procedural Management Of Ocular Surface Disease

Given the potential challenges related to compliance and side effects when adding dry eye topical medications to a topical glaucoma regimen, procedures to treat OSD may be of particular interest in this patient population. Patients with MGD may benefit from thermal pulsation (e.g., LipiFlow, Johnson & Johnson Vision, Jacksonville, FL, USA) or Intense Pulsed Light (e.g., OptimaIPLM22, Lumenis, Salt Lake City, UT,USA) (40). Thermal pulsation heats and massages the eyelid margins to liquefy and express gland secretions to improve flow. For patients with significant eyelid margin inflammation, such as those with ocular rosacea, including many patients on chronic glaucoma drops, IPL may be helpful. IPL works by thermal selective coagulation and ablation of superficial blood vessels and telangiectasias of the eyelid skin, reducing the release of inflammatory mediators and tear cytokine levels, and improving meibomian gland outflow. Manual gland expression may be performed after treatment.

Patients with glaucoma and OSD may have aqueous deficiency and dysfunctional tear film circulation. Recent studies have highlighted the utility of punctal plugs in treating these patients (41,42).One randomized controlled study of punctal plug placement in glaucoma patients treated with a prostaglandin analog found reduced OSDI scores, increased tear break up time, reduced corneal staining, and reduced osmolarity.

Advances In Glaucoma Interventions And Application To Patients With Ocular Surface Disease

Traditionally, reducing medication burden in glaucoma patients has been challenging. Bleb-based glaucoma surgeries have been associated with a significant postoperative risk profile and may not be ideal for patients with mild to moderate glaucoma (43). With the advent of MIGS and sustained release medications, reducing preservative burden to the ocular surface in glaucoma patients is becoming possible earlier in the disease course. Durysta (Allergan plc, Dublin, Ireland) is the first intracameral, sustained release medication implant to be approved by the FDA (44,45 & 46). Durysta is a bimatoprost implant which is administered through injection into the anterior chamber in patients with open angle glaucoma or ocular hypertension. This implant is approved for one-time injection per eye and is reported to be effective for 4–6 months. The ARTEMIS 1 study found noninferiority of Durysta to timolol over the 12-week study period with up to two additional administrations at 4-month intervals. An animal study has shown low concentrations of bimatoprost in the ocular surface tissues and increased concentration at the ciliary body compared to topical administration. Durysta may be particularly helpful in giving patients with glaucoma and OSD a period of respite from topical prostaglandins in order to give the ocular surface time to improve and minimize inflammation, during which period glaucoma surgery could be performed or preservative-free drops could be tried.

A multicenter randomized controlled trial (LiGHT) recently investigated the role of selective laser trabeculoplasty (SLT) versus eye drops for firstline treatment of ocular hypertension and open angle glaucoma and highlighted the utility of SLT early in the management of glaucoma (47,48). The study demonstrated a higher percentage of patients with intraocular pressure at target, fewer patients requiring glaucoma surgery, and 74.2% of eyes requiring no drops at 36 months in the SLT group. This study supports SLT as a first-line treatment for glaucoma.

Multiple trabecular bypass and angle-based MIGS procedures are now available and have the potential to greatly reduce glaucoma drop burden in patients with mild and moderate disease. Given the safety and efficacy of these approaches, patients may be candidates for earlier surgical intervention. The reduction in eyedrop burden postoperatively after iStent Inject (Glaukos Corporation, San Clemente, CA, USA) or Hydrus Microstent

(Ivantis, Inc, Irvine, CA, USA) is reported to be significant (49). The COMPARE study demonstrated an average medication reduction of 1.6 drops after implantation of the Hydrus compared to reduction of 1 drop in patients who received the iStent Inject (P=0.004). Performing MIGS earlier with or without cataract surgery may reduce ocular toxicity related to chronic drop administration. MIGS may also avoid potential tear film circulation issues, bleb dysesthesia, and side effects of antifibrotic agents that are often seen with traditional glaucoma surgeries (43). For those patients suffering from OSD without visually significant cataract, SLT is often a good first step. Goniotomy, viscocanaloplasty, GATT, trabectome, and endocyclophotocoagulation are available as standalone surgical options without the need for concomitant cataract extraction.

