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INTERVIEW WITH PADMASHREE DR. T. P. LAHANE

Hon. President, MOS

Dr.T. P. Lahane needs no special introduction.
Here are exerpts of interview of this dynamic personality, conducted by **Dr. Prakash Marathe**, Hon. President, POS, held after the medicolegal workshop in Pune, in January 2010

Dr. P M : I sincerely thank you for spending time with us. We wish to know about your fantastic journey of life, from a small village to achieving a prestigious award like 'Padmashree'. We believe this story of yours is an inspiration to us all & particularly to young ophthalmologist in the country. I want to begin by asking you about your early days & your experience in school .

Dr. T L : First of all, I wish to thank you for interviewing me. I am indeed very glad that, Poona Ophthalmological Society, under your leadership is conducting such good activities (like today mornings workshop on Medicolegal issues). I congratulate you for this. I also wish all your members a very happy & prosperous new year. I hail from very humble background. I belong to a poor farmers family. I was born & brought up in a small village-Makegaon, Tal:Renapur. I have 4 sisters & 2 brothers. My father owned about 2,1/2 acres of farm land.
I started schooling at our village school. I was admitted there because there weren't many students to keep the school running. I would attend school, only after work in the farm was done or when there was no work in the farm(between two seasons).
I remember my mother carrying us brother & sisters on her arms & shoulders to & from school. It so happened that since I scored good marks in 4th standard, my school teacher coxed my parents to continue my schooling. All through my schooling, there was very little formal training given. I used to study mostly when I would take cattle out for grazing or when there was no farm work. I had the additional duty of taking care of younger siblings.

Dr. P M : Was there any ideal who was your inspiration, to make you attend school & continue education?

Dr. T L : Well to be frank, there was no such person or persons who inspired me. When I was in 7th standard, I realized that the only way out of my present situation was to get educated. I probably had thought of becoming a school teacher, because they were the only educated people around. I remember one of my school teachers, Mr. Bhondve, who I looked upto & admired him for his knowledge. I even didn't get opportunity to see enough of my surroundings, to help me form any opinion. I happen to see the State transport(S. T.) bus only in my 5th std, since my village is 14 km from the main road.

Dr. P M : So given these circumstances how did you decide to become a doctor?

Dr. T L : As I said, there was this realization that acquiring education was the only way to improve the financial situation of my family. Since I was good in studies, when I was in

10th std, our school headmaster suggested that I should take up science, which would give me a chance to take up medicine later.(Till then I knew only one person from my region- one of my seniors, who (had got admission to MBBS). But the main hurdle was to arrange for paying the college fees. My monthly expenses were approximately Rs. 30 and even this was difficult to get from my father. During this period there was a scheme by the university, the 'earn & learn' scheme for students from economically backward families. I enrolled myself for this scheme. The deal was to water 100 saplings (in 8 days) in return for a monthly stipend of Rs.30. This one opportunity kept alive my dream to learn.

Dr. P M : Did you ever have any inferiority complex or did you feel yourself different from other students?

Dr. T L : Yes. I had some feeling of inferiority complex. Since I belonged to a poor farmers family, my style of living was far simpler than others in my college. However I had a constant thought in my mind to study hard & study more hours than others. This was the only way to get admission for MBBS. I was working part time as well, so I had to study late in the night, sometimes by tying my hair with a string to the ceiling, so that I would not fall asleep.
My father told me to come home & start working in the farm, after giving my interscience exam. I thought that as the end of my education. As it happened I stood 10th in my division in interscience & secured admission to Ambejogai Medical College. I remember one my teachers coming 14 kms by cycle to my village to tell my family this good news.

Dr. P M : Tell us about your experience during MBBS.

Dr. T L : Getting admission to MBBS was fine, but arranging to pay the fees was a very difficult thing. I took a loan of Rs.1000 for paying the course fees. Few of my friends had started a mess, where I volunteered to be manager, in return for two square meals. I continued doing this for all 4 & ½ years. Then when I was in the final MBBS, our local MLA, Mr.Raghunathrao Munde came & offered his daughters hand in marriage. My only precondition was that they should first visit my family in my village (to know our situation) & if possible help me financially in finishing my education. He graciously agreed to both & I got married. This way I could continue my education.

Dr. P M : What made you take Ophthalmology as your specialization?

Dr. T L : When I was to appear for my ophthalmology & ENT final paper, my father-in-law, unfortunately died in a car accident. Since I din't get time to study during his funeral & performing his last rites, I failed in that paper. I cleared it in my next attempt, with very good marks. I had decided to pursue Orthopedics or Pediatrics. But because of a extra attempt in 3rd MBBS, I could not get into the Residency program in either subjects (although I was getting a pure PG seat). This was important for me, because I would continue my PG only if I got regular stipend in the residency program. Since I was getting admission in the Residency programme in Ophthalmology, I decided to do specialization in this subject. Getting stipend was very critical for me, because I had to support me, my wife & also provide for the education of my younger siblings. I was also quiet active in MARD, during my residency. We had a fight with the state government, against their decision to start private medical colleges, where we felt,

merit would not be the deciding criteria for admission. My name was mentioned in the assembly & I feared that my registration would be cancelled. We had a partial victory, when the government, decided not to start any new colleges (although they had given permission to 3 colleges).

Dr. P M : After you finished your education, how did you decide to enter government service?

Dr. T L : I had passed MPSC exam when I was doing my PG. Hence I was eligible to be a lecturer. I decided to join Ambejogai Medical College. Since I was known as son-in-law of ex-MLA from that region, a lot many people came to me to seek a variety of medical aid. This was mainly for non-ophthalmology related ailments. After few years, we started with free eye camps. We conducted 267 camps where we did 33,000 surgeries.

I thought I had enough experience & had decided to start private practice. One fine day, while taking my daily rounds, I felt uneasy & so went & checked my B.P. I was shocked to know that my B. P. was 260/140. Later I was diagnosed with bilateral renal failure. I was advised bi-weekly dialysis. Unfortunately Ambejogai did not have these facilities. So I had to come to Mumbai for this. This was when I decided to stay in service.

I used to go for dialysis every Thursday & work in the ophthalmology department on all other days. Doctors had predicted that I would not live for more than a few months or a year. I ultimately underwent a renal transplant & here I am, still alive.

Dr. P M : People look at government service from a very negative way. It is said there is too much of red tapeism around, very little work is done efficiently. But you have made us change that perception. How did you achieve so much success in this environment?

Dr. T L : Frankly, I never looked at government service from that angle. I think of government service as if working for a NGO. I have tried to do the best possible in the given circumstances.

Just to give you the measure of success we could achieve. When I started working in this department, there was very limited work being done. We (myself, Dr.Ragini Parekh & others in the department) decided to improve things. I learnt SICS & Phaco. We then decided to take these services to the most remote areas of Maharashtra. Our aim was that these services should reach the poorest of the poor people, like the farm labourers, the tribals, who had no access to these kind of services. This is how we have conducted 154 free eye camps all over the state(plus 72 camps in Mumbai). We did close to 45,000 surgeries in these camps. All these were by the sutureless method. This ensured that the labourer had faster visual rehabilitation & less financial loss, because he/she could start working early. More importantly, there was not a single case of endophthalmitis in any of these camps.

Dr. P M : Did you have to face any difficulties, considering that you were doing a lot of 'extra' work?

Dr. T L : Yes I did face a lot of resistance from people in the administration. I was transferred to Ambejogai in 1998, because of this reason. But even there I continued in the same fashion. During my stay there I did 485 surgeries in only 3 months. I went to the most remote areas, to hold eye camps. I had started becoming quiet popular in that region. This created fear in the minds of some of the local politicians & thinking that I might fight assembly elections, they transferred me back to Mumbai!

Dr. P M : Your work in the department of ophthalmology in Sir.JJ group of hospitals is a landmark effort. Tell us something about it.

Dr. T L : This achievement is not one mans job. Dr. Ragini Parikh & many more dedicated people like her, have made this possible. Today we have 7 phaco machines, 1 Visu, 1 Lumera T with inverter tube microscopes, OCT, Anterior OCT, FFA, ERG, UBM, Automated perimeters & now even an Excimer laser. (We are the first state government ophthalmology department to install an Excimer in this country).
All this was not possible from the government funds. Many of these facilities have been donated to the department by various trusts & other organizations. Government of Maharashtra generously agreed to get these equipment for the department.
Because of all this, we are able to do all kinds of surgeries at no costs or very minimal costs. Infact we have done more than 1200 VR surgeries till date & this has reduced the flow of patients going to Chennai or Hyderabad, from Maharashtra by 60%.

Dr. P M : What advice will you give to people aspiring to join the government service?

Dr. T L : We need more & more committed & dynamic people in government service. As I said earlier, think of government as an NGO. One can achieve a lot of good things, for the society, by taking advantage of government facilities & infrastructure.
One may not earn money here, but you definitely can get public recognition for your work.
One can even get an award like Padmashree, inspite of being in government service, by doing dedicated work.
Difficulties are temporary. If you are sincere & committed, you will find a solution & results automatically follow.

Dr. P M : Your work in the 'Ananadvan' ashram of Baba Amte is huge. Tell us something about your association with him.

Dr. T L : Baba Amte was a living legend, or a god like figure.
In 1999, I was going through a very low emotional phase. I was frustrated with the constant transferes in service & had almost decided to quit. This was when I happen to have visited 'Anandvan'. When I experienced Baba's work, I realized that as compared to this man, my difficulties were like a speck of dirt. He was a colossus. I decided to forget my frustrations & to continue in service.
We then started conducting regular eye camps for people from that region. In the first year, we did 184 eye surgeries. In the next year we did 213 & I am happy to say that in the last year's camp, we screened 11,000 patients & did 1650 surgeries. Initially we would get patients from the leprosy affected families, because others had some inhibition in there mind. Later as the word spread, we started getting patients from all sections of the society. Baba felt this is the best way to change the mindset of the common man, towards leprosy, where all kinds of people mix & share their experiences. Baba, lovingly named this as 'Lahanu babachi andhayatra'.
I must add here the meticulous planning & execution done by Dr.Ragini Parekh, which has ensured zero cases of endophthalmitis.
It was Baba's inspiration because of which my name changed from Tatya to Tatyarao.

Dr. P M : Coming to one more important aspect in your life. The Dr. Sandeep Wagh oration award was conferred on you by POS. We would like to know something about this & also your association with POS.

Dr. T L : POS, I believe is a very dynamic association. I have had high regards for your association.

I am always grateful to Dr.Shreekant Kelkar, who was the first person from our fraternity to have invited me to perform live surgical demonstration in his annual conference. I was perhaps one the first person, from government department to perform live surgery. As it happened, our minister of health, Mr. Shetty, was present during my live surgery. This obviously changed the state governments view about me (seeing the quality of surgery). My work got recognition & that helped us a lot in getting aid.
I had received Dr.Bhalchandra award earlier, but this was my first big & significant award. Getting any award is a huge inspiration & this award was truly a big turning point in my career.
Hence I am always indebted to Dr. Kelkar & POS for giving me a platform to show my talent.

Dr. P M : What has been your experiences of your work in the Bombay Ophthalmologist Association(BOA) & Maharashtra Ophthalmologist Association(MOS)?What are your plans for MOS during your tenure?

Dr. T L : In my initial days in Mumbai, I was not at all active in any of these associations. A phase came in BOA, where we had very poor attendance for our yearly conference. This was a sign of decline for such an illustrious association. This was when people suggested me to take over the Hon. President's post of BOA. I did not know much about running these organizations. But our team decided to take up this challenge. We upgraded the scientific content, invited a lot of speakers from the country & the result was in the first year of my tenure we had 700 delegates registered for our conference. This number went up to 1000 & then last year we had 1500 delegates who attended this conference.
We have specifically encouraged people from government service to come & attend these conferences, by arranging for their stay & providing other allowances. We also have consciously invited doctors from remote areas to showcase there talent (I even went to the extent of making presentations for some of them). We also have included a minimum of 3 surgeons from non-metro, during the live surgery demos. All this has resulted in increase in attendance.
As far as MOS is concerned, I would request all ophthalmologists to become members of this association. Secondly we would encourage formation of associations at smaller towns (eg : Yavatmal, Buldhana,etc). Thirdly, there is a great demand to learn phaco, so we are planning to conduct wet lab sessions in smaller towns in the state. For this I must thank, Alcon & Appasamy, who have promised us help. We already have conducted one such workshop at Ambejogai.
I also would like all MOS members to take membership of AIOS. We also want more & more eligible people to hold responsible post in AIOS. In this regard, we have proposed name of Dr. B. K. Nayak for secretary's post & name of Dr. Yogesh Shah for the treasurer's post.

Dr. P M : It was a rare occasion that 2 individuals from the same city were conferred 'Padmashree' in the same year. (you & Dr.Keki Mehta). There is big difference in both individuals (your background) I want to ask you about your experience of getting this award.

Dr. T L : In 2002, I was requested to send my bio data for consideration of this award. When the awards were declared, I was in Hemalkasa with Dr. Prakash Amte. He was given the award, but my name was not there in the list. I was a bit disappointed, but happy that Dr. Amte had received the award.

In 2008, Dr.Ragini Parekh, sent my biodata again, because she felt that I deserved to get the prize.
On the 21st January 2008, I was in Mhada (Kurduwadi) for hospital inspection, when I got a phone call from the Home Secretary, Government of India, asking me if I would accept this honour (I consented to this). Since I was advised not to declare it to anybody till 6 in the evening, I kept this news to myself. Later in the day, the news was declared & I was inundated with phone calls. My family & colleagues were indeed very happy that I had received the prestigious honour.
You will be surprised to know that, I was invited for more than 40 public felicitation functions. This was a unique honour. There would be a queue of people to meet me at these events.
This was when I realized the love & affection people had for me and also the value of my work done over so many years. (I even got a compliment from Mr. R R Patil that this honour was accepted & applauded by all sections of the society)

Dr. P M : We wish you get many more such honours in future.

Dr. T L : Thank you!

Dr. P M : Coming back to an important topic, Post-Graduate education. What is the state of affairs & what are your suggestions?

Dr. T L : I am not very happy about PG education at present.
There are 2 aspects. The quality of teachers & the students. Amongst all the medical colleges in the state, many of the teachers do not operate or perform very limited surgeries. Many do not have the desire to learn new techniques. This is not a good thing for the students, because they will not get to see many of the routine procedures required to be done by an ophthalmologist.
On the other hand, the students also have no inclination to learn the art & science of ophthalmology. Most are happy to learn only Phaco. Students prefer departments where one gets more number of surgeries('cutting experience'). But everybody should realize that they are operating on human eyes & hence everybody should develop the right attitude to learn the basic & advanced techniques with responsibility.
If we do not train our doctors properly, how can they manage patients properly?

Dr. P M : What advice will you give to young ophthalmologists?

Dr. T L : The most important thing is to train oneself well in the surgical & clinical skills before starting practise. It is equally important to practise ethically.
Considering the cost of starting practise, maybe it is advisable to start with 2 or 3 colleagues. This way the financial burden can be shared & they can give affordable service to the society.

Dr. P M : What is your ultimate dream?

Dr. T L : I wish to die when performing surgery.

Dr. P M : Dr.Lahane, it was indeed a special learning experience talking to you. I wish to thank you (on behalf of POS) for gracing our programmes & encouraging & appreciating our work. We wish you all the very best for your future. Thank you.

RELEVANCE OF CLINICAL TRIALS IN GLAUCOMA PRACTICE

Dr. Shraddha Satav

MBBS, DNB, FLVPEI (Glaucoma)

Glaucoma is a complex, progressive optic neuropathy. It is the leading cause of irreversible and preventable blindness, affecting an estimated 70 million people worldwide. Numerous clinical trials have been published and their findings at touted at every meeting but their relevance to the general ophthalmologist is never explained.

We must remember that clinical trials have been conducted on a group of patients with specific inclusion and exclusion criteria. Individualisation and comorbidity needs to be considered before extrapolating these outcomes to our patients.

Overview of Clinical Trials :

Trial	N	Dx	Randomization	Follow-up
OHTS[1] (NEI)	1636 pts	OHT	Medical treatment versus observation	5 years
EMGT[2] (NEI)	255 pts	OAG	Treatment (ALT + betaxolol) versus observation	4–9 years
CNTGS[3] (GRF)	140 eyes	NTG	Medical treatment and/or surgery versus observation	7 years
CIGTS[4] (NEI)	607 pts	OAG	Medical treatment versus surgery	5 years
AGIS[5] (NEI)	738 eyes	OAG	ALT versus surgery	8 years

OHTS – Ocular Hypertension Treatment Study

Despite the lack of convincing evidence, approximately 1.5 million people in the U.S. with elevated eye pressure and no glaucoma damage were being treated with medications that lower this pressure. There was a need for a well-controlled clinical trial to determine whether medical reduction of elevated intraocular pressure could delay or prevent the onset of glaucoma. The resulting data would enable clinicians and patients to make rational choices and health care planners ensure that medical resources were being allocated in a safe and cost-effective manner.

The primary goal of the Ocular Hypertension Treatment Study was to determine whether reducing elevated eye pressure delayed or prevented the onset of glaucoma and subsequent vision loss in people at risk of developing the disease. Patient recruitment took place between February 28, 1994 and October 31, 1996. 1636 patients aged 40 – 80 years with IOP between 24 to 32 mm Hg in one eye and 21 to 32 mmHg in the other eye with no other pathology were included. These were randomized into 2 groups -Medical treatment versus observation.

Target IOP was 20% reduction to 24 mmHg. The primary outcome was development of POAG, defined as continued visual field abnormality or reproducible optic disc deterioration.

Results – In the treated group, mean IOP reduction was 22.5% and in the controls, the decrease was 4.0%. 4.4% in treated group vs 9.5% in observation group (P < 0.001) progressed to POAG – a **50% reduction in risk.**

Conclusion - Medical therapy effective in delaying/preventing onset of POAG in subjects with elevated IOP.

Baseline factors that predict the onset of POAG - older age, larger C:D ratio, greater PSD, higher IOP

Strengths of the study - large sample, strictly applied protocols, masking, true incidence cases

Weaknesses - limited IOP range, sample was not population based, criteria for conversion to POAG adjusted during study, some patients with normal white on white perimetry were later reported to have defects on SWAP, casting a doubt on their normal status.

Clinical implications -

All patients with elevated IOP need not be treated. A large proportion of untreated patients (>90%) did not develop POAG.

Glaucoma medications have their own side effects, use only when needed, not as a preventive medicine. Consider age, medical status, life expectancy and likely treatment benefit.

20% lowering of IOP is not the blanket target for OHT, treat every case as individual.

Consider measuring corneal thickness in all patients with OHT or glaucoma.

Goldman tonometers is calibrated for IOP of 550 microns, use correction for thicker or thinner corneas.

EMGT - Early Manifest Glaucoma Treatment Study

Prior to this study, the natural history of glaucoma was not well defined. Researchers did not know how rapidly, if at all, early stage glaucoma would progress if it were not treated initially. Because most eye care professionals immediately treat newly-diagnosed glaucoma by reducing intraocular pressure, the natural progression of the disease (in its untreated state) was not clear. Researchers were also unclear as to how effective treatment was for early stage glaucoma, because they did not know how rapidly the disease would progress without treatment.

This raised a key question: What price, in terms of side effects, inconvenience, and cost, can be considered acceptable when treatment effects are uncertain? To begin answering this question, a randomized study was designed with a control arm in which participants were followed without treatment as long as progression did not occur, thus not exposing study participants to unacceptable risks.

The Early Manifest Glaucoma Trial is the first large, controlled, randomized clinical trial to evaluate the effects of treatment versus no treatment on early stage glaucoma. More specifically, the study compared glaucoma progression in treated (lowering intraocular pressure) versus control patients. The study also determined how much treatment reduced eye pressure, and helped researchers chart the natural history of the disease.

Patient screening began in October 1992 and ended in April 1997. Study participants came from the Swedish cities of Malmö and Helsingborg. The study followed 255 patients, of which 66 percent were women. All patients were between 50-80 years of age, inclusive (average age : 68), and all had early stage glaucoma (open angle glaucoma or normal tension glaucoma) in at least one eye. One group (129 patients) was treated immediately with medicines and laser to lower eye pressure. A second, control group had 126 patients who were left untreated. Both groups were followed carefully and monitored every three months for early signs of advancing disease, using indicators that are extremely sensitive for

detecting glaucoma progression. Any patient in the control group whose glaucoma progressed was immediately offered treatment.