Conclusion

OSD in glaucoma patients is generally underdiagnosed and undertreated. Managing glaucoma and OSD simultaneously presents numerous challenges. Concomitant treatment of OSD along with glaucoma may yield better outcomes for both OSD & glaucoma. Recent advances in diagnostic testing, anti-inflammatory agents, and procedural interventions for OSD have been encouraging. For glaucoma, the availability of preservativefree medications, fixed-dose combination products, sustained-release delivery devices, and MIGS may reduce both the medication burden and preservative exposure and may also enhance surgical outcomes of future bleb-based surgery. Importantly, more research is necessary to better understand the multifaceted relationship between glaucoma and OSD and to gain valuable insights from evidence-based treatment algorithms for the management of these complex patients.

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and istent microinvasive glaucoma surgery implants for standalone treatment of open-angle glaucoma: the COMPARE study. Ophthalmology 2020; 127:52–61.

IMAGE ESSAY

AN OVERHANGING BLEB

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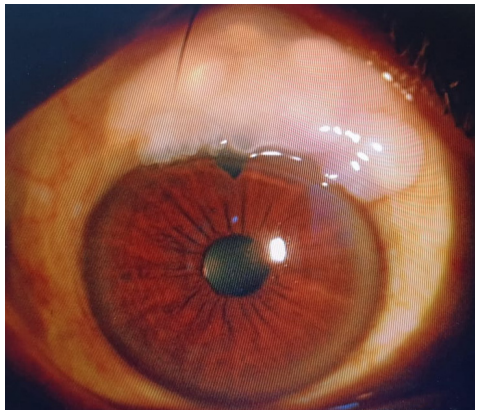

Slit Lamp image showing overhanging filtering bleb.

Introduction

Overhanging blebs are overfiltering blebs that project over cornea. Though rare, they are known complication post trabeculectomy[1]. They cause discomfort, watering, visual disturbances due to astigmatism[2]. Excision of the overhanging part is usually the preferred treatment[1]. Here we would like to discuss a case overhanging bleb through a photo essay.

Case Report

A 65 year old male came to our OPD with complaints of foreign body sensation in right eye since one year. There was history of trabeculectomy with mitomycin C 4 years before in the same eye. Intraocular pressure was maintained at 14mmhg in this eye with visual acuity of 6/60. An avascular, thin walled cystic bleb was seen on slit lamp examination. Resection of the overhanging bleb was done under local anaesthesia for this patient.

Discussion

Overhanging blebs can occur days to years after trabeculectomy[3,4]. The incidence of overhanging bleb has increased with increased use of antimetabolites in glaucoma filtering surgery[2,4]. It has been postulated that an overhanging bleb is filtering cicatrix that grows downward due to action of the upper eyelid[1]. Ulrich et al[5] suggested that formation of overhanging blebs involved aqueous humor dissection between the corneal epithelium and the stroma. Ou-Yang[6] claimed that anti- proliferative action of antimetabolites alongwith aqueous humor and gravity cause overhanging filtering bleb especially in older population. Overhanging blebs produce symptoms like foreign body sensation, epiphora, visual disturbances due to astigmatism and poor cosmesis[6].Medical management of overhanging blebs include lubricants. Various surgical approaches includes excision of the corneal portion of the bleb[1], cryotherapy[7], argon laser[8], and Nd:yag laser[3]. Compression sutures have also been described in the management of overhanging blebs[4]. Overall, the management of overhanging blebs requires careful consideration of the patient's individual needs and circumstances. With proper management, most patients with overhanging blebs can achieve improved comfort and vision.

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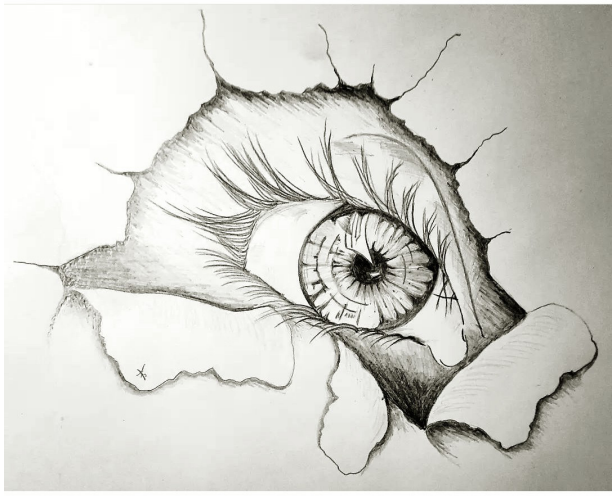
BEYOND EYE

Dr Varsha Vaishnav
Consultant Opthalmologist, Ronak Hospital, Bhopal
Email: drvarsha_2669@yahoo.com



PRECEPTORS OF FUTURE – WEBINARS

PRECEPTORS OF FUTURE

Scientific competitive webinars for PGs of divisional societies of MPSOS

BHOPAL DIVISION WEBINAR

15-01-2023, SUNDAY (3 pm - 5 pm)

S NO	PG NAME & COLLEGE	MENTOR	TOPIC
1	Dr Nishi Prasad (GMC)	Dr Kavita Kumar	Recent advances in Myopia Management
2	Dr Bhawna Parmar (GMC)	Dr Vivek Som	TASS- toxic anterior segment syndrome
3	Dr Amir Munshi (GMC)	Dr Suraj Kubrey	Fungal corneal ulcer management
4	Dr Garvita Khandelwal (GMC)	Dr Aditi Dubey	Glaucoma drainage devices in India
5	Dr Swati Singh (LNMC)	Dr Rahul Agrawal	Corneal topography - how to interpret
6	Dr Mehek Gupta (PCMS&RC)	Dr Harpal Singh	How to read single HVF print out
7	Dr Aishwarya Raghuvanshi (SSEH)	Dr Prerna Upadhyay	Collagen cross linking
8	Dr Aditya Sharma (BMHRC)	Dr Hemlata Yadav	EDOF- Extended depth of Focus IOLs
9	Dr Karthik Iyer (CMC&RC)	Dr Ulka Shrivastava	Treatment of Diabetic macular oedema
10	Dr Sumita Chaturvedi (CMC&RC)	Dr Khalid khan	How to asses Ptosis
11	Dr Rajat Singh Yadav (RKDFMC)	Dr Vasudha Damle	Vision assessment in infant & children
TOTAL	11 presentations of 5 min each		5-min Discussion after each presentation