Early, previously untreated open angle glaucoma was considered and the study compared treatment (ALT + medical treatment) versus observation. Secondary aims were to assess factors related to glaucoma progression and to study the natural history of the disease.

No Target IOP. Avg IOP drop 25% (range 0% –0 29%)

Results - Disease progression 45% in treated vs 62% in untreated (P=0.007) A 25% decrease in IOP reduced the risk of progression by 50%. **Each 1-mmHg reduction in IOP reduced risk of progression by 10%**

The study also demonstrates very large inter-patient variations in disease progression rates. It is important to remember that Betaxolol was the medical treatment used, hence extrapolation to a clinical scenario where this may not be the drug of choice is doubtful.

Progression was more common in :

Patients with higher IOP, Older patients, Patients with more advanced baseline damage, Exfoliation glaucoma, Treatment was associated with progression of nuclear lens opacities

Clinical implications -

Newly diagnosed primary POAG and NTG should be treated aggressively

Some patients do not progress. After median follow up of 8 years, 24% untreated and 44% treated patients did not progress.

In EMGT, the average sustained IOP reduction was 25%

IOP reductions of 30% or 35% will preserve the visual function of many patients

The goal of therapy should be to achieve pressures as low as is safely possible

Since approximately half of EMGT participants had a mean IOP of 21 mm Hg at baseline but had visual field loss characteristic of glaucoma, glaucoma screening that relies on IOP alone may miss half of all patients with glaucoma

Structural change may not catch progression earlier. Visual fields almost always demonstrated progression before disc photographs.

CNTGS – Collaborative Normal Tension Glaucoma Study

This study came about because glaucoma specialists in the 80's were in disagreement as to whether normal tension glaucoma (NTG) existed at all. Some believed that NTG was true glaucoma, while others thought that NTG was an optic neuropathy that looked like glaucoma. In any event clinicians had had less success in controlling progression of glaucoma in patients with NTG.

145 eyes with NTG - (Medical treatment ± surgery) versus observation. The primary outcome measure was disease progression.

Target IOP - 30% IOP reduction. (Beta blockers and adrenergic drugs were excluded because of the potential crossover effects)

Results - 80% survival (no progression) in treated group versus 60% in control arm at 3 years. VF progression 18% in treated versus 30% in untreated

Cataract amongst treated eyes was 38% and 14% in the controls group.

Strengths - long follow up, masking of observers

Weaknesses - visual field criteria were changed during the course of the study, CCT values were not taken, IOP values upto 24 mm Hg, higher than normally considered for NTG, Intent to treat analysis affected by cataract formation

Clinical implications -

IOP is a factor in deterioration of vision from NTG

Since 65% of patients did not progress

during the first 3 years, a factor other than IOP influences progression in individual patients

In NTG patients, lowering IOP by at least 30% led to a lower rate of visual field loss than is seen in NTG patients who did not receive treatment

CIGTS – Collaborative Initial Glaucoma Treatment Study

Recent studies have challenged the conventional wisdom of treating all newly diagnosed open-angle glaucoma (OAG) with eyedrops; rather, these studies suggest that more effective control of glaucomatous damage can be obtained by immediate filtration surgery. In addition, increased attention to the impact of therapy on health-related quality of life has added another consideration in deciding upon appropriate treatment of such patients.

The Collaborative Initial Glaucoma Treatment Study (CIGTS), a randomized, controlled clinical trial was conducted to determine whether patients with newly diagnosed OAG are best managed by the conventional approach of topical pharmacologic agents or by immediate filtration surgery. Eligible patients were randomized to receive either a stepped medication treatment regimen or filtration surgery to control their OAG. Sample size requirements indicated that 300 patients were needed for each treatment approach; a total of 607 patients were ultimately recruited for the CIGTS.

607 patients with newly diagnosed OAG - Medical treatment versus surgery (trab with or without 5FU) Primary outcome variables were VF loss and Quality of life. Secondary outcome variables were visual acuity, cataract formation and IOP.

Target IOP - Low target pressure set by formula

Conclusion- With aggressive therapy aimed at IOP-lowering, VF loss in general is minimal

Both medical and surgical treatment reduced IOP by an average of more than 30% in patients with newly-diagnosed open-angle Glaucoma

Few patients in either treatment group progressed

Both medical treatment and surgery effectively reduced IOP (by 38% and 46%, respectively) and the risk of progression over 5 years (to 10.7% and 13.5%, respectively)

Target pressures used were aggressive. A target IOP reduction of $\geq$ 25% from baseline was required for a patient with baseline IOP of 25 mm Hg and no field damage; if minor field damage (scored as 5) was present, the target was a $\geq$ 30% reduction from baseline IOP

Quality of life was initially better in the medical treatment group

Strengths - individualised target IOP approach, Newly diagnosed patients, Quality of life prospectively addressed.

Weaknesses - Inclusion criteria may have allowed inclusion of Ocular Hypertensives who have little risk of progression, Only preliminary results reported. Follow up may not have been long enough to show differences.

Clinical implications -

Surgical treatment reduces IOP more but also causes more cataracts. There is no significant difference in progression whether you bring down IOP by medication or surgery as long as target IOP is attained. Always consider quality of life of your patient.

AGIS – Advanced Glaucoma Intervention Study

In advanced glaucoma, medication alone no longer reduces intraocular pressure adequately, and the eye has field defects. Before 1980, some type of filtering surgery, such as trabeculectomy, was the usual method of intervention. Since then, laser trabeculoplasty has become a popular alternative. Sometimes the first intervention chosen succeeds in

controlling pressure for many years; at other times, the success lasts only a few weeks or months. Because success is limited, some patients, over time, need to undergo a sequence of surgical interventions. Little is known about which sequence gives the best long-range outcome.

The Advanced Glaucoma Intervention Study (AGIS) was designed to provide a comprehensive assessment of the long-range outcomes of medical and surgical management in advanced glaucoma. The study uses visual function status to compare two intervention sequences in managing the disease.

738 eyes with uncontrolled glaucoma - ALT versus surgery

Target IOP - < 18 mmHg

Results -

100% of visits <18 mm Hg: no change in visual field

<50% visits <18 mm Hg : worsening of VF by 0.63 units

Conclusion - Consistently low IOP associated with reduced progression of VF defect

The association between low IOP and reduced progression of visual field defects persisted throughout follow-up

IOP levels often considered adequate (from 14 to 17.5 mm Hg) were associated with greater visual field loss than low IOP levels (under 14 mm Hg), although the difference was not statistically significant

A proportion of eyes in the < 14 mm Hg group experienced visual field loss, even though on average the change in VF defect score was close to 0

Strengths - large sample size, long follow up

Weaknesses - only one visual field was considered as baseline, despite the title, very advanced glaucoma was excluded while some cases of early glaucoma were included, Visual field spread was very small, statistical significance was achieved due to large numbers

Clinical implications :

- IOP reduction reduces visual field progression.
- Fluctuation plays a significant role
- Consider cataract formation as a risk factor of glaucoma surgery

One of the strongest messages to emerge from all these trials is that glaucoma differs in its aggressiveness from one patient to another. Initial assessment allows us to quantify damage and risk at that point in time. Setting appropriate baselines of disc structure (careful drawings, photographs, imaging devices) and of function (SAP, FDT, SWAP, perhaps even multifocal multichannel visual evoked potentials) allows us to use time intelligently.

IOP reduction does benefit at risk patients. But greater IOP reduction is not inevitably better for all. At each visit we need to ask ourselves - has the situation changed? Is an at-risk eye that was being monitored now in need of treatment? Is a treated eye insufficiently protected? Must therapy be accelerated, aiming at a lower target IOP? Only through this kind of careful monitoring can we identify those of our patients who need help, or more help, and only in this way can we separate out patients who do not need treatment (with all its associated costs, inconveniences, and potential side effects) or who need only limited treatment.

The aim of treatment need not be no progression at all but reducing the rate of progression so that the Quality of vision is maintained during the patient's lifetime.

It is often said, 'What the mind does not know, the eyes do not see'. Unless we are aware of what literature says and what experience already exists, we cannot give our patients the best treatment there is.

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OCT IN GLAUCOMA PRACTICE PEARLS & PITFALLS

Dr. Medha Prabhudesai
MBBS, DOMS, Glaucoma Fellow, SN

To interpret OCT in glaucoma, careful evaluation of ONH and RNFL is necessary. OCT analysis should correspond to the ONH and RNFL changes. If the correlation does not exist and OCT analysis is abnormal then other causes should be ruled out.

Normal RNFL is seen within two disc diameters of ONH. It has a uniform pattern of tightly packed bundles radiating from the disc. It shows bright striations at superior & inferior poles. Papillomacular bundle has less bright striations. Texture of striations is continuous over retinal blood vessels

Various types of RNFL defects are seen with the help of red free photography.

- Spindle shaped defects which are narrower than retinal vessels & which do not reach ONH margin are physiological
- Localized defects which are wider than retinal vessels & which reach ONH margin are pathological
- Wedge defects are seen at superotemporal or inferotemporal margin broaden towards periphery are pathognomonic
- Diffuse loss is seen as absence of striations.

Here is a brief summation how ONH findings, RNFL and OCT findings correspond with each other.

RNFL	ONH	OCT
Slit defects	Normal Splinter hemmorrhage Localized thin NRR	Normal Sectoral thinning
Multiple slit defects	Focal Notch Thin NRR	Single or localized quadrant loss
Wedge defects Infero-temporal Supero-temporal	Focal Notch V enlargement Bayoneting Beta atrophy	Sectoral loss Quadrantic loss 2 quadrant loss
Diffuse loss	Thin NRR High C/D Bayoneting Laminar dot sign Beta atrophy	2 or more quadrant loss Temp. PMB preservation

The conditions where OCT analysis should be interpreted carefully include myopia, other neuropathies, retinal diseases especially BRVO, hemispherical venous occlusion or tributary BRVO.

In moderate to high myopia, peripapillary atrophy gives rise to thinning of RNFL either all around or in the area of atrophy. This mimicks thinning of RNFL in glaucoma.

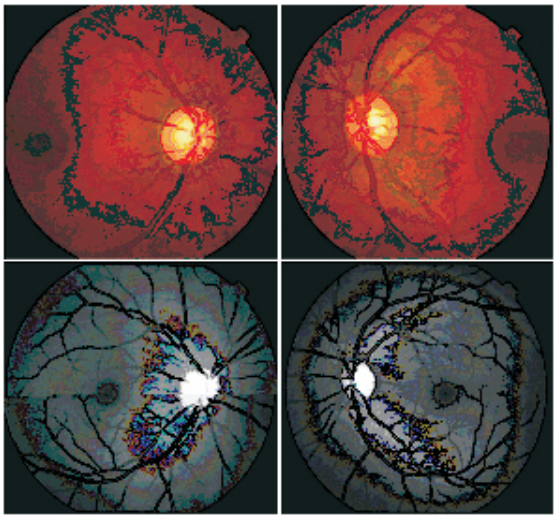
BRVO itself gives rise to arcuate defect in Perimetry along with corresponding defect in OCT and can be easily diagnosed as glaucoma,

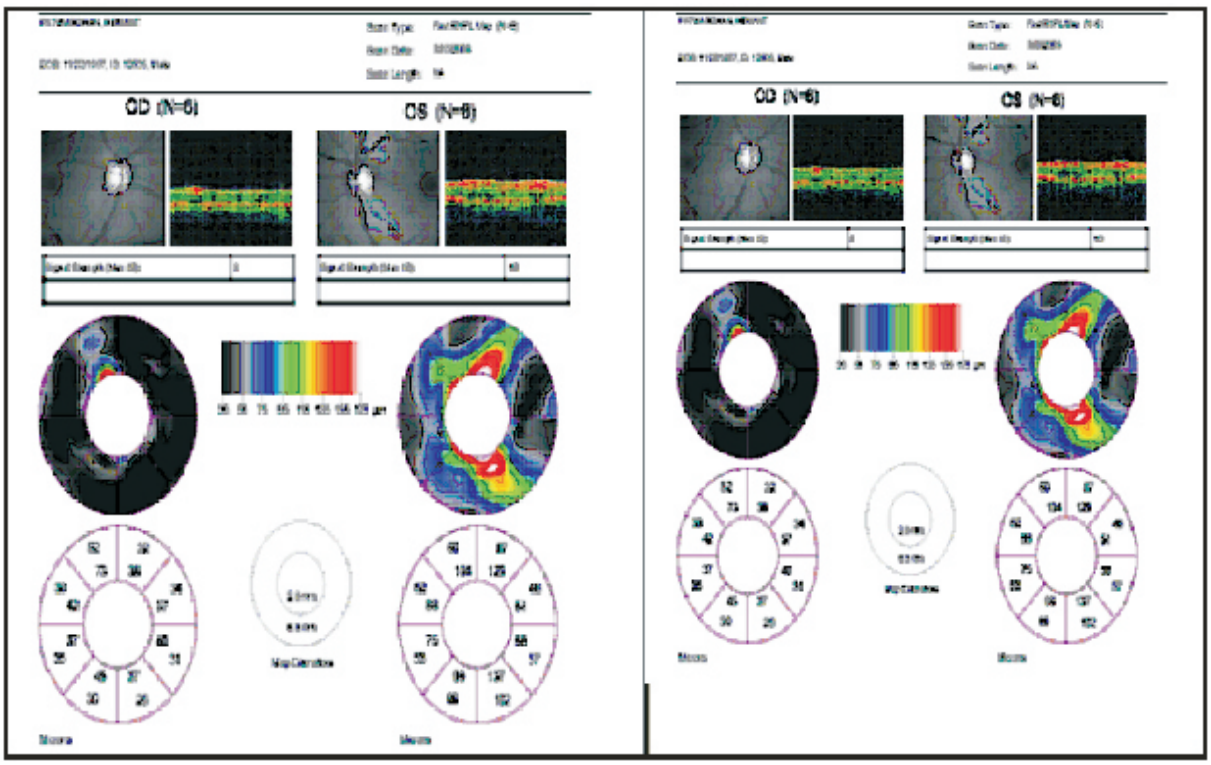
LASER done for BRVO or sectoral NV for any other reason gives rise to similar defects. Patients who have diabetes mellitus and glaucoma , specific attention should be given to diabetic naculopathy. If macular scans show thickening then RNFL measurements will appear normal even if patient has glaucoma, due to macular edema .

Here are some examples of how ONH and RNFL along with OCT confirm or exclude glaucoma.

Case 1

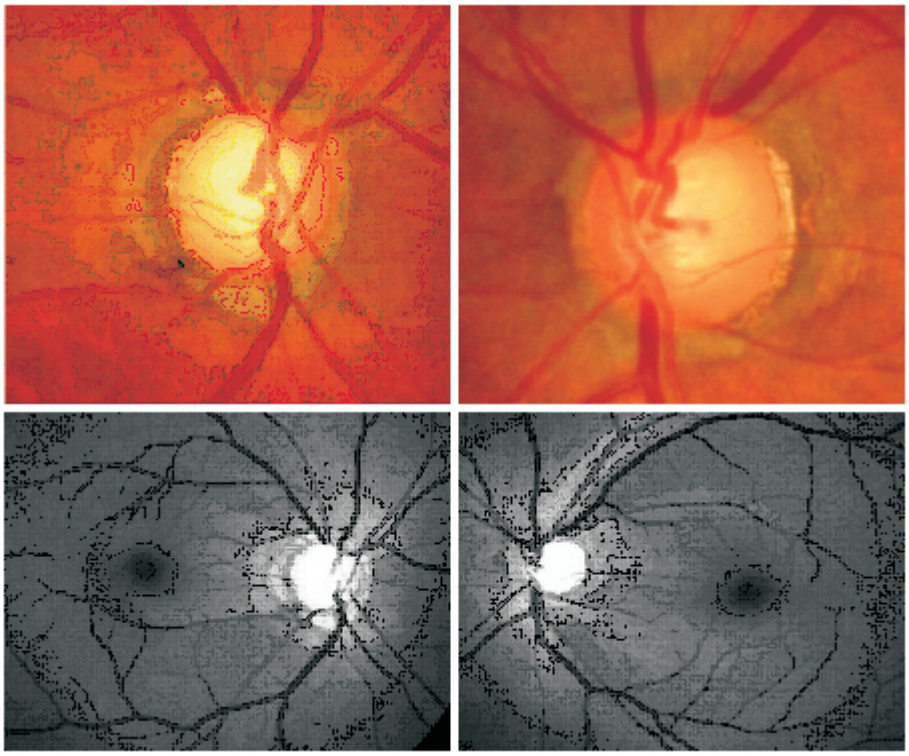
In right eye vertical CD is more than horizontal CD with thin NRR and bayoneting. IN red free photograph RNFL shows multiple slit defects in superotemporal & temporal quadrant. These correspond very well with the OCT findings

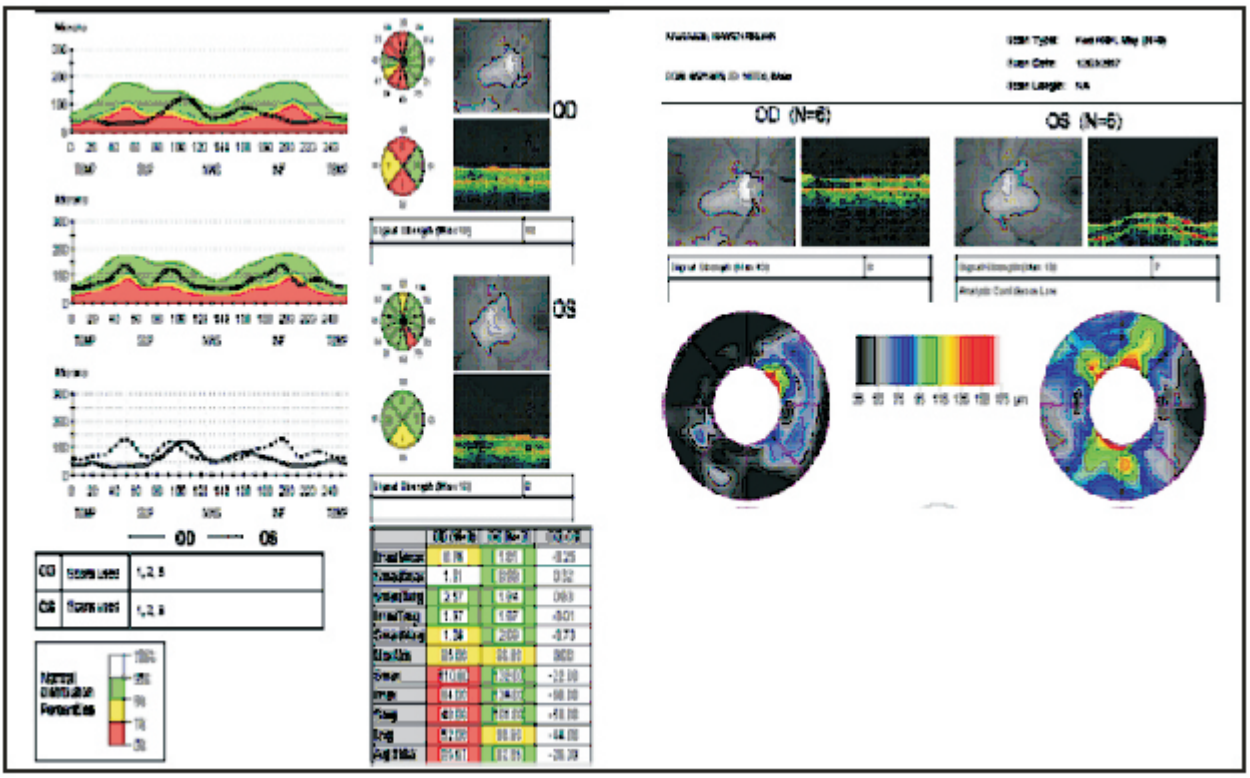




Case 2

Right eye shows thinning of NRR in all quadrants. There is evidence of irregular beta zone atrophy in both eyes more in the right eye as compared to left eye. Red free photograph shows bilateral wedge defects at superior and inferior poles. OCT also shows corresponding defects in right eye. Left eye OCT does not show the superior defect. Subsequent periodic OCT may pick up this defect.