Coordinators for Preceptors of Future PGs Program

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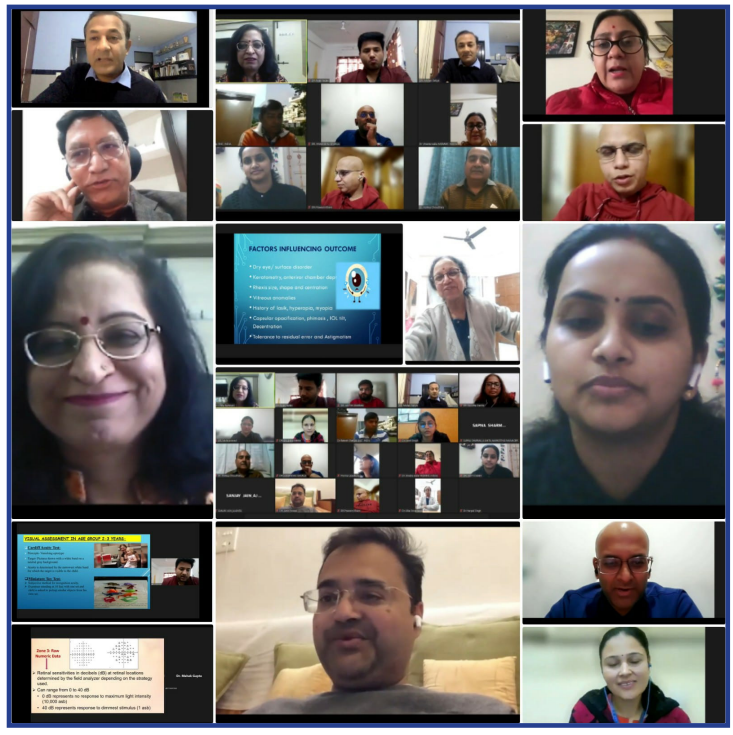
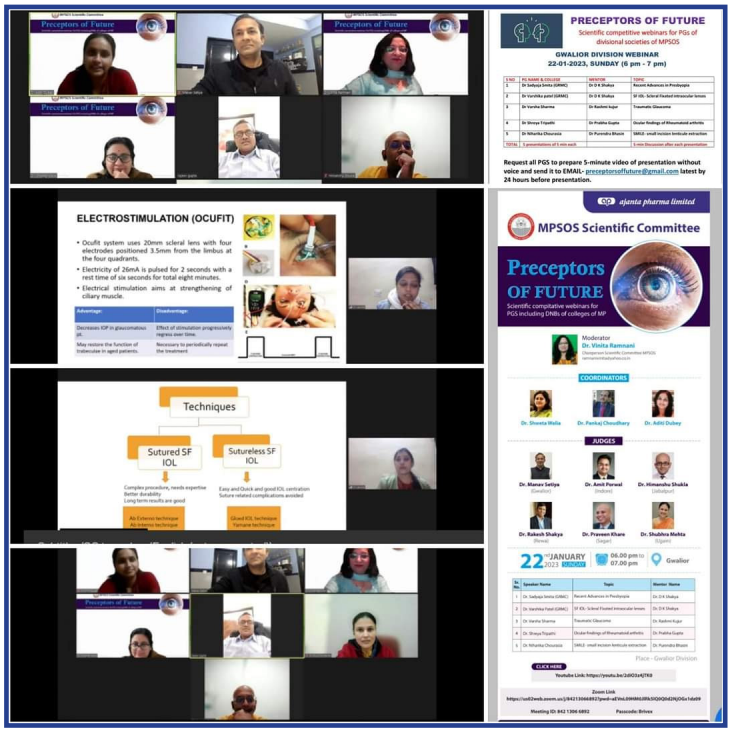
•Dr Pankaj Choudhary (Mob 9826468847)

•Dr Aditi Dubey (Mob 9340787807)



BHOPAL (15/01/2023)



PRECEPTORS OF FUTURE

Scientific competitive webinars for PGs of divisional societies of MPSOS

GWALIOR DIVISION WEBINAR

22-01-2023, SUNDAY (6 pm - 7 pm)

S NO	PG NAME & COLLEGE	MENTOR	TOPIC
1	Dr Sadyaja Smita (GRMC)	Dr D K Shakya	Recent Advances in Presbyopia
2	Dr Varshika patel (GRMC)	Dr D K Shakya	SF IOL- Scleral Fixated intraocular lenses
3	Dr Varsha Sharma	Dr Rashmi kujur	Traumatic Glaucoma
4	Dr Shreya Tripathi	Dr Prabha Gupta	Ocular findings of Rheumatoid arthritis
5	Dr Niharika Chourasia	Dr Purendra Bhasin	SMILE- small incision lenticule extraction
TOTAL	5 presentations of 5 min each		5-min Discussion after each presentation

Request all PGS to prepare 5-minute video of presentation without voice and send it to EMAIL- preceptorsoffuture@gmail.com latest by 24 hours before presentation.

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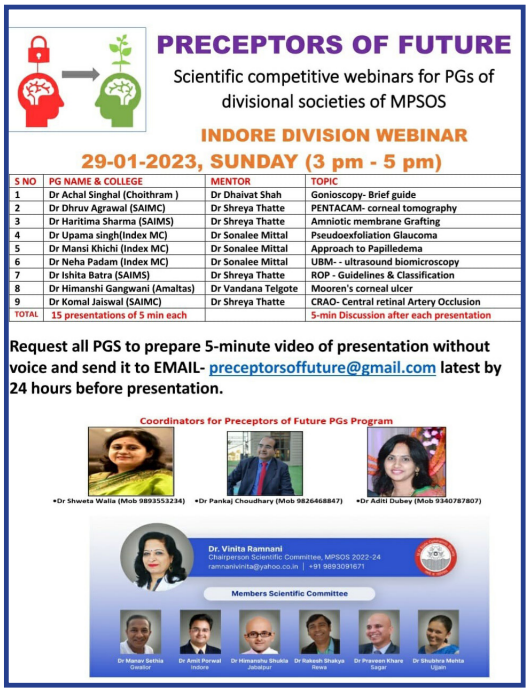
GWALIOR (22/01/2023)



PRECEPTORS OF FUTURE

Scientific competitive webinars for PGs of divisional societies of MPSOS

INDORE DIVISION WEBINAR

29-01-2023, SUNDAY (3 pm - 5 pm)

S NO	PG NAME & COLLEGE	MENTOR	TOPIC
1	Dr Achal Singhal (Choithram)	Dr Dhaivat Shah	Gonioscopy- Brief guide
2	Dr Dhruv Agrawal (SAIMC)	Dr Shreya Thatte	PENTACAM- corneal tomography
3	Dr Haritima Sharma (SAIMS)	Dr Shreya Thatte	Amniotic membrane Grafting
4	Dr Upama singh(Index MC)	Dr Sonalee Mittal	Pseudoexfoliation Glaucoma
5	Dr Mansi Khichi (Index MC)	Dr Sonalee Mittal	Approach to Papilledema
6	Dr Neha Padam (Index MC)	Dr Sonalee Mittal	UBM- - ultrasound biomicroscopy
7	Dr Ishita Batra (SAIMS)	Dr Shreya Thatte	ROP - Guidelines & Classification
8	Dr Himanshi Gangwani (Amaltas)	Dr Vandana Telgote	Mooren's corneal ulcer
9	Dr Komal Jaiswal (SAIMC)	Dr Shreya Thatte	CRAO- Central retinal Artery Occlusion
TOTAL	15 presentations of 5 min each		5-min Discussion after each presentation

Request all PGS to prepare 5-minute video of presentation without voice and send it to EMAIL- preceptorsoffuture@gmail.com latest by 24 hours before presentation.