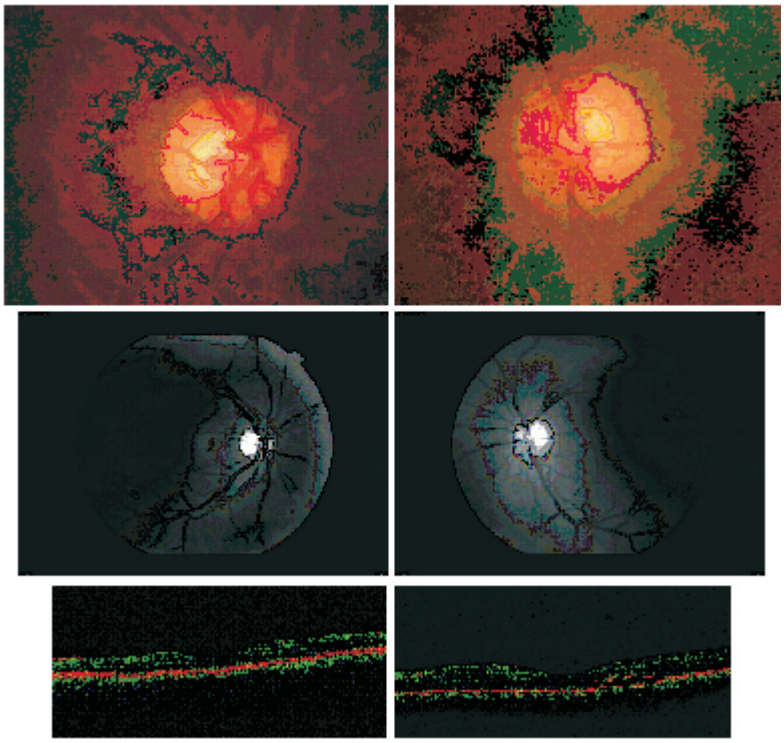


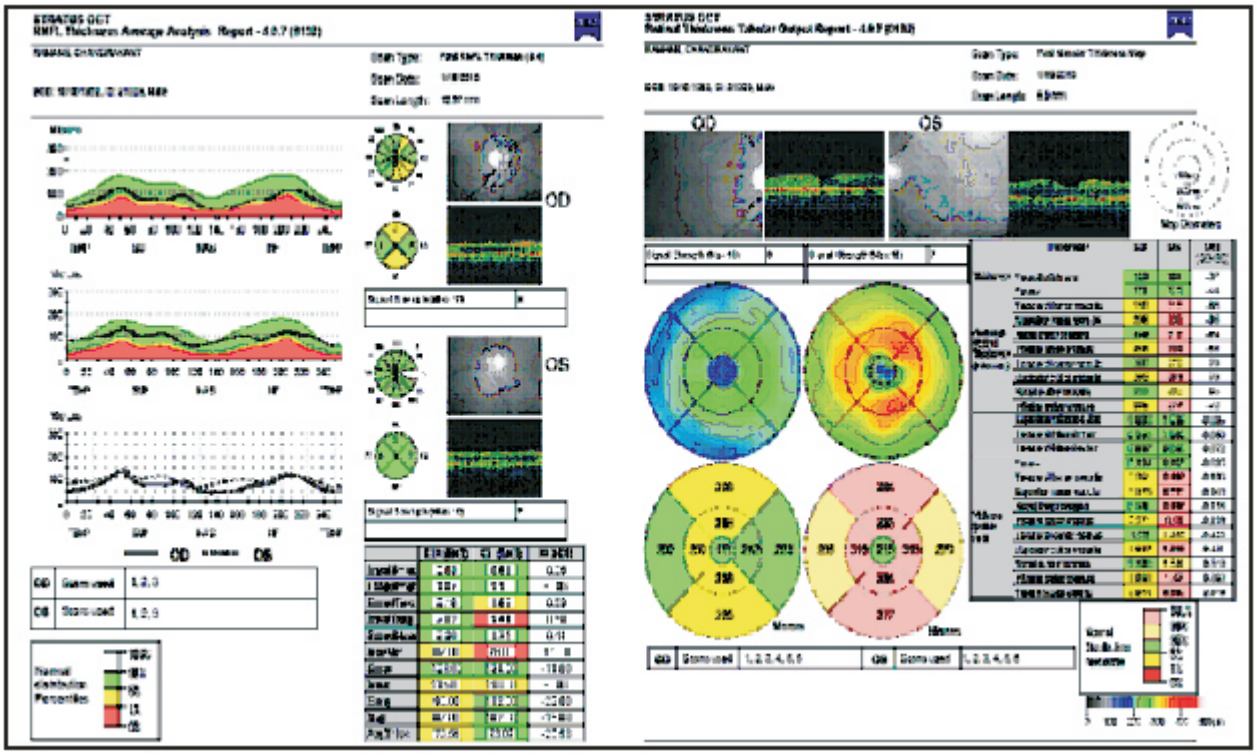


Case 3

This patient has thinning of RNFL in superior & inferior quadrant in OCT in right eye which is consistent with his ONH findings. Left eye OCT shows normal RNFL. Left eye has nuclear sclerosis with hazy view of macula and ONH. Same eye in radial OCT scan shows macular thickening and intraretinal hard exudates. Also there is increase in the thickness of macula in OCT macular map.

In this case labeling left eye as non glaucomatous will be erroneous. This needs repeat evaluation of RNFL after treatment of diabetic maculopathy, when the macular map shows normal macular thickness.

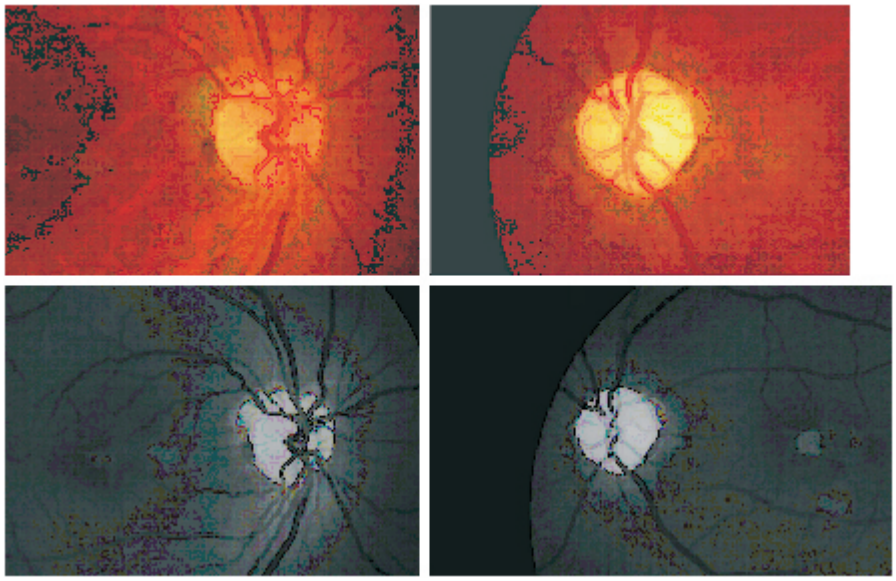


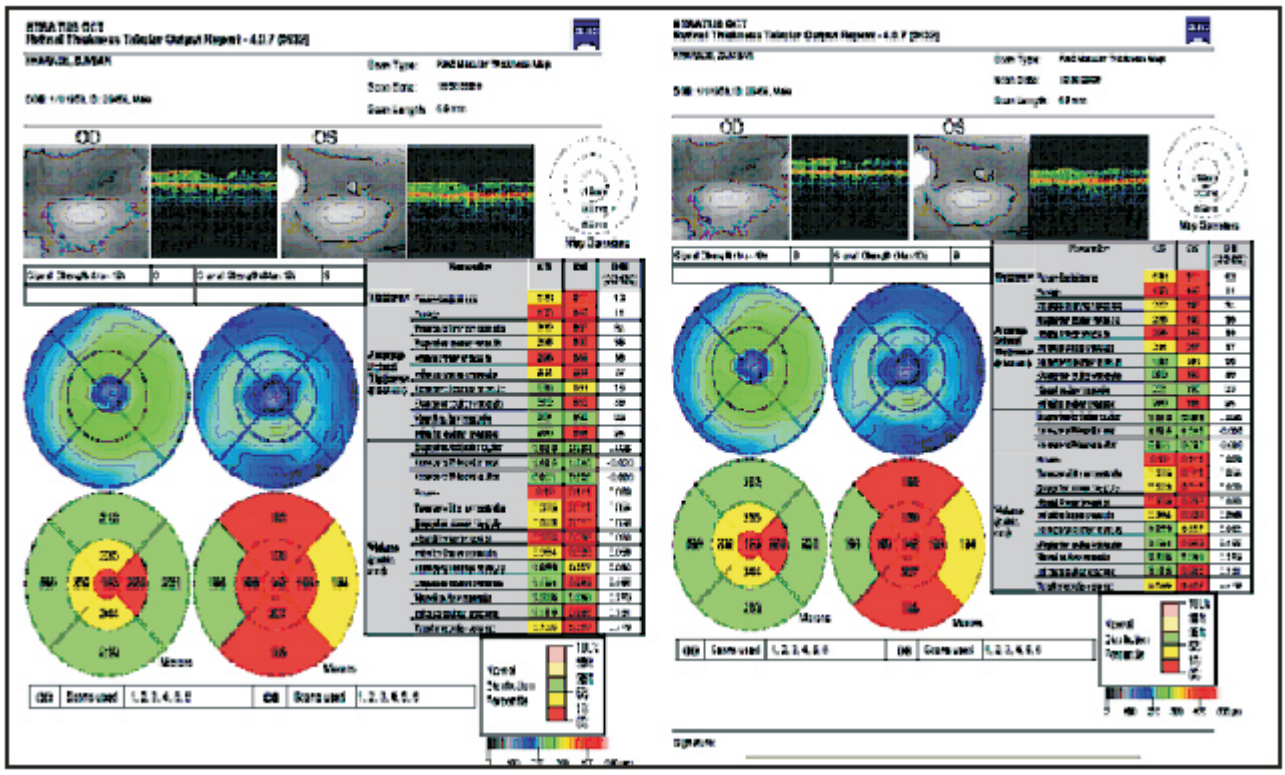


Case 4

This patient has normal ONH & RNFL in the right eye. Left eye RNFL and macular - OCT analysis is abnormal with RNFL thinning in superior, temporal and inferior quadrants. ONH fundus photograph shows pallor more than cupping and no signs of glaucomatous optic neuropathy.

This patient needs further investigations to rule out neurological, nutritional and systemic disorders.





These examples would emphasize the need for good correlation of ONH & RNFL findings seen clinically with RNFL thickness and macular thickness noted on OCT, we should avoid temptation of looking at the OCT findings in isolation.

SUMMARY

In the last few years various imaging technologies have evolved for diagnosis of glaucoma. Among them OCT gives direct measurement of RNFL along with macular thickness. For diagnosis of glaucoma correlation between structural changes of ONH and analysis of imaging techniques is important. This can be achieved to a good extent by OCT evaluation

Specific ONH findings can be seen in Glaucomatous optic neuropathy. OCT along with clinical evaluation of ONH and RNFL has maximum specificity and sensitivity for diagnosis for glaucoma. The sectoral or quadrantic thinning of RNFL on OCT should match thinning of NRR, notching, bayoneting of vessels or RNFL defects.

However RNFL thickness as measured by OCT should be interpreted only if there is no macular pathology. Macular thinning or thickening due to macular disorders would give rise to corresponding thinning or thickening of RNFL on OCT, leading to erroneous interpretation.

To summarise OCT is an ever-evolving powerful tool to study glaucomatous changes, still clinical correlation is more important and we should avoid temptation of looking at the OCT findings in isolation.

GLAUCOMA PROGRESSION

Dr. Rupali Nerlikar
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What is progression?

Progressive loss of ganglion cells in patients diagnosed to have or suspected to have glaucoma resulting in progressive loss of visual function

Why does progression occur?

Neuronal dysfunction (manifests as reduction in sensitivity/increased variability) gradually proceeding to neuronal loss as a part of secondary degeneration or due to continued presence of initiating noxious stimulus (raised IOP/impaired vaso regulation)

Structural loss often precedes functional loss. Rate of progression of neuronal loss varies with extent of damage already present.

SYMPTOMS :

- Usually asymptomatic
- Patients may complain of decreased vision/impaired depth perception/difficulty in climbing stairs/ reduced night vision

DETECTING PROGRESSION :

1. Clinically
2. On ONH or NFL imaging (structural loss)
3. On Perimetry (functional loss)

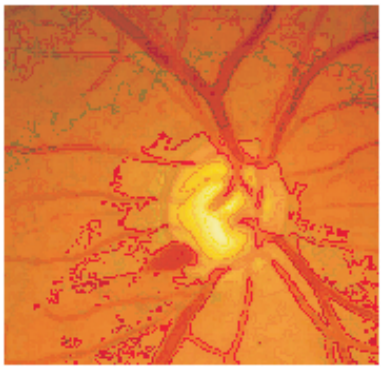
Ability of structural and functional tests to detect progression is limited by the normal variability within the tests which tends to be greater in glaucoma. Thus to reliably detect change it has to exceed any change that may be accounted by normal variability.

Clinically

Progression can be seen as

- Increase in extent of neuro retinal rim loss
- Widening of NFL defects/appearance of new NFL defects (seen with red free)
- Widening of (width/circumference of)$zone
- Appearance of a disc hemorrhage

These changes are not easy to determine especially on undilated disc evaluation. The best way to look for them is on fundus photos (disc centric)-color as well as red free.



Splinter haemmorrhage

ONH or NFL imaging

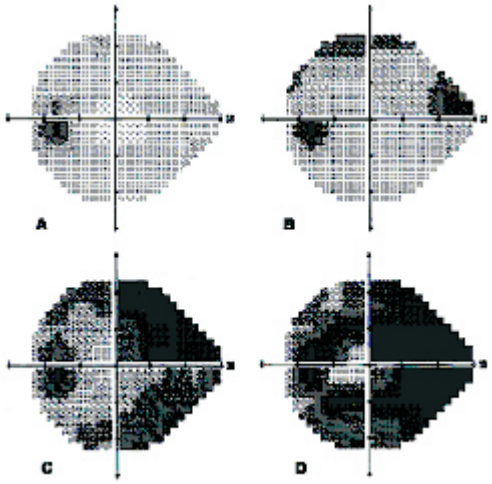
At present the HRT 3 has 2 algorithms (trend analysis and topographic change analysis) and gives a glaucoma probability score.

Not much data is available about the new GDx VCC progression analysis software and cirrus OCT does not have statistically backed progression analysis software

Small studies have shown poor correlation between the structural and functional tests to determine progression and none of the structural tests are reliably able to detect subtle changes in structure.

PERIMETRY PROGRESSION :

Visual field testing is benchmark for testing progression



Example of VF progression

Detecting perimetric progression

Observing change in VF loss over time can give an idea as to how the GC loss is progressing (linear/episodic/stable)

Progression can be a deepening of defect or widening of defect or both. Progression on perimetry has to be differentiated from noise and fatigue and LTF

Stages of progression

- Latent-not clinically detectable on visual fields
- Threshold-fields show increased variability but not consistent repeatable defects
- Manifest-the critical threshold has been crossed and consistent field defects present.

Progression is often asymptomatic and periodic evaluation is important in order to detect it.

Detection of progression is affected by -

- Rate of progression
- Inherent variability
- Extent of existent disease
- Interval of follow up

In an advanced disease with rapid progression and high variability, frequent testing is needed to pick up progression reliably

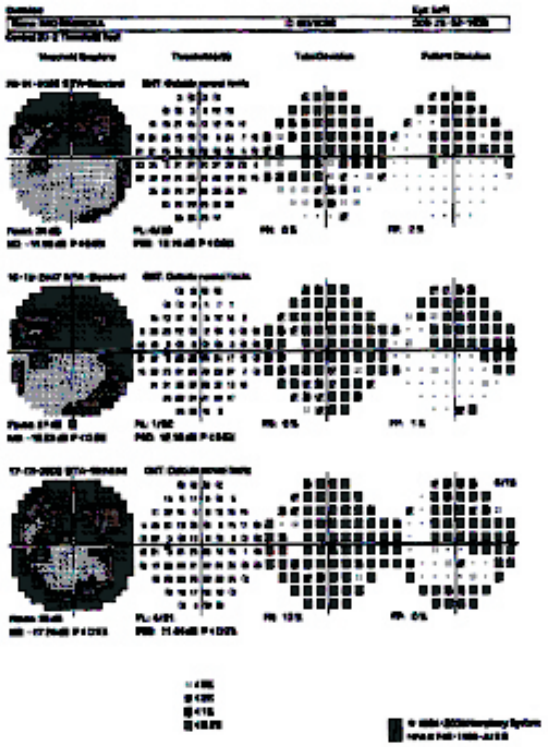
Detection of progression -

1. Subjective clinical judgment :

Serial tests are visually assessed-easy

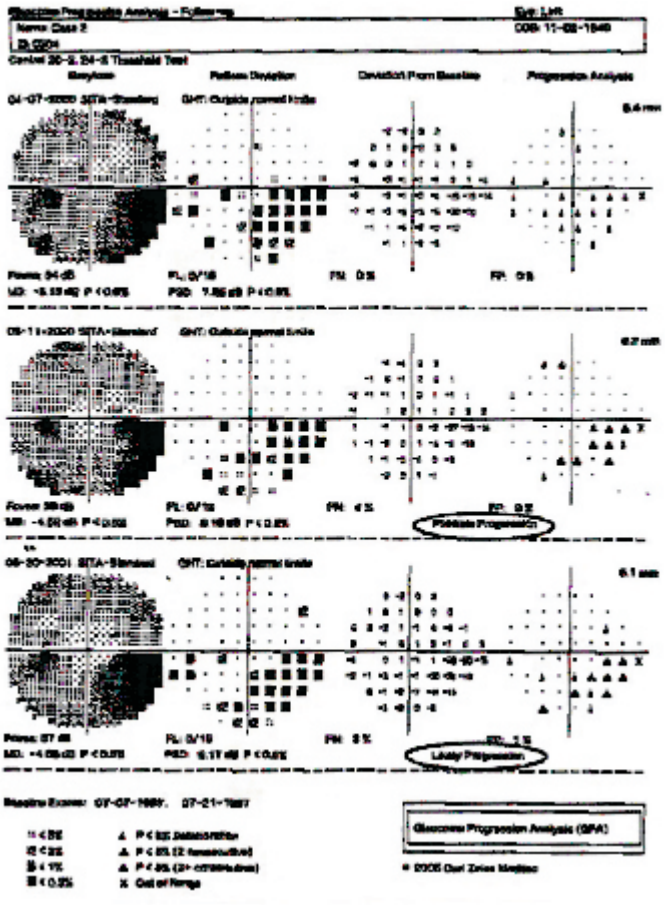
but not standardized

E.g. overview printout



2. Event analysis (GPA) :

This software available with the Humphrey perimeter allows detection of progression. Here each point tested in a visual field is compared to its corresponding point on a baseline test (average of 2 reliable tests). Any change is taken to be progression if the change exceeds that accounted for by variability (compared to a data base of age matched stable glaucoma patients). Thus focal as well as generalized progression can be picked up within as few as 3 follow up tests. The change is depicted pictorially in the printout with triangles representing likelihood of progression and a comment- eg. likely/possible progression

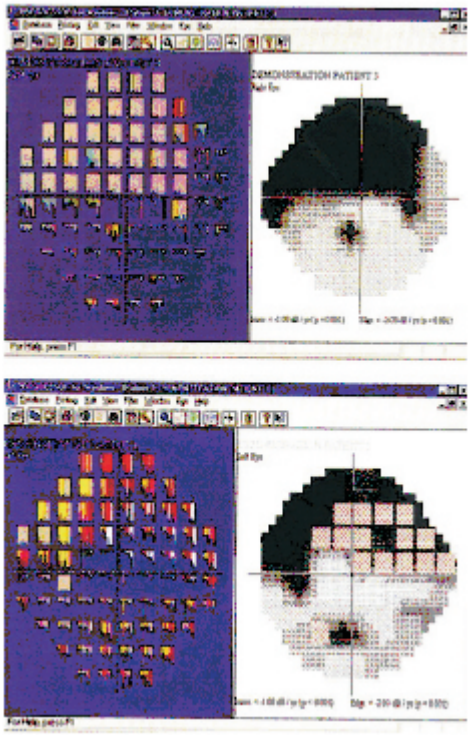


Criteria used to diagnose progression :worsening at 3 or more locations on 3 consecutive tests.

Limitation : acceptable limit of variability is a statistical entity and some individuals may have high variability and be falsely labeled as progressing. Also some with low variability and actual progression may be missed.