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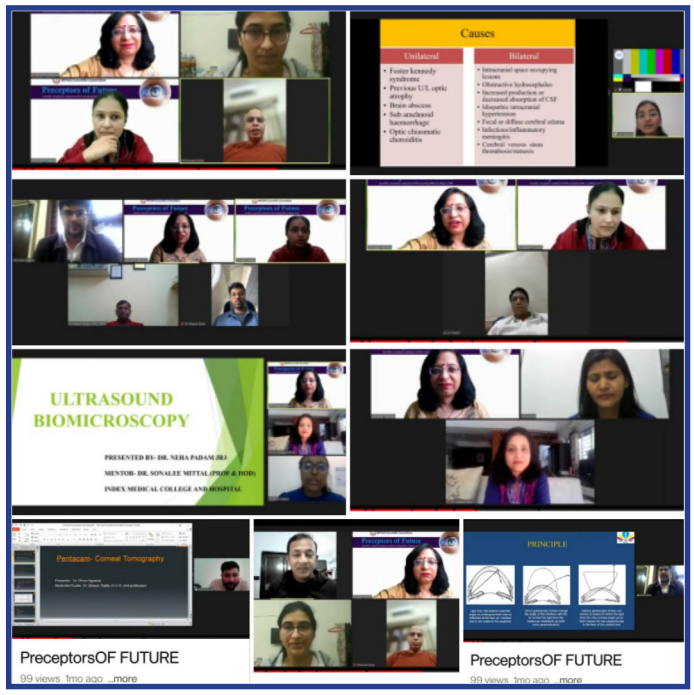
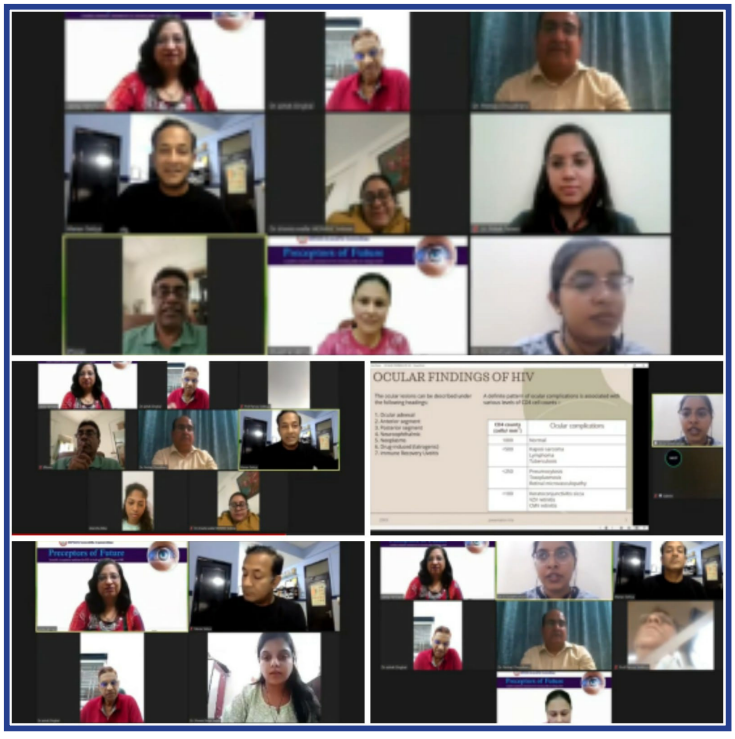
•Dr Aditi Dubey (Mob 9340787807)



INDORE (29/01/2023)



PRECEPTORS OF FUTURE

Scientific competitive webinars for PGs of divisional societies of MPSOS

JABALPUR DIVISION WEBINAR

19-02-2023, SUNDAY (3 pm - 4 pm)

S NO	PG NAME & COLLEGE	MENTOR	TOPIC
1	Dr Palak Patwa (NSCBMC)	Dr Navneet Saxena (Prof.& Head)	Anterior Segment OCT
2	Dr Sowmya Iyer (NSCBMC)	Dr P. A. Siddiqui (Professor)	Ocular findings of HIV
3	Dr Sheweta Singh Jadon (NSCBMC)	Dr U. P. Deepankar (Professor)	keratoprosthesis
4	Dr Akanksha Ekka (NSCBMC)	Dr P. Warkhede (Designate Professor)	Trifocal IOLs
TOTAL	4 presentations of 5 min each	5-min Discussion after each presentation	

Request all PGS to prepare 5-minute video of presentation without voice and send it to EMAIL- preceptorsoffuture@gmail.com latest by 24 hours before presentation.

Coordinators for Preceptors of Future PGs Program

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•Dr Pankaj Choudhary (Mob 9826468847)

•Dr Aditi Dubey (Mob 9340787807)

JABALPUR (19/02/2023)

PRECEPTORS OF FUTURE

Scientific competitive webinars for PGs of divisional societies of MPSOS

REWA DIVISION WEBINAR

26-02-2023, SUNDAY (3 pm - 5 pm)