3. Trend analysis (statpac 2, progressor) :

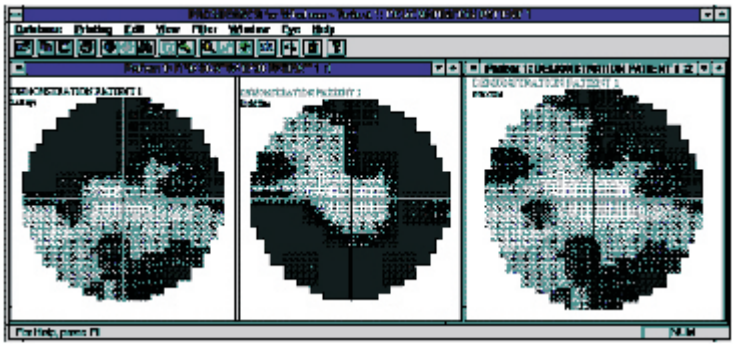
Pattern of change at each point over serial fields is determined (linear regression). It is not dependant on predetermined limits of variability in a standard population. However it requires a greater number of tests (7-8) to obtain a trend which may show progressively reduced sensitivity suggesting progression.



The Progressor software from Moorfields gives a point wise linear regression analysis and represents it in a color coded manner giving a fairly good idea about stability or progression. The slope at each point is assessed-positive/negative or flat (i.e. no change)-the color code then gives the probability of this occurring in a stable population.

Limitation : It is not loaded on any VF machine. The VF test data has to be transferred to the desktop and run through the program which can be cumbersome. Also a large number of tests are needed to determine a trend.

Advantage: It gives an idea of integrated binocular field and hence the extent of functional vision



EVALUATING CAUSE OF PROGRESSION :

- Poor compliance
- Diurnal fluctuation in IOP(monitor diurnal)
- Nocturnal dip in BP resulting in reduced perfusion pressure(diurnal BP)
- Systemic problem i.e. Cerebral/coronary ischemic episode

MANAGING PROGRESSION :

The aim of monitoring in glaucoma is detection of Progression/lack thereof, as treatment is modified accordingly. In stable disease the minimum medication tolerated is continued while in progressive disease treatment is intensified (additional medication/laser/surgery).

Reinstruct patient about compliance-consider laser trabeculoplasty (for OAG) in an attempt to reduce medication and improve compliance.

With diurnal large fluctuations despite prostaglandin therapy consider SLT/Surgery to blunt these and achieve low IOP.

In consult with physician adjust BP medications to avoid nocturnal hypotension.

Progression tends to be faster in ACG, Juvenile and Pseudoexfoliation glaucoma. Thus these need closer monitoring. Progression tends to continue despite best efforts in advanced glaucoma with ischemic element, thus needs aggressive IOP lowering to low teen or single digit levels in an attempt to achieve adequate perfusion pressure.

With improved technology and statistical programs it is possible to assess progression more reliably. However with current technology subtle progression still cannot be detected.

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4. $-Zone Parapapillary Atrophy and the Velocity of Glaucoma Progression. Christopher C Teng et al.
5. Visual Field Index Rate and Event-Based Glaucoma Progression Analysis: Comparison in a Glaucoma Population. Pilar Casas-Llera et al, BJO 2009

MEDICAL MANAGEMENT OF POAG

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Here we present 2 articles on a single topic by 2 authors. The first of these articles given us a brief guideline to Medical Management, while the second is a more detailed study on this very important aspect of glaucoma management

The aim of treatment is not so much a “normal” IOP but a pressure that proves safe in an individual. The oretically the treatment of each patient should be tailor made. The weakest drug for achieving the desired IOP with the least side effects should be selected and administered prior to individual peak pressure levels on the diurnal curve. The two eyes should be treated individually and given differing regimens if they show differing behaviour. In practice such a management is unrealistic and rarely possible or even useful.

The current practice is to estimate the pressure level (range) below which further damage to the optic nerve is unlikely to occur (target pressure) and then aim to keep the IOPs consistently below this level or, at least, within the estimated range. The target pressure is estimated by noting the untreated level of IOP; the degree of optic nerve cupping and visual field loss; the family history of glaucoma; the presence of any other aggravating conditions such as diabetes mellitus or arteriosclerotic vascular disease, and the rate of progression if known. In the average patient, the clinician should aim for a pressure 20–30% below the initial untreated pressure. With greater optic nerve damage (e.g., 0.8 disc diameter cupping or more), increasing age, and more risk factors, the target pressure should be lowered. The target pressure should be reassessed periodically and lowered if progression, optic nerve hemorrhage, or increase in risk factors occurs. One should also keep in mind that in the AGIS (Advanced Glaucoma Intervention Study), not only was lack of progression associated with a low average IOP but also with no IOPs exceeding 18 mmHg during the entire 6 years of the study. So, maintaining the IOP consistently below 18 mmHg in the average glaucoma patient and lower yet in the patient with advanced disease seems like a reasonable goal.

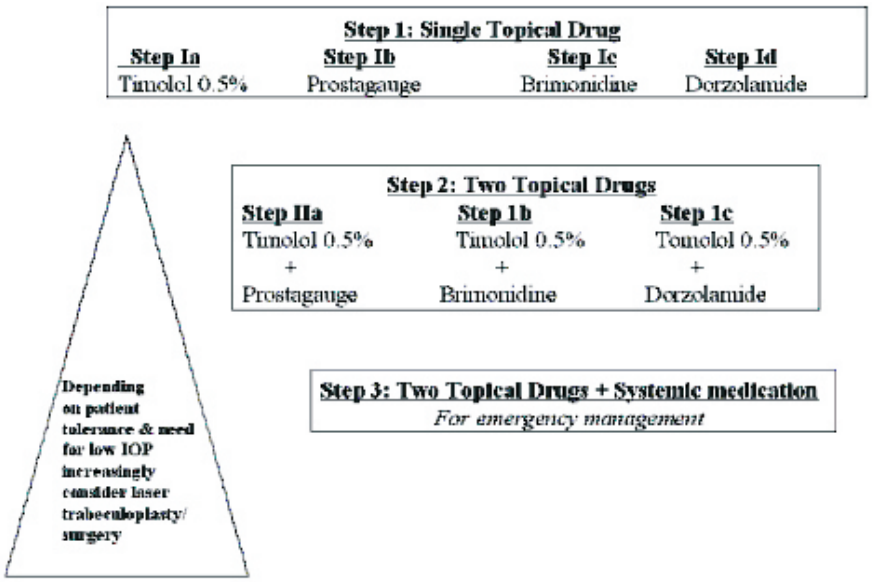
Treatment should be based on simplicity. The choice would depend upon the drugs available in the Indian market. These are as per Table 1 -

Table 1. Pharmacology of the topical anti-glaucoma medicines available in India

Drug	Ocular side-effects	Systemic side-effects	Efficacy	Contraindications	Daily dosage
Timolol 0.5%	+ Stinging, local anaesthesia	+ to +++ Bronchospasm, bradycardia, decreased cardiac output, hypotension, depression, impotence, altered lipid profile	+++	Asthama, COPD, CHF, Sinus bradycardia, atrio-ventricular blocks	1x to 2x
Brimonidine	++ Conjunctival blanching, ocular allergy	+ to ++ Drowsiness, fatigue, blood pressure changes	++ to +++	Use of MOA inhibitors, Parkinson disease, hypertensive crisis	2x to 3x
Dorzolamide	++ Punctate keratitis, ocular allergy	0 to ++ Bitter taste, headache, asthenia, renal calculi	++	Hypersensitive to sulfonamides, eye injury	2x to 3x
Prostagouges Latanoprost 0.005% Bimatoprost 0.03% Travoprost 0.004%	+ to ++ Iris pigmentation, punctuate keratitis	0 Headache, symptoms of URTI	++++	Ocular infection or inflammation	1x

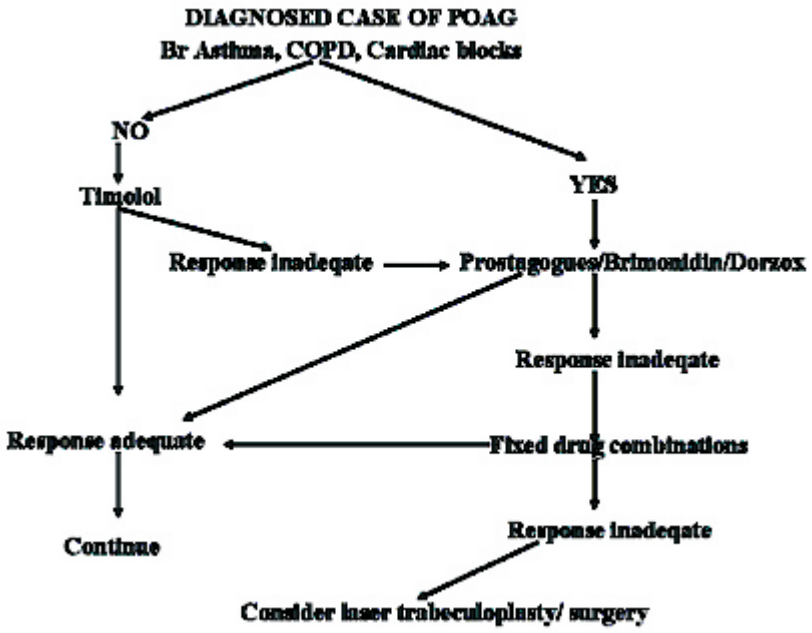
The pharmacological basis of drug treatment is based on the following steps

Table 2. **"Steps" of treatment**



Based on the drugs available and the treatment steps the following treatment algorithm is suggested.

Table 3. **Suggested treatment Algo**



The following general considerations are also of importance.

1. **Selection of the most appropriate medication**
   a. Greatest chance of reaching target
   b. Best safety profiles
   c. Minimally inconvenient
   d. Affordable
2. **Treatment should start low and slow**
   a. Minimal concentration
   b. Mimimal frequency
3. **The likelihood of compliance should be maximised**
   a. Patient and family education
   b. Least complex regimen
   c. Least disruption of lifestyle
4. **Technique of drop instillation**
   a. The preferred method is punctual occlusion for three minutes and eyelid closure for three minutes ('double DOT' – **D**igital **O**cclusion **T**echnique and **D**on't **O**pen **T**echnique)
   b. If two or more drops are being instilled, there should be a gap of more than 05 minutes

Other considerations

1. Patients suffering from severe dry eyes would benefit from non-preserved medication.

2. Fixed drug combinations are preferable. Fixed combinations of medications offer several potential advantages over combined use of the separate component medications including enhanced convenience, improved adherence, reduced exposure to preservatives, and possible cost savings.

Suggested Reads

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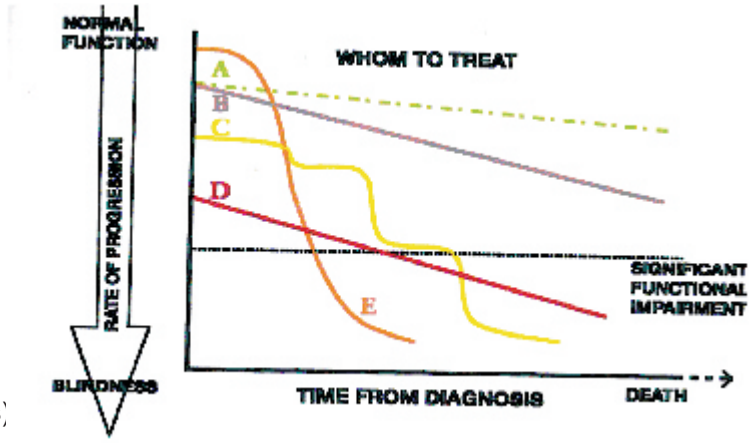
CHOICE OF APPROPRIATE MEDICATION IN GLAUCOMA PATIENTS

Dr. Udayan Dixit
MBBS, MS, DNB

There is a paradigm shift in the management of Glaucoma. Right from the definition of Glaucoma which takes away the dictum of IOP value as the sole criterion of diagnosis to the treatment protocols. At present the treatment is started only after a certain diagnosis of glaucoma and not empirically. The gold standard of diagnosis is appearance of optic disc, the cup disc ratio, the Neuro Retinal Rim thinning and notching, Nerve Fiber Layer defects etc. . It is recommended to be more aggressive in treating progression. Evolution of Automated Perimetry, GDX nerve fiber analyzer, OCT, UBM, Visante, Multifocal ERG have helped a lot in diagnosis of doubtful cases and to judge progression.

Here it is important to remember, that by treating a patient of glaucoma, we are trying to 'slow down' the rate of progression of nerve fibre layer loss & not 'curing the disease. This fact is very well illustrated in the following graphical plot (from European Glaucoma Society Guidelines)



(From EGS Guidelines)

As far as medical treatment is concerned we have come a long way from treating a patient with a single drop called Pilocarpine to a plethora of medicines available at our disposal. Sometimes too many choices of drugs can be confusing rather than useful. One may use medicines haphazardly, without discretion or any scientific basis. It is very necessary to choose a drug appropriately to treat a patient scientifically.

Days of deciding medicines on a typical expression of "In my experience" or anecdotal reports are over. A consencious doctor should now decide his medical treatment on Evidence Based Medicine or follow useful guidelines from GSI [Glaucoma Society of India] and EGS [European Glaucoma Society.] We will see in a nutshell how in an Indian scenario we should decide about "Appropriate Medicines" to treat a glaucoma patient. Let us imagine a situation of a patient in your clinic whom you have diagnosed to have glaucoma. How should one proceed to decide about the medication.

I will recommend the following parameters.

1] Decide the type of glaucoma that the patient has. Primary or secondary. If the type of primary glaucoma is open angle or angle closure. What is the cause of secondary glaucoma viz Uveitis with glaucoma, aphakik or pseudophakik glaucoma, angle recession glaucoma, drug induced glaucoma, glaucoma associated with growths and tumours, Anterior

Segment Dysgenesis etc. In case of secondary glaucoma one has to off course treat the primary cause first.

2] What is the extent of damage that glaucoma has already caused. This will be decided by evaluation of the optic disc, extent of field loss noted by automated perimetry and the level of IOP noted at baseline. One can note if the patient has minor, moderate or advanced glaucomatous damage.

3] General health of the patient especially conditions which would contraindicate use of certain drugs. One should see the socio economic status of the patient. Patients education or more practically the extent to which the patient is aware of the disease. Try and judge the compliance and follow up a patient is likely to keep.

Without going into pharmacological intricacies we will see the pros and cons of commonly used anti glaucoma medicines. This should enable us to have some kind of protocol in our mindset while treating a patient of glaucoma with medication.

1] Beta - blocker : Non Selective : Timolol Maleate 0.25% and 0.50% Dose twice daily Levobunalol 0.25% Dose once or twice daily. More soothing locally than Timolol.

Carteolol 1% and 2% Dose twice daily. Better for maintaining normal HDL lipid levels.

Gel forming solution of Timolol : Dose once daily. Less absorption and hence less systemic side effects

Selective Beta-blocker : Betaxolol 0.25% Dose twice daily.

Today, in India, this is the most commonly used, time tested medicine. It acts by reducing aqueous formation. It is useful to treat minor or moderate glaucomatous damage even as a mono therapy. Unsafe in patient having Bronchial asthma, cardiac block and arrhythmias. Caution has to be used in Insulin treated diabetics since it can mask symptoms of hypoglycemia. Caution should be followed to use it at night time for likelihood of nocturnal hypotension. Selective beta blockers are not totally selective in avoiding beta-blocker related complications and are less effective to reduce IOP compared to non selective beta blockers. Topical beta blockers have very negligible local side effects. It has great value in young or old, healthy patients with minor and moderate field loss as mono therapy. It is the most useful adjunct in all prepared combination drugs.

2] Alpha adrenergic agonist : Brimonidine Tartarate : 0.20%, 0.15%, 0.10% Dose twice or thrice daily

Acts by reducing aqueous formation , facilitates drainage through uveo sclera channel and tall but as yet unproven claim of Neuro protection. It is useful in all grades of glaucomatous damage. It has a good safety profile as far as systemic side effects are concerned. Local side effects of congestion, eyelid pruritus are may be bothersome in few patients. Drowsiness can be encountered. It is unsafe in small children. By reducing it's strength from 0.20 % to 0.15% to 0.10% is an attempt to reduce local side effects and drop outs due to the same. The dosage recommended is thrice for 0.15% and 0.10 % or twice daily for 0.20 %.

Apraclonidine : Iopidine : Used before and after procedure of Yag Laser Iridotomy. Not useful in prolonged treatment of glaucoma due to its very transient action and local side effects.

3] Carbonic Anhydrase Inhibitors : Local drops Dorzolamide or Brinzolamide

Acts by reducing aqueous formation. Useful in all grades of glaucomatous changes. Drops have no significant systemic side effects. Local side effects (of congestion, pruritus) are not commonly seen. Brinzolamide is more tolerable than Dorzolamide locally. The ideal dosage is thrice daily. It is useful in combination therapy.

Systemic Acetazolamide : Oral : 250 mg. tablets maximum thrice daily. sustained release 500mg. capsules maximum twice daily.

It is very Potent in reducing IOP. May not be tolerated as long term therapy unlike topical medicines which can be used lifelong. This is due to side effects of tingling, numbness, gastritis, and increased chance of renal stones. Caution has to be followed in patients with allergy to Sulfa drugs. It also potientiates hypoglycaemic effect of oral anti-diabetic drugs.

Intra Venous Acetazolamide : Useful in acute episodes of elevated IOP, in situations like Central Retinal Artery Occlusion .

4] Cholinesterase related drug: Pilocarpine 1%, 2% & 4% Dosage is decided on individual case twice to four times daily.

One of the oldest remedies to treat glaucoma. Acts by pupillary constriction and also cilliary body rotation. This leads to opening up of peripheral angle crowding. Useful in Primary angle closure glaucoma not caused by pupillary block. Used as intense therapy to abort acute attack of angle closure glaucoma .

Causes congestion, headache. Can cause reduced vision in patients having cataract. Tachyphylaxis is known. It has dreadful complication of precipitation of pupillary block glaucoma. It is useful in preparation before doing YAG Laser Peripheral Iridotomy. It has a minor role in treating open angle glaucoma too by rotating the ciliary body.

5] Prostaglandins, Prostamides and related drugs :

Latanoprost : 0.005 % Cold chain has to be maintained till the bottle is opened

Bimatoprost : 0.03 % : No need of refrigeration

Travoprost : 0.004 % : No refrigeration needed

Dosage of all the three drops is once at bedtime

Today in the western world these class of drugs have become “Gold Standard Of Medical Treatment”. It is the first drug of choice in the developed and economically sound countries. This “King of Anti Glaucoma Drugs” acts by increasing facility of outflow through trabecular meshwork and Uveo Sceral Outflow too. Very potent in reducing IOP. Hence it is useful in treating advanced as well as moderate and minor grades of glaucoma. It has a good systemic safety profile. Local side effects can be congestion, pruritus, SPK on cornea, increased lengthening of eye lashes. Increased pigmentation of iris of no consequence in dark irides. Cystoid macular edema in pseudophakics not having intact posterior capsule is a possible risk. Low grade anterior uveitis in patients prone to inflammation may be noted. Today there is a neck and neck race to claim the fame for maximum IOP reduction, duration of action, etc. These drugs are combined with Beta blockers & Alfa Agonists to give additional IOP lowering effect.

An important thing to remember is , Prostaglandines may take some time to initiate the full action. Hence they should not be abandoned as “non effective” too soon.

6] Hyperosmotic agent- Mannitol : 20% Intra Venous medicine.

Used as an drug of choice in emergency situations, like sudden rise of IOP in acute angle closure attack or intra/post-op rise in IOP.

They increase the blood volume, thereby reducing aqueous secretion. They may cause overload effect on heart & also cause metabolic imbalance (in renal & diabetic patients).

Other options like Eserine, Dipivefrin Epitrate, Carbachol Ecothiophate & Epinephrine are not used any more.

IMPORTANT CONSIDERATIONS :

Before we consider long term use of anti glaucoma medication as a line of treatment some considerations are important. We should categorize patients into two groups.

Group I	**Group II**
Educated	Uneducated
Understands disease	Ignorant of disease consequences
Good compliance in medication and follow up	Poor compliance
Affording capacity good	Poor affording capacity

For group I patients, lifelong medical treatment can be a good option to preserve visual function till the end.

For group II patients consider medication as initial line of treatment but option of early Laser or surgical intervention may have to be considered at a much earlier stage, because of poor compliance.

CONCEPT OF TARGET IOP : It is an estimate of the mean IOP obtained with treatment that is expected to prevent further glaucomatous damage. Target IOP is individualized to each eye of each patient & is not a constant number, but a range of IOP which will reduce the rate of neuronal damage to minimum.