S NO	PG NAME & COLLEGE	MENTOR	TOPIC
1	Dr Prathamesh Agashe (SNC)	Dr Gautam Parmar	Fuchs endothelial dystrophy
2	Dr Drashti A. Kathiriya (SNC)	Dr Alok Sen	BSCAN
3	Dr Aishwary Duddalwar(SNC)	Dr Navjot Singh Ahluwalia	Dry eye treatment
4	Dr Akshita Gupta (SNC)	Dr Rakesh Shakya	MMC (mitomycin C) in ophthalmolog
5	Dr. Aashi Jain	Dr Sujata Lakhtakia	HSV- Keratitis
6	Dr. Dhirendra Kumar Pandey	Dr Charudatt Chalisgaonkar	CRVO – Central Retinal Vein Occlusio
7	Dr. Pankaj Kushwaha	Dr Sujata Lakhtakia	ENOPHTHALMITIS
8	Dr. Sonu Badole	Dr Anamika Tiwari	Lens Induced Glaucoma
9	Dr Urvashi Dwivedi.	DrCharudatt Chalisgaonkar	Virtual Reality Perimeter
10	Dr Aditi Mishra	Dr Anamika Tiwari	Dacryocystitis
TOTAL	10 presentations of 5 min each		5-min Discussion after each presentation

Request all PGS to prepare 5-minute video of presentation without voice and send it to EMAIL- preceptorsoffuture@gmail.com latest by 24 hours before presentation.

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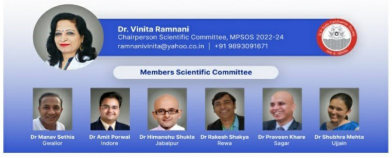
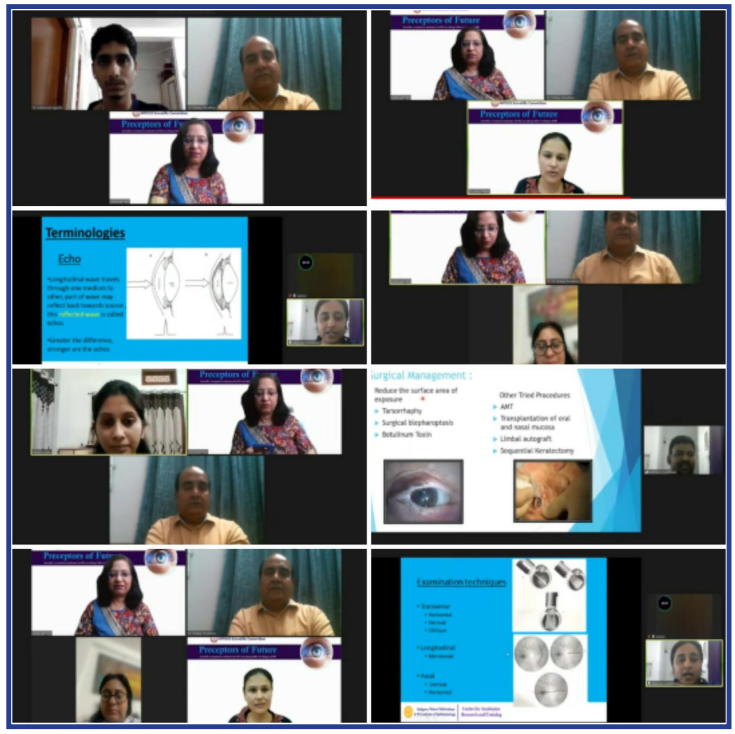
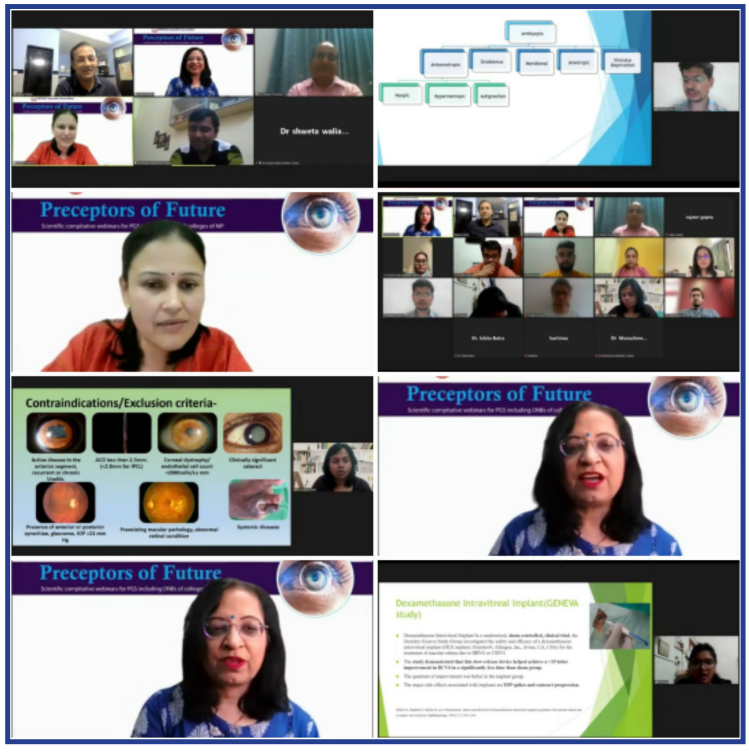
•Dr Pankaj Choudhary (Mob 9826468847)