Various glaucoma studies have shown that a 25-30% reduction in IOP from baseline or maintaining IOP below 18mm Hg on all follow up visits helps in preventing damage.

The goal for us clinicians is to try & achieve the target IOP with least amounts of medications & minimum side effects.

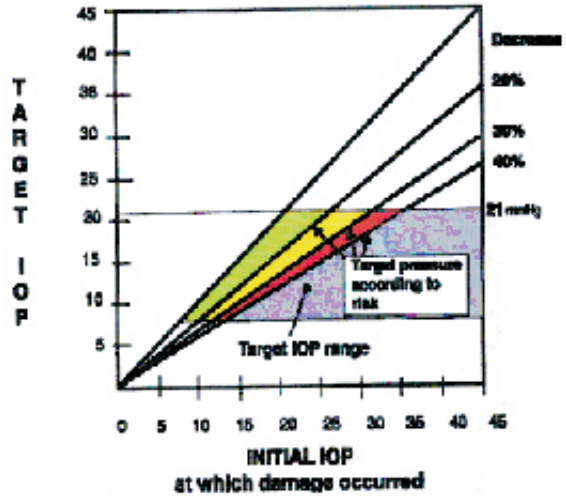


Fig. 3.2.1 - TARGET IOP Diagrammatic evaluation of the desired therapeutic outcome in form of IOP-lowering. The target pressure should be situated within the shaded area. The lower is the initial IOP, the lower will be the Target IOP and viceversa. The percentage of IOP reduction targeted (i.e. 20%, 30%, 40% respectively) depends mainly on the degree of VF damage at diagnosis and on rate of progression (RoP).

(From the European glaucoma society guidelines)

The factors on which the target IOP is decided are -

1. IOP before treatment
2. Risk of IOP related optic nerve damage, which depends on average IOP, maximum IOP & IOP fluctuations
3. Stage of glaucoma, more severe the glaucomatous damage, lower the target IOP.
4. Rate of progression of glaucomatous damage
5. Age of patient

6. Life expectancy of patient
7. Other risks factors.

There has to be periodic re-evaluation of target IOP depending on efficacy of the treatment & cost vs benefit (effect on quality of life).

Unfortunately one of the limitations of target IOP approach is that, we only know in hindsight whether the target IOP selected initially was adequate or not. In other words a patient must get worse before we verify that the target pressure was inadequate!

SUCCESS OF MEDICAL TREATMENT :

To judge success of medical treatment one has to see if IOP is lowered to adequate grade so that there is no progression of field loss or further narrowing of Neuro Retinal Rim.

If IOP is not lowered at all : Change or substitute the drug

If IOP is lowered but not to the desired level : Add a drug.

If the drug is not tolerated : Change the medicine.

If Maximum Tolerated Medical Treatment [MTMT] does not achieve target IOP : Consider Laser or Surgery.

COMBINATION THERAPY :

Combination therapy is used when two or more drugs are necessary to control glaucoma. Comparison of combination vs successive instillation of two drops have shown "Non Inferior Results" meaning they are almost equally effective as serial instillation of two different drops.

Advantages of Combination therapy are cost, convenience and compliance is very good with combination drugs.

Multiple combination drugs are available :

Pilocarpine with Beta blocker
Alpha agonist Brimonidine with Beta blocker
Dorzolamide with Beta blocker
Brinzolamide with Beta Blocker
Latanoprost with Beta blocker
Bimatoprost with Beta Blocker
Travoprost with Beta Blocker

DRUG COMBINATIONS ADDITIVE EFFECT

CURRENT DRUG	ADDITIONAL DRUG				
	"2 agonists	$-blockers	Topical CAIs	Cholinergle	Prostagladin / Prostamides
"2 agonists		+	+	+	+
$-blocks	+		+	+	+
Topical CAIs	+	+		+	+
Cholinergic	+	+	+		+ / -
Prostaglandins/ prostamides	+	+	+	+ / -	

(From EGS Guidelines)

TIPS AND PEARLS :

In brief in a patient in whom IOP needs significant lowering, prostaglandin derivatives are a good choice. Compliance is good, but affordability is an important factor.

In moderate or minor glaucomatous damage with no systemic contra indication Beta Blocker is a good choice. It has very few local side effects. It is easily affordable.

Alpha agonists and topical Dorzolamide would lie in between. It has good IOP lowering effects although local side effects can create many drop outs.

Never start on two medicines from the word go. We will never know which drug is acting optimally and to what extent. We may be doing unnecessary over medication to the patient. In exceptional situations if the IOP is very high and ocular structures like optic disc or vasculature is vulnerable for damage, one can commence more than one medicine.

If possible start treatment in the worst affected eye, to know a drugs pressure lowering effect in an individual patient. In case one drug doesn't reduce IOP to target level, replace it with molecule from another class(after waiting for washout effect of the first drug). Only when either a single drug or a combination of drugs is proven to be effective, should it be started in the contralateral eye.

COMMON CASE SCENARIOS :

1] 65 years old patient. During routine examination found to have IOP of 24 mm. of Hg. (both eyes). Vision 6/6 (both eyes). Optic disc has 0.7 cup. Gonioscopy showed open angles. Minimal field loss in automated perimetry. Field loss corresponds to thinning of NRR. What medicine to start? Topical Beta Blocker will be a good choice, given the relatively advanced age & mild damage of the ONH.

2] 46 years old patient with vague complaints. On examination vision 6/6. C:D ratio 0.8, thin NRR. Family history of glaucoma.

Topical Prostaglandin will be a good choice, because we want maximum IOP effect, with a single drug, to ensure better compliance, minimal systemic sideeffects, given the longer duration of treatment.

3] 56 years old patient. Felt he has diminution of vision because of cataract. Vision 6/24 both eyes. Grade I nuclear sclerosis. 0.9 C:D ratio. IOP 42 mm of Hg. Perimetry showed advanced field loss.

One can consider prostaglandin and call for early review or start a two drugs or combination since optic disc is very vulnerable.

4] 39 years old female patient. Complains of headaches off and on. IOP 26 mm. of Hg. Refraction + 2 sphere each eye. Van Herick test and Gonioscopy showed closed angles. Optic disc showed C:D ratio of 0.3, fields normal.

The definitive treatment will be YAG PI and not just medical treatment. Short term medication like Beta blocker or Pilocarpine can be used. Angles must be reviewed after PI. The IOP must be monitored to see if PI by itself is sufficient or medical supportive treatment is necessary.

As a carry home message, one must see the facilities of diagnosis in his or her clinic, access to higher diagnostic modalities. One has to judge the kind of patients one gets in the clinic. You should take guidelines from carious important glaucoma studies. Ultimately wisdom lies in the fact that **"NEVER EMULATE ANYBODY. FORM YOUR OWN PROTOCAL BUT RESTRICT THE PROTOCOL BASED ON SCIENTIFIC AND PROVEN PRINCIPLES".**

ANGLE CLOSURE GLAUCOMA

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Primary glaucomas are classified into open angle and narrow angle types primarily because the pathophysiology and management is of each of is different. Primary Open Angle Glaucoma (POAG) is a slowly progressive optic neuropathy, many a times asymptomatic, where the trabecular meshwork is the primary seat of pathology ,the IOP may be raised or normal, typical disc and visual field changes occur.

Primary Angle Closure Glaucoma (PACG), on the other hand may present with redness and pain, often recurrent, which can be seen as an acute/ subacute attack or present as a chronic glaucoma. There are certain specific risk factors which can be easily recognized. The basic pathology is a pupillary block, hence relieving this is an important step in management.

The prevalence of manifest PACG was found to be 1.08% and that of occludable angles 2.21% in subjects >40 years in the urban area of Andhra Pradesh eye disease study. It was found the prevalence of manifest PACG and occludable angles increases with age, and was more common in females, in hyperopia >2 D, and lower socioeconomic groups. Manifest PACG has caused blindness in one or both eyes in 41.7% of the subjects !

The Chennai Glaucoma study found prevalence of primary angle closure disease in the rural population to be 1.58%.They found women were at greater risk than men and increasing age was a significant risk factor. Almost all were unaware of the disease as it was asymptomatic !

Hence the need for a comprehensive examination cannot be over emphasized !

PRIMARY ANGLE CLOSURE GLAUCOMA

Primary angle closure is appositional or synechial closure of the anterior chamber angle caused by pupillary block. Primary angle closure glaucoma is not associated with other ocular abnormalities. Conditions included in this group are pupillary block glaucoma , plateau iris and combined mechanism glaucoma. Other forms of angle closure glaucoma related to lens conditions, inflammation, postsurgical have been grouped into secondary angle closure glaucoma.

PUPILLARY BLOCK GLAUCOMA

Due to relatively smaller anterior segment and a normal sized lens, associated with mid dilatation of the pupil there is increased resistance to flow of aqueous from posterior to anterior chamber through the pupil. This functional block results in forward bowing of the peripheral iris causing appositional closure of the angle of the anterior chamber, often associated with elevation of intraocular pressure (IOP). Such episodes may occur repeatedly and may lead to formation of peripheral anterior synechiae (PAS) and functional damage to the trabecular meshwork.

Angle closure may occur in acute, subacute or chronic form.

Acute angle closure glaucoma : the symptoms are sudden and severe, with marked pain, blurred vision and a red eye. Patient may also have nausea and vomiting.

Subacute angle closure glaucoma : the symptoms are mild or absent. There may be repeated subacute or subclinical attacks before finally having an acute attack or developing PAS with chronic pressure elevation.

Chronic angle closure glaucoma : a portion or the entire angle is permanently closed by PAS and IOP is chronically elevated.

Combined mechanism glaucoma : the diagnosis is made after an acute angle closure glaucoma attack in which the IOP remains elevated after peripheral iridotomy, despite an open, normal appearing angle

CLASSIFICATION :

Anatomic narrow angle (primary angle closure suspect) -

This includes any eye that has a primary, abnormally narrow angular width of the anterior chamber recess, wherein the peripheral iris is located close to, yet not touching the posterior pigmented trabecular meshwork. These patients are at risk for subsequent PAC.

Primary Angle Closure and Primary Angle Closure Glaucoma -

This is caused by pupillary block and can exist in acute or chronic state or they may have both and present with acute attacks superimposed on chronic angle closure. In PAC the eye is at risk of developing glaucomatous optic disc damage, particularly when associated with elevated IOP. In this case the eye has progressed to from PAC to primary angle closure glaucoma (PACG)

Acute primary angle closure -

If the entire circumference of the angle is obstructed suddenly, IOP will rise rapidly to high levels. This may cause corneal edema, vascular congestion, eye pain, or headache. High IOP may be accompanied by nausea and vomiting. Acute attacks may be self limited and resolve spontaneously or may occur repeatedly. Untreated this may cause permanent vision loss.

Chronic primary angle closure -

If only a portion of angle closes with PAS, IOP may be in the normal range or mildly elevated, and symptoms of acute PAC may be mild or absent. Continued, slowly progressive closure of the angle may ensue, eventually leading to sustained elevation of IOP and glaucomatous optic neuropathy. Patients with asymptomatic chronic PAC may present with severe visual field loss compared with mild to moderate defects found in patients with prior episode of symptomatic angle closure

CLINICAL FEATURES :

When examining every patient we must consider general risk factors and look for anatomic features that may predispose to angle closure. The gold standard examination is gonioscopy which can help to identify eyes with some form of angle closure or those at risk for ACG (occludable angle). The correct diagnosis will depend on understanding the symptoms, predisposing circumstances and physical findings as well as the differential diagnosis.

RISK FACTORS : Advancing age, Asian races, Hyperopia, Family history of angle closure, Female gender, Shallow peripheral anterior chamber.

DIAGNOSIS : Evaluation of both primary and secondary type of angle closure should be done. **Both eyes** should be examined completely.

HISTORY : blurred vision**,** recurrent pain, redness, coloured haloes, eyeache and use of ,sulfonamides, phenothiazines should be noted. Family history of acute ACG should be asked for.

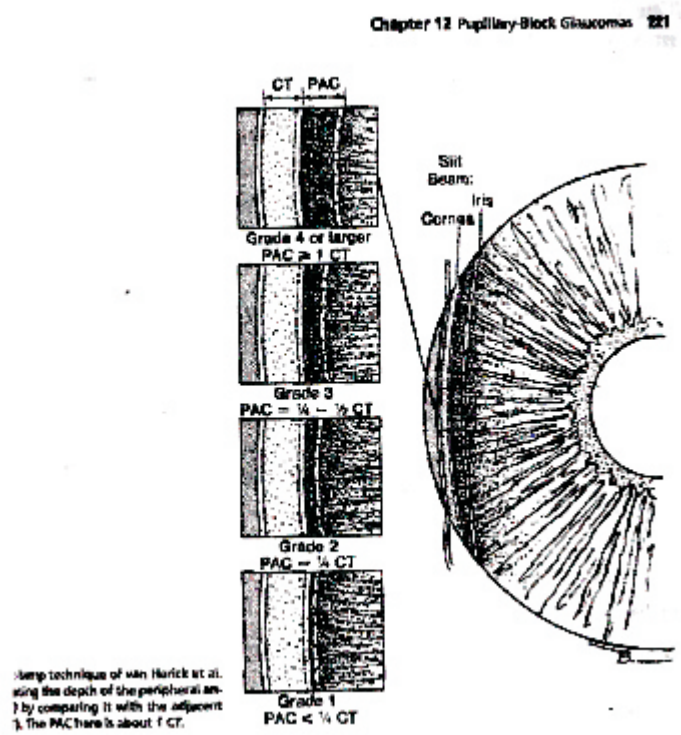
Role of provocative tests : These tests such as mydriatic and prone test do tell us if a patient is at risk, but could also precipitate an attack in a patient at risk. False negative and false positive rates are also high. Hence these test are avoided in routine practice. Careful gonioscopy in the context of history and other physical findings can help to make management decisions.

PHYSICAL EXAMINATION -

1. **Assessment of refractive status :** as hyperopic eyes are at increased risk of PAC.
2. **IOP :** measured in both eyes by applanation tonomtery.

   Slit Lamp Examination.
3. **Conjunctiva :** May be normal. Congestion is seen in Acute cases.
4. **Cornea :** is clear in most cases. Corneal edema is seen in acute cases and in long standing chronic cases. Pigment dispersion on the endothelium may be seen following an acute attack.
5. **Anterior Chamber :** Central and Peripheral Ac depth is assessed. Van Herick technique of comparing peripheral AC depth (PAC) to the thickness of adjacent cornea (CT) is helpful to assess peripheral AC depth. Anterior chamber inflammation may suggest recent or current attack.
   - ✂ Grade 4 : PAC >= CT
   - ✂ Grade 3 : PAC =1/4 -1/2 CT
   - ✂ Grade 2 : PAC =1/4 CT

Grade 1 : PAC<1/4 CT (occludable)



6. **Iris :** mid stromal iris atrophy, especially sectoral, posterior synechiae
7. **Pupil :** sphincter atrophy is seen following an acute attack and repeated subacute attacks. There is spiraling of the adjacent iris, both signs indicative of ischemic damage. There is resultant mid dilated pupil, vertically oval pupil and sluggish pupillary reaction.
8. **Lens :** pigments on the anterior surface**,** glaukomaflecken may be seen**.**
9. **Gonioscopy :** Should form a fundamental part of comprehensive eye examination**.** The purpose of gonioscopy is to evaluate angle anatomy, appositional angle closure, and presence of peripheral anterior synechiae.

This is done with a Goldmann 2 or 3 mirror goniolens **or** Zeiss or Sussman 4 mirror lens. The patient should be examined in dim roomlight using a short, narrow slit beam to avoid constricting the pupil and artefactually opening the angle. Care should be taken to avoid pressure on the cornea.

The normal angle structures as seen from posterior to anterior are :

Root of iris, ciliary body band, scleral spur, trabecular meshwork posterior and anterior part and schwalbe's line.

Gonioscopic grading of the angle structures.

- Grade 4 = ciliary body band and sclera spur visible
- Grade 3 = posterior trabecular meshwork visible
- Grade 2 = anterior trabecular meshwork visible
- Grade 1 = schwalbe's line visible
- Grade 0 = no angle structures seen.

Grades 0 - 2 in ¾ or more of the angle circumference constitute an occludable angle.

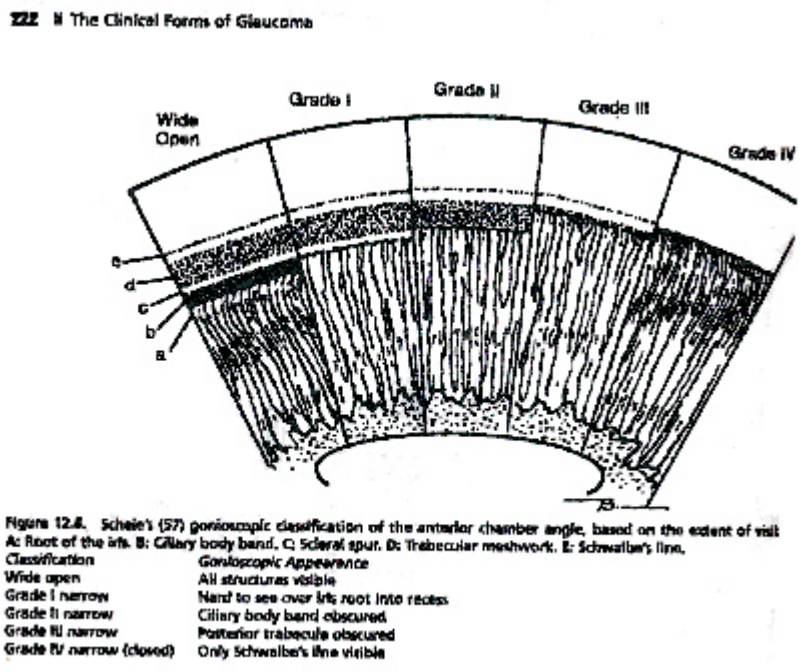


Figure 12.8. Scheie's (57) gonioscopic classification of the anterior chamber angle, based on the extent of visi
A: Root of the iris. B: Ciliary body band. C: Scleral spur. D: Trabecular meshwork. E: Schwalbe's line.

Classification	Gonioscopic Appearance
Wide open	All structures visible
Grade I narrow	Hard to see over iris root into recess
Grade II narrow	Ciliary body band obscured
Grade III narrow	Posterior trabecula obscured
Grade IV narrow (closed)	Only Schwalbe's line visible

Dynamic Indentation by 4 mirror Gonioscopy -

When gentle pressure is applied by the lens on the centre of the cornea, the aqueous is pushed back, thus widening the angle recess. When the iris lies in contact with the trabecular meshwork in appositional closure, the angle can be re-opened by gentle pressure. If there are adhesions between the iris and the meshwork, i.e synechial closure, that portion of the meshwork would remain closed even after pressure. The angle recess widens but iris strands remain attached to the angle.

This technique is useful where the curvature of the peripheral iris is convex making it difficult to identify the different angle structures.

10. **Fundus :** An attempt should be made to evaluate the fundus and optic nerve using a direct ophthalmoscope or slit lamp biomicroscope .Dilatation is not advisable until a Peripheral iridotomy is done. The optic disc may be hyperemic with congested vessels in an acute case, while pallor ensues following an acute attack. There may not be the typical disc signs that are seen in primary open angle glaucoma. In chronic cases, typical disc changes may be observed.
11. **Visual field examination :** Is done when the acute/subacute stages are adequately controlled.

Newer Techniques for diagnosis -

The **Ultrasound Biomicroscope (UBM)** uses high frequency ultrasound to identify the relationship of iris, posterior chamber, lens, zonules and ciliary body. This technique has potential value in understanding the mechanisms of glaucoma especially when the media are not clear. One can perform dark room provocative testing with UBM to study spontaneous occlusion of the angle . This helps to identify people at risk. It can help in identifying relative pupillary block, PAS, and plateau iris configuration.

Anterior Segment OCT enables high resolution realtime imaging of anterior segment structures. The AS-OCT has the benefit of being a rapid, non contact method that may be performed with the patient sitting in an upright position, therefore having an advantage over UBM. The AS –OCT allows qualitative and quantitative evaluation of the anterior chamber angle. The dynamic relationships between peripheral iris and trabecular meshwork, can be assessed , functioning as a light dark provocation test. The dynamic interactions between lens and iris are useful to assess pupillary block. As it is non contact method done in upright position, artificial opening the angle caused by indentation or posterior movement of lens iris diaphragm that may occur in the supine position are avoided here. This proves to be a promising diagnostic and screening tool for detection of angle closure.

Differential Diagnosis -

Primary narrow angles and PAC tend to be bilateral. Finding a wide open angle in the fellow eye suggest a diagnosis other than PAC. The DD of PAC include secondary angle closure glaucomas which may be unilateral or bilateral.

1. Neovascular glaucoma
2. Iridocyclitis (posterior synechiae, iris bombe)
3. Ciliary body engorgement caused by CRVO, panretinal photocoagulation, use of systemic medications (sulphonamides, phenothiazines)
4. Aqueous misdirection syndrome (after incisional or laser iridotomy)
5. Lens induced angle closure (phacomorphic or subluxated)

Other mechanism of angle closure are as follows :

Plateau Iris -

Causes angle closure by direct obliteration of the chamber angle recess, crowded by the iris base when pupil is dilated. This can occur with

1. The peripheral iris is thick (iris rolls)
2. The iris base inserts anteriorly
3. The ciliary processes are displaced anteriorly and push the iris base into the chamber angle.
4. The iris profile from the periphery to far periphery is flat and steep, creating an extremely narrow angle recess.

This condition can be confirmed by ultrasound biomicroscopy. These anatomical findings explain why plateau iris mechanism (configuration) is not altered by iridotomy. While this configuration is common, plateau iris syndrome is relatively rare in which acute angle closure occurs with a raise IOP inspite of a patent iridotomy.

Plateau iris can be treated by argon laser iridoplasty and/or use of miotics.

Lens mechanism : Large and/or anteriorly placed crystalline lens can predispose to angle closure and can worsen a pupillary block.

Creeping Angle closure mechanism: The iris base creeps on to the trabecular meshwork forming irreversible PAS and causes chronic angle closure glaucoma.

Posterior aqueous misdirection mechanism: rarely this can be a cause for primary angle closure.In these cases, the ciliary processes come in contact with the lens equator and/or posterior capsule, causing misdirection of aqueous into vitreous. The lens iris diaphragm is pushed forward and occludes the chamber angle.

MANAGEMENT :

GOALS 1. Reverse or prevent pupillary block. 2. Control IOP 3. Limit damage to optic nerve

Anatomic narrow angle (primary angle closure suspect) -

Laser Iridotomy is THE first step of treatment in all cases of PACG, irrespective of the stage at which they are seen. Peripheral Iridotomy (PI) should be considered in eyes at risk for developing angle closure.

A PAC suspect can be explained about the symptoms and can be observed. Alternately an iridotomy can be done, especially if the patient is unable to comprehend the symptoms and comply with follow up instructions. Iridotomy is indicated in patients with narrow angles who require repeated pupillary dilatation for treatment of other eye disorders (e.g., ARMD, diabetic retinopathy)

Indications for iridotomy :

- ✂ A potentially occludable angle is present
- ✂ Presence of PAS with progressive narrowing of the angle
- ✂ Symptoms suggestive of prior angle closure attack.
- ✂ For the fellow eye in the patients who have had an attack of acute ACG.
- ✂ Medication is required that may provoke papillary block.

Patients at risk should be warned of the danger of taking medicines that could cause pupil dilatation and induce an angle closure attack. (over the counter decongestants, motion sickness medication, anticholinergic agents).

Acute primary angle closure -

The aim of treatment is to break the acute attack by doing a peripheral iridotomy by means of laser or incisional iridotomy. Iridotomy allows aqueous to bypass the pupillary block and reduces pressure gradient between the anterior and posterior chambers.

Medical therapy is initiated to control IOP , to reduce pain and associated inflammation and clear the cornea for preparation of iridotomy. Treatment with miotics is effective as iris becomes stretched and less bowed with a small pupil, and this lowers the IOP by opening the pupillary block. Pilocarpine also opens up the angle to a little extent.

Laser iridotomy should be done in all cases. The iridotomy reverses appositional angle closure, and prevents the formation of PAS. If iridotomy cannot be performed due to corneal edema , the cornea may be cleared with topical hyperosmolar agents. Appropriate timely treatment will prevent damage to optic nerve, trabecular meshwork, iris, lens and cornea. Assess the angle again after laser iridotomy to look for PAS and angle damage, and continue antiglaucoma medications as required. Disc and visual field damage has to be assessed. Filtering surgery is indicated when IOP is not controlled as desired , there is extensive synechial closure and disc and field damage is progressing.

The fellow eye should be evaluated since it is at high risk for similar event. The fellow eye should be treated with prophylactic iridotomy if the anterior chamber is anatomically narrow. This is very effective in preventing acute angle closure in the fellow eye.

Chronic primary angle closure -

These patients have chronically elevated IOP. Peripheral iridotomy is performed to relieve pupillary block. It halts the progression of synechial closure and progressive elevation of IOP.

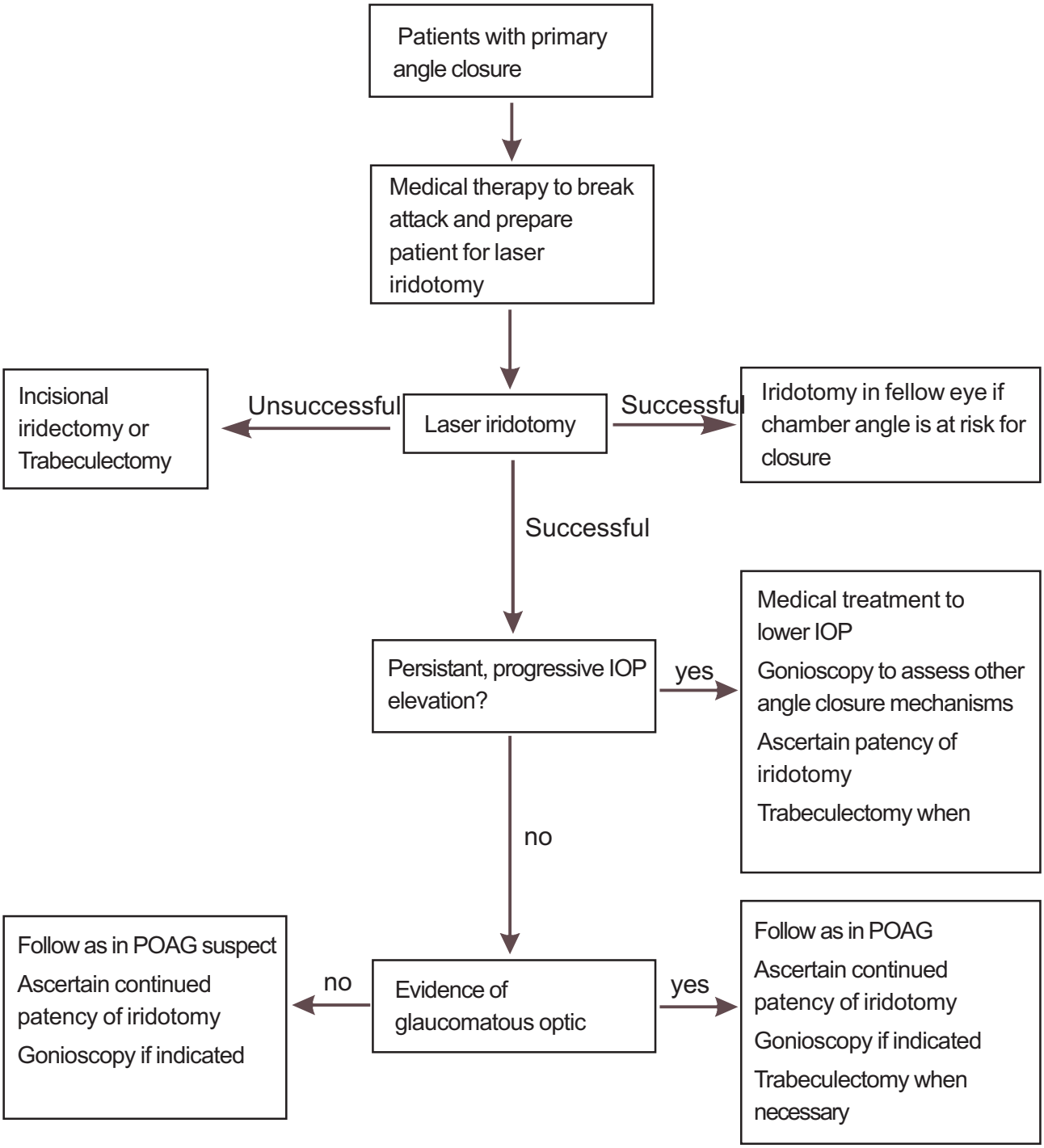
Follow up evaluations after iridotomy :

- ✂ Check patency of iridotomy
- ✂ IOP measurement
- ✂ Gonioscopy
- ✂ Pupil dilatation to decrease risk of posterior synechiae formation
- ✂ Fundus evaluation.
- ✂ Visual field evaluation.

Medical treatment is continued based on IOP, disc and field damage. Filtering surgery is indicated when IOP is uncontrolled or disc and field damage worsen.

Role of Lensectomy : Cataract surgery with a PCIOL implant in eyes with a narrow angle results in a wider angle and will reduce the risk of pupillary block. Cataract surgery has been shown to lower p.o medication requirement and decrease complications, However there is insufficient evidence currently to recommend use of cataract surgery in the management of Primary Angle Closure.

Algorithim for management of angle closure glaucoma -



Source : American Academy of Ophthalmology

AHMED GLAUCOMA VALVE

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The chequered history of aqueous drainage devices began in 1906 when Rollet introduced horse hair through a corneal puncture in a case of absolute glaucoma. Silk threads, gold drains, lacrimal cannula, tantalum, glass tube, platinum wire, protoplast loop, absorbable gelatin, polythene tubes have all been tried by various investigators but none have proved to be of any value as they failed in their primary intention of pressure reduction.

Understanding the causes of their failure gave birth to a host of newer methods of draining the anterior chamber. The causes of failure of these earliest devices were due to

A) Plugging of the outer unprotected end of the tube with fibrous tissue which naturally was excessive due to the foreign body reaction incited by the materials of the implant,

B) Plugging in of the posterior end of the tube with conjunctiva due to the weight of the hydrated Tenon's capsule causing the conjunctiva to collapse,

C) Implant exposure and extrusion through the thin anterior conjunctiva.

For an implant to be ideal it should -

a) Be made of non reactive synthetic material which does not induce foreign body reaction and to which fibroblasts adhere to poorly;

b) Should be able to shunt aqueous posteriorly so that there is reduction of long term inflammation, thinning and fibroblastic reaction;

c) Should be able to establish a potential space around which drained aqueous can pool and be sequestered for incremental absorption and

d) Should be made up of a tube attached to a plate of greater height than the plate thus preventing the collapse of conjunctiva.

The features of the Ahmed glaucoma device which consist of a flexible plate made of polypropylene or silicone, silicone valve, elastomer membrane and silicone tube would seem to qualify it to conform to the qualities of an ideal implant. This device has become very popular amongst all the glaucoma drainage devices. Several models and modifications of the Ahmed valve are also present.

The **indications** for implantation of this device are -

1) Paediatric glaucomas
2) Neovascular glaucomas
3) Uveitic glaucomas
4) Glaucoma due to pseudophakia
5) Glaucoma after post vitreo-retinal surgery, post penetrating keratoplasty
6) Failed trabeculectomies
7) Scarred conjunctiva
8) Any multi- operated, unresponsive glaucoma

SURGICAL METHOD OF THE AGV – FP7

Ahmed glaucoma valve implantation is done under peribulbar anaesthesia for adults and under general anaesthesia for children. IV Mannitol is maybe given before surgery if necessary. Preoperative antibiotic drops are instilled.

The thickness of the sclera should be assessed **pre operatively** so that scleral graft or cornea can be procured before surgery. We have also used goretex in a patient whose sclera was found to be very thin pre operatively He was a case of bilateral aniridia with superiorly subluxated cataracts who had repeated surgeries including a trabeculectomy in the superotemporal quadrant.This assessment will prevent a surgical surprise after incising the conjunctiva.

The conjunctival sac is thoroughly irrigated with betadine solution and then washed with saline. Superior rectus bridle suture is then applied.

The conjunctiva is incised in the superotemporal quadrant from ten o'clock position to at least 12 ºclock position at the limbus and from 10 ºclock extended posteriorly for at least 6-7 mm. Other quadrants can also be used for valve implantation. The incision should liberal to allow easy insertion of the plate. The conjunctiva is dissected deeply with the help of conjunctival scissors and any fibrous adhesions are excised. The conjunctival pocket should be deep for valve insertion. The valve is then prepared by holding the plate firmly between the fingers without any pressure on the valve as pressure will spoil its delicate valvular mechanism. The cannula of a 10 ml syringe, filled with saline, is carefully inserted into the tube and fluid is injected into it. Utmost care is taken to ensure that the valve does not fall because the pressure of the injecting fluid is > 100 mm water pressure. Once the saline flows out of the tube, the valve is considered to be **primed** and the plate is then held with forceps on the side of the plate and pushed deeply into the conjunctival pocket. The anterior end of the plate should be at least 8 mm away from the limbus. Then, 8-0 silk is used to attach the eyelets on plate to the sclera. The suture can be taken from the eyelets in the plate onto the sclera or vica versa .The bite should pass through partial scleral thickness and the knots should be tight to prevent movement of the plate.

The tube is then inserted into the anterior chamber but has to be covered so that there is no erosion of the overlying conjunctiva. Molteno initially used a partial thickness scleral tunnel which is being successfully used now. For this method a partial thickness incision is made on the sclera about 2 mm from the limbus and a parallel incision about 6 mm behind it. Then with the help of a cresent blade the two incisions are joined so that a scleral tunnel is created. Other methods of covering the tube consist of partial thickness scleral flap, use of partial thickness preserved sclera and preserved cornea.

A 23 g needle is then bent at right angles and passed through the limbus into the anterior chamber in line with the tube. The tube is the gently held with non toothed forceps or a tube inserter provided by the manufacturers and pushed into the opening after passing it through the scleral tunnel. The length of the tube that should be inside the anterior chamber is 2-3 mm and its cut end should be beveled up. The opening in the limbus should not be enlarged as the tube should fit snugly in the passage preventing leakage of aqueous by the sides of the tube and postoperative shallowing of anterior chamber. One suture can be used to fixate the tube firmly to the sclera near the limbus.

To prevent post operative hypotony, Healon is injected into the anterior chamber at the end of surgery. The conjunctiva is then reposited and sutured firmly with continuous 8-0 vicryl sutures.

Antibiotic eye ointment is applied after subconjunctival injection of dexamethasone with amikacin. The eye is bandaged for 24 hours after which topical antibiotic drops are given 6 times a day, steroid antibiotic combination 6 times a day.

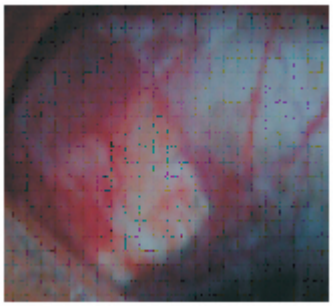
Complications

Proper surgical care and correct technique are able to prevent most post operative complications. The most commonly occurring complications are as follows:

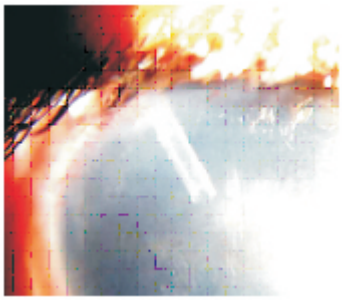
1. Shallowing of anterior chamber – This is most commonly due to leakage of fluid by the sides of the tube
2. Hyphema
3. Post operative rise of IOP due to Healon. The IOP also rises postoperatively after about a 2-4 weeks postoperatively due to the encapsulation of the plate.
4. Malignant glaucoma – recognized by shallow anterior chamber and rise of IOP
5. Infection
6. Endothelial cell loss
7. Tube retraction
8. Tube exposure
9. Iris incarceration into tube – which can be managed by dilatation of the pupil, if found early during the post operative period.

Photographs of Ahmed Glaucoma valve implantation in (A) buphthalmic eye with partial thickness scleral graft, (B) Tube in a case of aniridia with uncontrolled IOP, (C) Partial thickness (ePTFE) Goretex patch on tube, (D) Scleral tunnel for tube cover (E) Well formed bleb with shallow AC and tube endothelial touch after malignant glaucoma

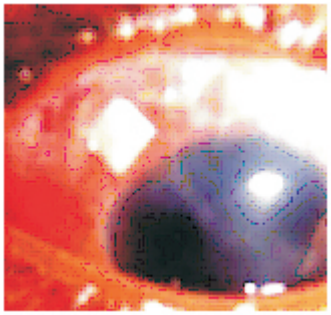
(A) Partial thickness Scleral patch covering the tube



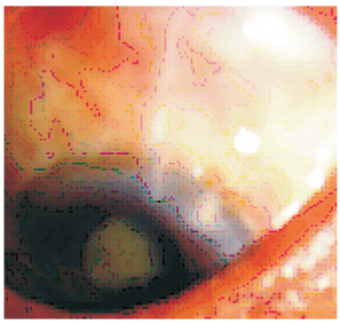
(B) Tube in anterior chamber in a case of aniridia



(C) GORETEX cover of tube



(D) Scleral tunnel for tube



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TECHNOLOGY UPDATE - ORA

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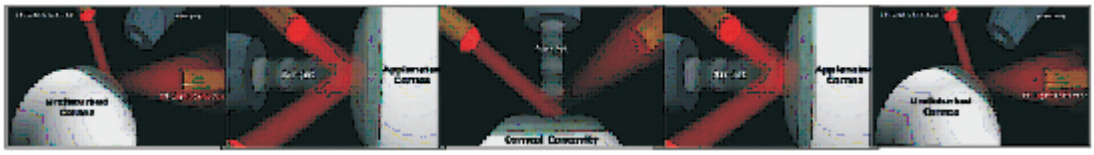
MBBS, DOMS
Eyelight Laser & Eyecare Pvt. Ltd., Pimpri, Pune

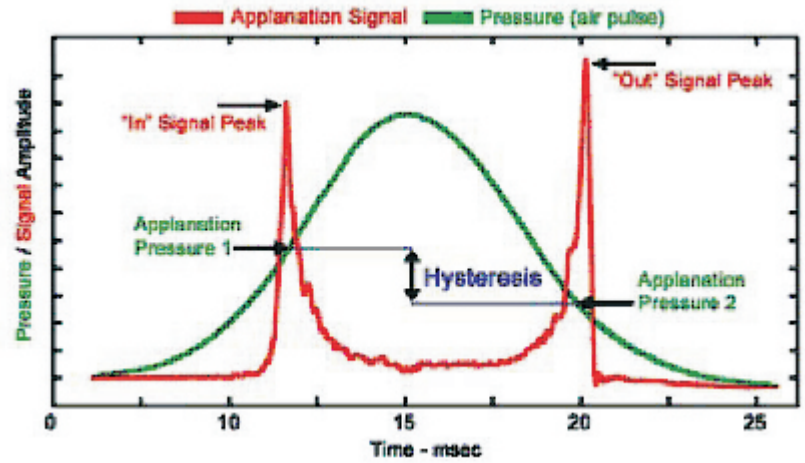
PRICIPAL OF ORA

Corneal biomechanical properties influence the results and outcomes of ocular measurements and procedures, and may hold clues to diagnosing and managing ocular diseases. Until now, assessing the biomechanical properties of corneal tissue has not been possible, confining practitioners and researchers to measuring purely geometrical aspects of the cornea, such as thickness and topography.

The Ocular Response Analyzer utilizes a rapid air impulse, and an advanced electro-optical system to record two applanation pressure measurements; one while the cornea is moving inward, and the other as the cornea returns. Due to its biomechanical properties, the cornea resists the dynamic air puff causing delays in the inward and outward applanation events, resulting in two different pressure values.



The average of these two pressure values provides a repeatable, Goldmann-correlated IOP measurement (**IOPG**). The difference between these two pressure values is Corneal Hysteresis (**CH**); a new measurement of corneal tissue properties that is a result of viscous damping in the corneal tissue. The ability to measure this effect is the key to understanding the biomechanical properties of the cornea.



The **CH** measurement also provides a basis for two additional new parameters :

Corneal-Compensated Intraocular Pressure (**IOPCC**) and Corneal Resistance Factor (**CRF**). **IOPCC** is an Intraocular Pressure measurement that is less affected by corneal properties than other methods of tonometry, such as Goldmann (GAT). **CRF** appears to be an indicator of the overall "resistance" of the cornea.