•Dr Aditi Dubey (Mob 9340787807)





REWA (26/02/2023)



PRECEPTORS OF FUTURE

Scientific competitive webinars for PGs of divisional societies of MPSOS

UJJAIN DIVISION WEBINAR

12-03-2023, SUNDAY (3 pm – 5 pm)

S NO	PG NAME & COLLEGE	MENTOR	TOPIC
1	Dr Yuvraj Hardia (RDGMC)	Dr Shubhra Mehta	Steroid induced Glaucoma
2	Dr Kshitiz Gupta (RDGMC)	Dr Shubhra Mehta	Astigmatism
3	Dr Simar kalra (RDGMC)	Dr Manoj Mehta	Toric IOL
4	Dr. Shivangi Sahu (RDGMC)	Dr Shubhra Mehta	BRVO- Branch Retinal Vein Occlusion
	From indore division		
1	Dr Trisha Yadu (MGMMC)	Dr Shweta Walia	OHT- Ocular Hypertension
2	Dr Rakshit Agrawal (MGMMC)	Dr V Bhaisare	FLACS -Femtosecond laser assisted cataract su
3	Dr Sakshi Tiwari (MGMMC)	Dr P Rawat	Phakic IOL
4	Dr Rishabh Khare (MGMMC)	Dr Shweta Walia	AMBLYOPIA - diagnosis & treatment
5	Dr Krutika Thorat (MGMMC)	Dr Manushree	Ocular findings in SLE
6	Dr Priya Singh (MGMMC)	Dr Neetu Kori	Proptosis evaluation
7	Dr Haritima Sharma (SAIMS)	Dr Shreya Thatte	Amniotic membrane Grafting
8	Dr Ishita Batra (SAIMS)	Dr Shreya Thatte	ROP - Guidelines & Classification
TOTAL	4 presentations of 5 min each from Ujjain and 6 from indore		5-min Discussion after each presentation

Request all PGS to prepare 5-minute video of presentation without voice and send it to EMAIL- preceptorsoffuture@gmail.com latest by 24 hours before presentation.

Coordinators for Preceptors of Future PGs Program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* In this study, at baseline on latanoprost, patients with IOP ≥21 mmHg in at least one eye were randomized to twice-daily fixed brimonidine tartrate 0.2%/timolol maleate 0.5% combination (n = 102) or timolol 0.5% (n = 102), each adjunctive to latanoprost for 12 weeks. IOP was measured at 8 am or ± 10 am at baseline, weeks 6, and week 12. | 1. Clinical Ophthalmology 2011;5:845-853 | 2. Oman J Ophthalmol. 2018 May-Aug; 11(2): 140–143.

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