DEFINITION OF TERMS USED IN ORA ANALYSIS

IOPg -

IOPg is a Goldmann-correlated pressure measurement derived from the Ocular Response Analyzer's dynamic bi-directional applanation measurement process. It has been clinically shown to have strong correlation with expertly executed Goldmann applanation pressure measurements.

IOPcc -

IOPcc (corneal compensated intraocular pressure) is a pressure measurement that is less effected by corneal properties than other methods of tonometry. It compensates for the biomechanical properties of the cornea, not just the thickness. This is an important distinction because "correcting IOP" measurements by using CCT data may result in significant errors in magnitude and direction of the adjusted IOP value. Thickness is not the same thing as resistance.

IOPcc was developed using pre and post lasik clinical data. Since the inward and outward applanation events measured by the Ocular Response Analyzer provide independent information about corneal tissue properties, it is possible to mathematically 'manipulate' these values to obtain an IOP measurement that is less influenced by the cornea.

IOPcc is still strongly correlated with Goldmann-obtained IOP measurements, but has no correlation with CCT and does not change significantly post-lasik.

CRF (Corneal Resistance Factor) -

CRF is a measurement of the cumulative effects of both the viscous and elastic resistance encountered by the air jet while deforming the

corneal surface. CRF exhibits the expected property of increasing at significantly elevated pressures. Though CH and CRF are, on average, the same for a normal population, they differ from person to person, providing us with distinct corneal information.

Corneal Hysteresis (CH) -

Corneal Hysteresis is simply the raw difference between the inward and outward applanation pressure values. CRF uses these same two data points but employs a specific algorithm to arrive at the CRF. This equation was developed by maximizing the correlation of P1-(k*P2) with CCT.

Range of Corneal Hysteresis in normal subjects -

Clinical data from numerous studies suggest that the typical range of Corneal Hysteresis (CH) in normal subjects is approximately 8 - 16 mmHg. However, it is very likely that CH values will vary depending on age and race. We have done the data for Indian (Western Maharashtra) population of 500 individuals, where the average CH values recorded is 10.1$\pm$ 1.1.

Range of Corneal resistance factor in normal subjects -

CRF is "calibrated" to match CH in normal subjects. Therefore, the typical range for CRF is approximately the same as the range for CH.

USE OF ORA IN GLAUCOMA & CORNEAL DISEASES :

Corneal Disease

Measuring the biomechanical properties of the cornea with the Ocular Response Analyzer enables researchers and practitioners, for the first time, to quantify various corneal conditions by means of a measurable and repeatable metric.

Low Corneal Hysteresis (**CH**) demonstrates that the cornea is less capable of absorbing (damping) the energy of the air pulse. The differences in **CH** between normal and compromised corneas are highly evident, and lead some experts to theorize that normal eyes exhibiting significantly lower than average **CH** may be at risk of developing corneal disorders in the future like Ectasia, keratoconus or Fuchs dystrophy.

Refractive Surgery -

Clinical data from several studies show a universal reduction in post-LASIK **CH**. Some experts hypothesize that this is not primarily a function of corneal thinning, but rather a result of weakening of the structure related to creation of the flap. The Ocular Reponse Analyzer's ability to characterize the biomechanical properties of the cornea means that potential refractive surgery candidates can be more effectively evaluated for potential post-surgical complications, such as corneal ectasia, than by using **CCT** and topography alone.

Glaucoma -

The Ocular Hypertension Treatment Study (OHTS), as well as other studies, have brought to light the importance of corneal parameters in diagnosing and managing glaucoma. These studies have shown that low **CCT** (thin cornea) is an independent risk factor for the development and progression of the disease. Many experts believe that corneal parameters other than **CCT** may provide clues that will aid in the diagnosis and management of glaucoma. There is evidence to suggest that the cornea may reflect the condition of the lamina cribrosa. Clinical studies utilizing the Ocular Response Analyzer support this hypothesis and have confirmed that low **CH** is an independent indicator of glaucomatous damage and progression.

It has also been demonstrated that lower-than-average **CH** is observed in subjects who have been identified as "Normal Tension Glaucoma" (NTG) patients. Currently, individuals who have NTG may be missed during routine IOP screening. If the **CH** parameter proves to be a reliable indicator of this condition, it would be a significant advance in glaucoma screening.

Accurate Pressure Measurements :

Evidence in ophthalmic literature continues to mount, leading experts to question whether the Goldmann tonometer is sensitive and specific enough to be used for the critical purpose of measuring IOP in the diagnosis and management of Glaucoma.

The Goldmann tonometer is designed to provide accurate measurements in eyes having average central corneal thickness (**CCT**). Corneal thickness, though, varies significantly more than previously thought. Furthermore, it is now known that the material properties of the cornea have an even greater influence on tonometry results than **CCT** alone. The Ocular Response Analyzer measures these complex tissue parameters, providing a pressure measurement (**IOPCC**) that is less influenced by corneal properties. Since **IOPCC** compensates for corneal influence, it facilitates post-LASIK pressure measurements that are not artificially lower than pre-LASIK values. Goldmann-measured IOP values are known to drop 2-6 mmHg, or more, post LASIK.

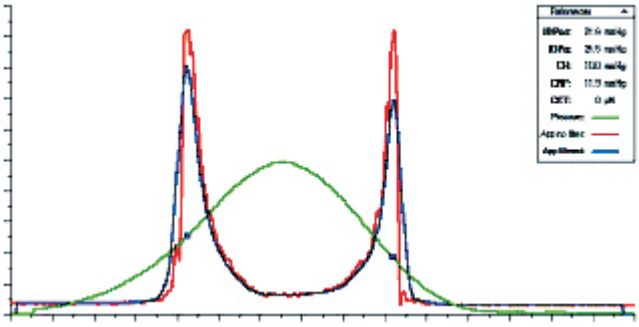
IOPCC values for a population of 54 eyes pre and post LASIK exhibit an average post-LASIK **IOPCC** reduction of less than 1 mmHg.

In addition, some investigators believe that Goldmann tonometry underestimates the true pressure in "Normal Tension Glaucoma" eyes. In a population of 24 NTG eyes, **IOPCC** is more than 2.25 mmHg higher, on average, than **IOPG**; a significant difference when diagnosing and managing glaucoma.

ORA SIGNAL ANALYSIS

The ORA optical system records 400 data samples of reflected IR light intensity during the rapid (30 ms) in/out corneal deformation.

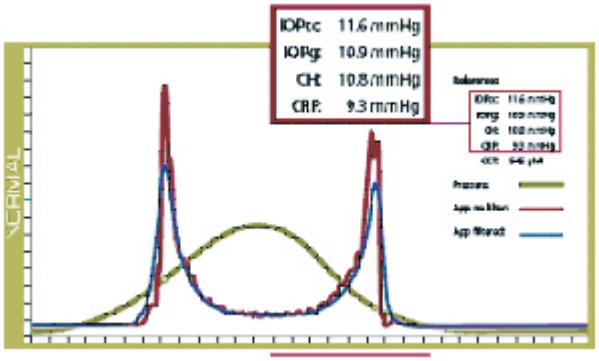
The optical signal (red curve) is a "dynamic map" of the cornea during the rapid in/out deformation.



- Peak amplitude
- Peak width
- Peak aspect ratio
- Peak up/down slope
- signal high-frequency content (noise)
- Signal repeatability

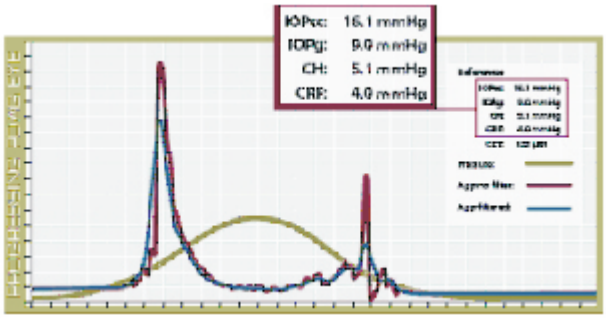
In a normal signal we have to Watch for :

- Clean, smooth signals
- Similar amplitude peaks
- Repeatable values
- Consistent measurements in both eyes

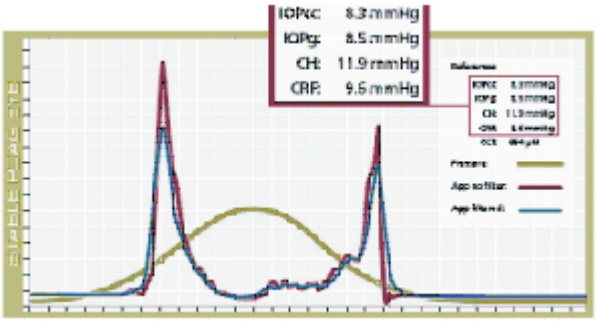


IOPcc and IOPg are close and in normal range, CH and CRF are close and in normal range

In a post lasik or post PRK the IOPcc is higher than IOPg but is in normal range and CH ans CRF is lower than normal. In POAG IOPcc and IOPg both are elevated and CH is lower than normal and lower than CRF

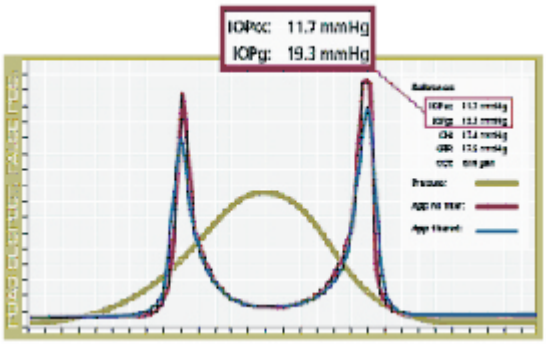


but if the POAG is on treatment and still progressing the IOPcc will be higher than IOPg and CH and CRF both will be lower than normal.



But in POAG stable on medicines will not show much difference in both IOPcc and IOPg and CH and CRF.

In a false positive OHT patient IOPg will be much higher than IOPcc and CH and CRF will be higher than normal. As on the other side in a NTG patient the IOPcc will be higher than IOPg and CH and CRF will be lower than normal and CCT will be on thinner side.



SUMMARY

The dynamic bi-directional applanation process employed in the Reichert Ocular Response Analyzer facilitates the measurement of Corneal Hysteresis (CH).

The CH phenomenon is observable due to viscous damping in the cornea and permits the calculation of Corneal Resistance Factor (CRF) which appears to reflect the overall resistance of the cornea. Both metrics are new measurements of the biomechanical properties of the cornea.

The ability of the device to characterize the biomechanical properties of the cornea enables the calculation of IOPCC, a measure of Intraocular Pressure that is less influenced by corneal properties than Goldmann or other currently available tonometers.

IOPCC is unaffected by corneal properties such as CCT and remains essentially unchanged after LASIK.

Published and preliminary results from clinical studies in process worldwide suggest these new parameters may be clinically useful in a number of different areas including, but not limited to: Identification of corneal diseases such as keratoconus and Fuchs' Dystrophy, Glaucoma diagnosis and management, screening potential LASIK candidates, and accurate IOP measurement (by excluding the effect of corneal factors in IOP measurement).

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3 Felipe A. Medeiros, MD and Robert N. Weinreb, MD. Evaluation of the Influence of Corneal Biomechanical Properties on Intraocular Pressure Measurements Using the Ocular Response Analyzer. 2006;15:364-370

4 The use of the Ocular Response Analyser to determine corneal hysteresis in eyes before and after excimer laser refractive surgery
Sunil Shah a,b,c, Mohammad Laiquzzaman a,*, Ian Yeung c, Xueliang Pan d, Cynthia Roberts e
a Heart of England Foundation Trust, Solihull, UK
b Ophthalmic Research Group, Aston University, Birmingham, UK
c Midland Eye Institute, Birmingham, UK
d Department of Statistics, The Ohio State University, Columbus, OH, USA
e Department of Ophthalmology and Department of Biomedical Engineering, The Ohio State University, Columbus, OH, USA

5 Comparison of corneal biomechanics in pre and post-refractive surgery and keratoconic eyes by Ocular Response Analyser. Sunil Shah a,b,c,1,*, Mohammed Laiquzzaman a,1

6 Factors affecting corneal hysteresis in normal eyes. Kazutaka Kamiya & Mana Hagishima & Fusako Fujimura & Kimiya Shimizu
Graefes Arch Clin Exp Ophthalmol (2008) 246:1491–1494

OLOGEN IMPLANT

Ologen is a new product that modulates wound healing in glaucoma surgery.

Steven R. Sarkisian
JR, MD

For many years now, ophthalmologists have used antimetabolites such as mitomycin C (MMC) and 5-fluorouracil to modulate wound healing in glaucoma filtering surgery. These agents, however, can cause many complications, including hypotony, wound leaks, and endophthalmiti. A new collagen matrix implant for wound modulation may reduce these problems.

BACKGROUND

Surgeons in Asia and Europe have used collagen matrix for several years. In the United States, Canada, Central and South America, and South Africa, the product is sold as Ologen Collagen Matrix Implant (Optous, Roseville, CA), which was approved by the FDA in August 2009. I have had the opportunity to use the implant for the past several months in patients of various ages and races, mostly with primary open-angle glaucoma. This artificial porcine extracellular matrix is made of atelocollagen cross-linked with glycosaminoglycan. Ologen is a biodegradable scaffolding matrix that induces a regenerative wound healing process without the need for antifibrotic agents. The surgeon places the device over the scleral flap during the filtering procedure.

It is well known that episcleral fibrosis and subconjunctival scarring are the major causes of failure in glaucoma filtering surgery. Collagen matrix is designed to prevent such scarring. Specifically configured to facilitate the repair of connective and epithelial ocular tissue, the implant is designed to minimize the random growth of fibroblasts and instead promote their growth through the pores in the matrix. Ologen is biodegradable in 90 to 180 days.

The efficacy of collagen matrix has been demonstrated in animal models.[1-3] Chen and colleagues performed standard trabeculectomy on 17 rabbits, with their left eye receiving the collagen matrix implant and their right eye serving as surgical controls. During the first few days, the postoperative reduction in IOP (15%) was equal in both groups. Pressure had decreased to 55% below baseline values at day 28 in the treated eyes but had returned to preoperative levels by day 21 in the control eyes. Histological examination showed a prominent bleb in the treated eyes compared with scarring and limited bleb formation in the control eyes.[2]

TECHNIQUE

Guarded filtering surgery is performed based on the surgeon's preferred technique. One can make either a limbus- or a fornix-based conjunctival incision. The main surgical change is in the closure of the scleral flap. With trabeculectomy, many ophthalmologists prefer to place several tight sutures to prevent early hypotony. With Ologen in place, it is better to tie the sutures loosely in order to encourage aqueous flow.

After tying the sutures, the surgeon places the collagen matrix over the scleral flap (Figure 1) No suture is required to secure the implant, and as soon as it touches the sclera, it absorbs aqueous and molds to the scleral tissue. Collagen matrix therefore need not be presoaked or prepared in any way.

Ologen currently comes in two sizes for glaucoma filtering surgery: 6 X 2 mm and 12 X 1 mm. The numbers 6 and 12 refer to the diameter of the round implant, and the numbers

2 and 1 refer to its thickness. I have used both sizes of the device with good success. I have been able to place the larger version through a 4-mm limbal incision with my fornix-based conjunctival wounds. The process does require some manipulation, however, and I have had to fold the implant slightly for insertion and then tease it flat (Figure 1). In my experience, the 6 X 2-mm device is much easier to place over the scleral flap with a small limbal incision because of the implant's greater thickness and smaller diameter, but it can be more difficult than with the larger implant to visualize the sutures for laser suture lysis during the postoperative period. With a limbus-based conjunctival flap, I have found that no manipulation is required at all because of the large exposure afforded by the larger posterior wound in the fornix. Both sizes of the implant can be used with either type of conjunctival wound, and the surgeon's comfort and experience over time will help dictate his or her preferred size.

After the collagen matrix's placement, the surgeon closes the conjunctiva in his or her usual meticulous fashion to ensure that the wound is watertight.

POSTOPERATIVE COURSE AND APPEARANCE OF THE BLEB

I am waiting until I have longer follow-up data before requesting my institutional review board's permission to research my results in a systematic fashion. I can, however, report that the blebs are not avascular in the eyes in which I have implanted the collagen matrix, even 4 to 6 months postoperatively (Figures 2 and 3). Although the period of observation is short, more than 100 patients of mine who have received the collagen matrix prior to the writing of this article have maintained low IOPs without medication, just like my patients who have received intraoperative MMC.

During my first several cases, I was concerned that I would not be able to visualize the sutures through the collagen matrix, so I placed the collagen matrix on the posterior edge of the flap. Since then, I have found that I can see the sutures through the implant if I press firmly with a Blumenthal Suturelysis lens (Volk Optical, Inc., Mentor, OH). I now therefore place the collagen matrix directly over the flap, which probably allows for better wound modulation. Moreover, the collagen matrix helps to limit hypotony through a tamponading effect over the scleral flap. I have observed the presence of the collagen matrix under the conjunctiva even 5 months after surgery, but the implant does thin as it biodegrades (Figure 4).

ADVANTAGES

Currently, Ologen retails for $250 a unit, but with an order of 10 or more devices, the company reduces the price to $200 per unit. Although collagen matrix is more expensive than MMC (by $100 or more), I believe that the former offers several advantages.

First, not using antimetabolites saves a significant amount of time intraoperatively. In my experience, each case is at least 5 minutes shorter, and the nurses do not have to take time for the special handling and disposing of an antimetabolite. On a high-volume surgical day, the time saved with collagen matrix can allow me to perform more surgery (one or two cases) during my allotted time in the OR. If I do not have additional cases, the ASC saves money by being able to send the nursing staff home early. The cost of health care is a pressing issue and demands thoughtful analysis. For example, in a retrospective, consecutive, comparative case series, investigators found that using fibrin glue to secure the patch graft and to close the conjunctiva after glaucoma drainage device surgery saved 10 minutes compared with the use of sutures. They calculated a cost savings of over $389 for every case.4 Using their calculations, I estimate that reducing surgical time by 5 minutes by using collagen matrix at the same institution would save over $100 a case, when the cost of the collagen matrix and the need for no MMC are taken into account.

Second, unlike with antimetabolites, collagen matrix need not be special ordered, and there is no risk of a shortage, as recently occurred with MMC. Instead, collagen matrix can be ordered like any other implant, and its shelf life is 2 to 3 years.

Third, because the collagen matrix is not a teratogen like MMC, the former may be used for pregnant patients, and a pregnant member of the OR staff will not have to excuse herself during the procedure. The practice at my ASC has been not to schedule pregnant staff members with glaucoma surgeons, so I lost one of my most experienced scrub technicians for 9 months last year.

DISADVANTAGES

To date, even with the Blumenthal Suturelysis lens, laser suture lysis has been difficult in a few cases. I was unable to visualize any sutures in one eye with a Tenon's cyst, and I needed a 27-gauge needle to break what sutures I could without no visibility of the flap. After the needling and digital pressure, the IOP decreased nicely. I have switched to using the 12 X 1-mm implant, which seems to have reduced the problem. The thinner implant can be more difficult to place than the 2 X 6-mm collagen matrix, however, and careful dissection of the posterior space under Tenon's capsule is necessary.

I do not recall any other postoperative problems in my patients who received collagen matrix. I was concerned at first, however, that tying the sutures more loosely after trabeculectomy would lead to a higher incidence of hypotony. Thankfully, this has not been the case.

The cost of the collagen matrix may be an issue for some surgeons, regardless of the possible cost savings of a more efficient OR day.

CONCLUSION

Driven largely by surgeons' desire to lower IOP more safely and efficiently than by standard trabeculectomy with MMC, an exciting period of innovation in glaucoma surgery is underway. The use of collagen matrix in glaucoma filtering surgery is the most recent such development. I have found this device to be safe and effective in the short term, and it may help to reduce costs, improve efficiency, and increase surgical volume. Clearly, a prospective, randomized trial comparing MMC with collagen matrix is warranted. If time and further study demonstrate that the device offers improved safety (ie, fewer cases of bleb leaks and endophthalmitis) compared with antimetabolites, it is possible that glaucoma surgeons will come to rely on collagen matrix for wound modulation after filtering surgery.

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GRAND ROUNDS IN GLAUCOMA

Proof of efficiently managing patients is the application of knowledge in treating an individual patient. In this special feature six emminent Glaucoma specialist from the city discuss management of 5 cases. The panellist are Dr. Medha Prabhudesai (MP), Dr. Sagarika Patiyal (SP), Dr. Vidya Cherelkar (VC), Dr. Tejaswini Walimbe (TW), Dr. Roopali Nerlikar (RN) & Dr. Shraddha Satav (SS).

The cases have been contributed by Dr. Medha Prabhudesai & Dr. Mandar Paranjpe.

CASE 1 CASE SUMMARY -

34 yrs, Male, came in February 2009, with H/O DOV, OS,of 1 month duration. He is a high Myope, with no H/O any Systemic diseases.

O/O/E: OD 6/9 N8 [-12 /-1.00@180]

OS 6/24 PH 6/12 N8 [-13/-0.50@180]

SLE ; OU Cornea Clear

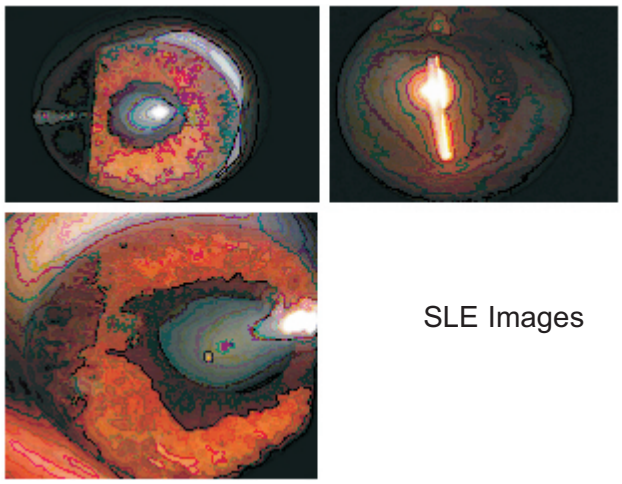
Megalocornea

AC deep

AT 14 /28 mm Hg

Gonio scopy– open angles

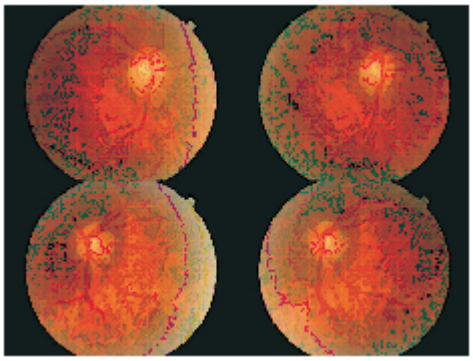
CCT -619/620 microns



SLE Images

Gonioscopy Image

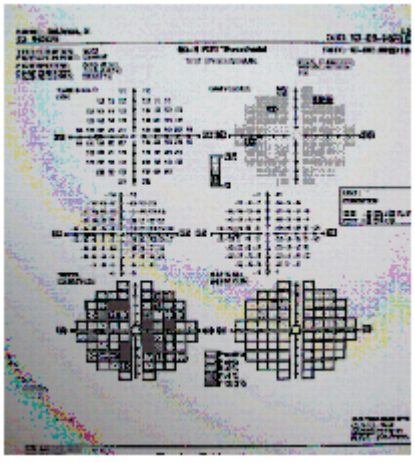
Stereo Disc Images

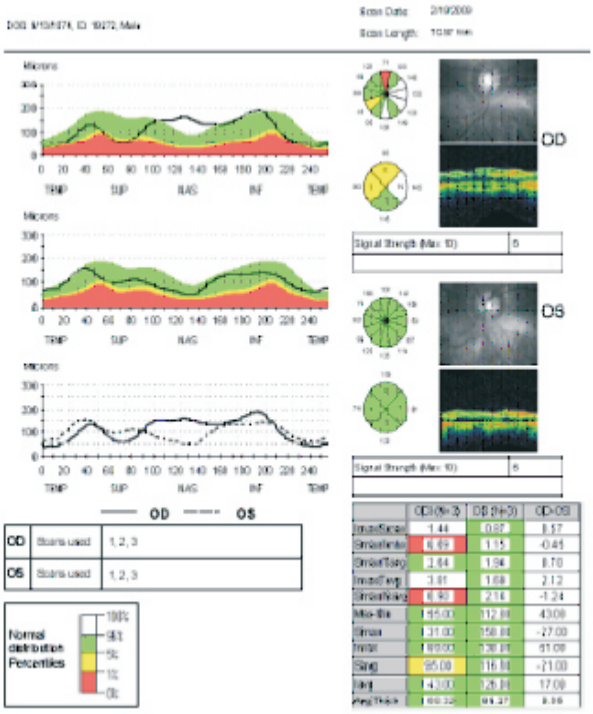


Perimetry (Feb 2009)

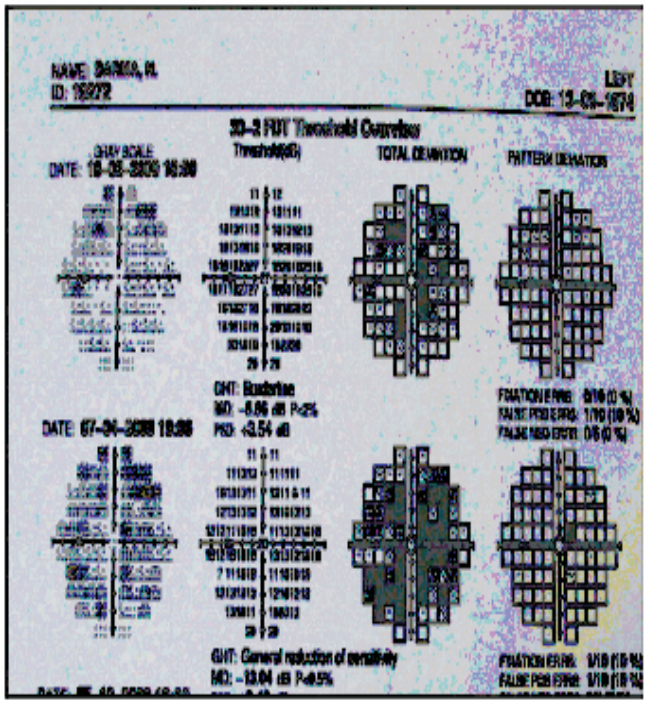
RIIGHT EYE

LEFT EYE





OCT Image February 2009



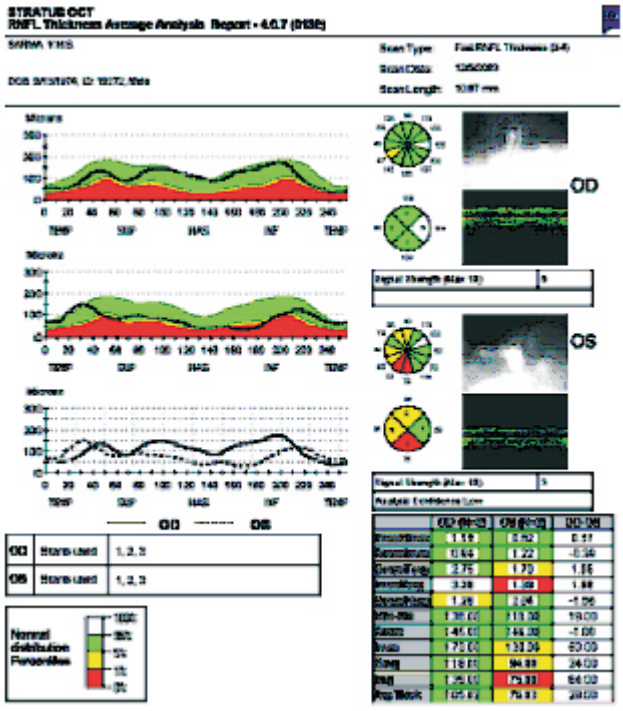
Automated Perimetry April 2009, OS

Treatment started : OS- Travoprost Eye drops
IOP 2 weeks post treatment was 16 mm hg.
APRIL 2009 : BCVA-OD 6/9; OS-6/36.

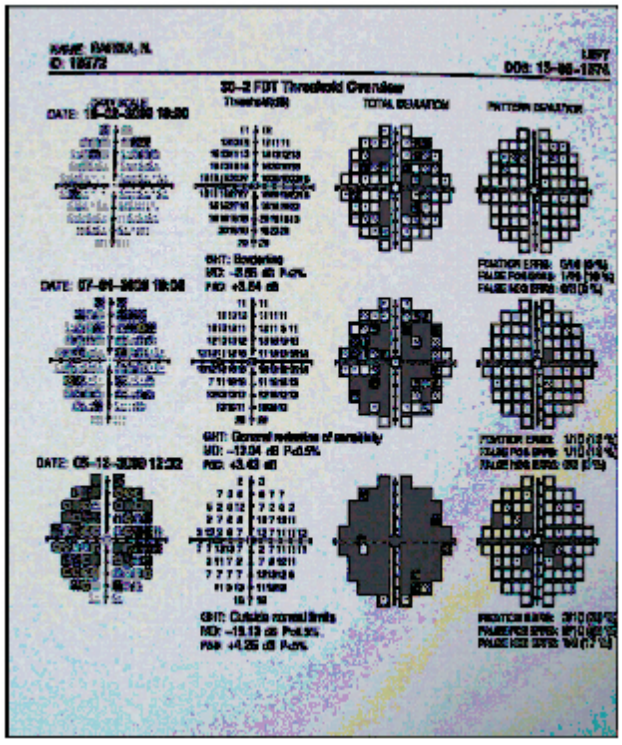
OS Myopia increased from -12 D to -16 D
AT 13/14 mmHg
ONH stable

December 2009 : BCVA : OD-6/9, N6 (-16 D), OS-6/36, N36 (-31 D)

AT 14/14 mm Hg



AP December 2009, OS



OCT December 2009

Questions to be answered are

* What is your diagnosis?
* What do think about the situation & status of OS?
* What could be the reasons for increase in Myopia?
* What should be the management of this patient?
* Any other comments.

Replies from faculty :

VC : My first impressions are, this could be ICE Syndrome or an Axenfeld Reigers Syndrome.

Points in favour of ICE syndrome, age at presenatation, iris atrophy, mild corectopia especially in the Left eye, peripheral iris holes and ectropion uveae.

Points against : Bilaterality, clear cornea, CCT?

ICE syndrome is a primary corneal abnormality, variably associated with corneal edema, anterior chamber angle abormalities and glaucoma. It has its onset in young aulthood, is unilateral, absence of family history.

In this case, the cornea is clear, and there is associated megalocornea. Specular microscopy may help in knowing corneal endothelial status, if there is any cellular pleomorphism. This might also be the reason for a high CCT.

A-R syndrome : has striking clinical and histopathological similarity to ICE syndrome. However, it is congenital, bilateral and has autosomal dominant inheritance. The cornea is clear except fo the peripheral posterior embyotoxon. Megalocornea has been mentioned in some variants of this disease. Typically, the corneal endothelium is normal.

This patient has features resembling both the conditions. In fact it has been suggested that the two syndromes are parts of a commom spectrum of disorders both coditions are characterized histopathologically by a membrane over the angle and iris, which is associated with many of the abnormalities seen.

Myopia has not been mentioned as an association in either of the conitions.

Treatment is aimed at control of glaucoma (medical and later surgical).

TW : My diagnosis is Pigmentary Glaucoma. Points supporting are- young myopic male, classical transilluminatin defects, pigment dispersion on lens surface, open angle with back bowed iris trabecular pigment & iris atrophy with ectropion uveae.

Ponts not in favour are, relatively unusual presentation of unilateral findings in pigmentary glaucoma (as seen in this case)

My D/D will be ICE syndrome, where there is progressive or essential iris atrophy & the angle will close progressively.

An UBM can be done to demonstrate reverse papillary block, posterior insertion & back bowing of iris.

Looking at the all the investigations, patient seems stable with present management i.e glaucoma is well controlled.

The reason for increase in myopia could be nuclear sclerosis or rarely posterior staphyloma (a B scan might give a better idea).

As far as treatment is concerned, any of the anti-glaucoma drugs can be prescribed except Pilocar, which is contraindicated in this patient because of high myopia. As Glaucoma is stable in this patient, we can go ahead with only cataract surgery & follow up with anti-glaucoma treatment. In general pigmentary glaucoma Selective Laser Trabeculoplasty has good results. A LASER PI can also be tried to remove reverse papillary block. Trabeculectomy (if required) has satisfactory results inthese patients.

SP : This is a case of Axenfield Reiger's syndrome. The cause of sudden increase in myopia is unusual and could have been caused by formation of staphyloma which is unlikely to occur so fast. The other cause of this unusual finding could be lenticular opacification. Since discs and fields are normal in both eyes but IOP is raised in the left eye a prostaglandin could be added to reduce the IOP to below 18 mmHg.

RN : Diagnosis: Axenfield Rieger anomaly (B/L, ectropion uveae, iris atrophy, post embryotoxon)-syndrome if jaw/dental abnormalities exist. I feel Gonio would show some synechiae..

Left : lenticular myopia- also responsible for poor quality on OCT and apparent worsening-not matched on FDT which shows gen depression.

As IOP controlled with single medication and glaucoma is in early stage, temporal clear corneal phacoemulsifcation LE first with IOP monitoring and continued Antiglaucoma meds (Diamox cover for 1 week postop till travoprost can be resumed)

One can repeat OCT and FDT thereafter.

SS : Did he develop a cataract? The only thing done is addition of the prostaglandin – did this cause the myopia. We can try changing the drops to Timolol or Brimonidine. Also the OCT seems worse after the Rx, so that is the likely cause. Frankly this case is very puzzling.

MP : This case was totally unusual. My diagnosis was Axenfeld Riegers with component of pigmentary glaucoma. The increase in myopia is due to development of dense nuclear sclerosis only in the centre [3 to 4 mm], which is falsely seen as progressive thinning of RNFL in OCT because of low signal strength [3out of 10}, and also matrix perimetry overview printout.

CASE 2 CLINCAL DATA :

33 YRS. Female, had past h/o Glaucoma, OD 5 years ago. Her IOP recorded was 30 mm Hg. She does not have any systemic ailments.

O/O/E: OD- CF 2mts, PH no improvement, N36 ; OS-6/9,p; PH-no improvement, N6

She is on anti-glaucoma treatment (Timolol, Timolol+ Bimatoprost, Bimatoprost, Travoprost, individually or in combination for variable period)

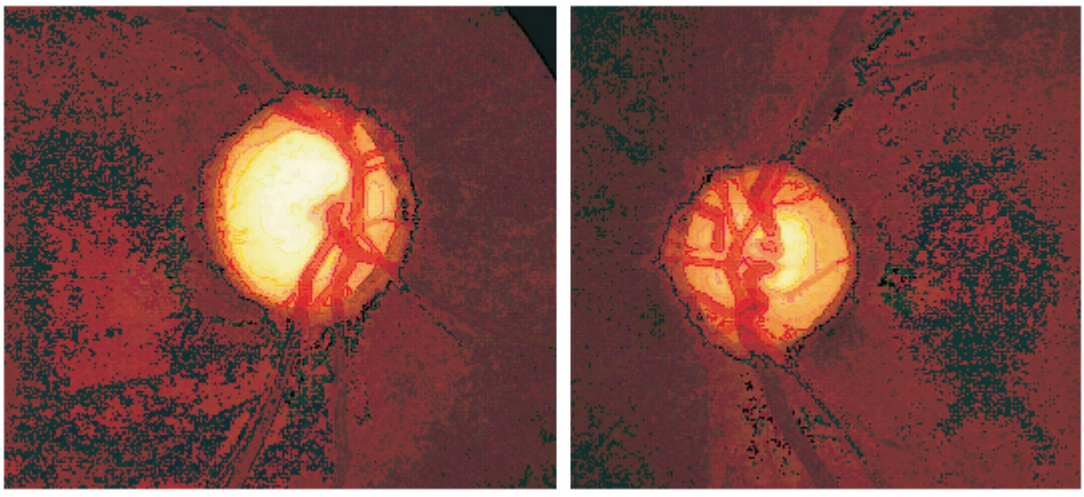
SLE; OU- Clear Cornea, Deep A, AT- 10/10 mm Hg (OU on Betoxolol & Travoprost)

Gonio : OU Wide open angles.

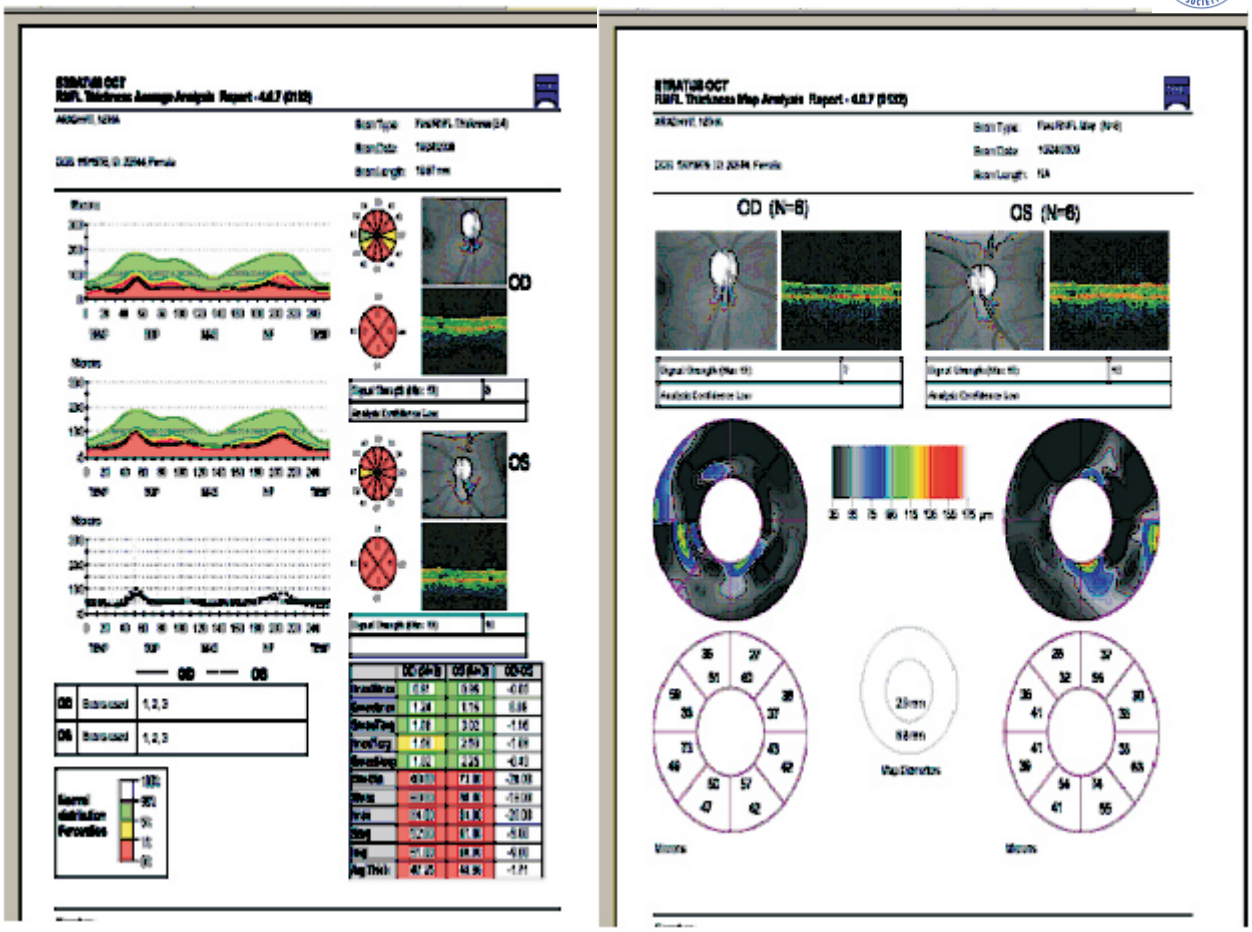
CCT- 559/572 microns

A Perimetry- OD was not possible, OS was Normal.

ONH image



OCT Report



Question to be answered are -

* Are any further investigations required?
* What should the management of this patient be?
* Any other comments.

SP : This is a young patient who has unilateral glaucoma which maybe secondary as gonioscopy does not show any features of JOAG. History of steroid use in affected eye and history of trauma are to be asked of this patient being among the commoner causes of sec unilateral glaucoma. The medication need not be changed as IOP control is good.

VC : She is a young patient with unilateral glaucoma. History is not very elusive. One needs to rule out history of trauma or any other secondary cause. Also family history of glaucoma is needs to be ascertained..

My impression -

1. Primary open angle glaucoma, more advanced in RE.
2. Unilateral secondary glaucoma RE

First, in history, we may need to know about any trauma as said earlier,and any steroid usage. The angle is open which does not rule out trauma. Also, there are no signs of allergy in the eye or any systemic disease requiring steroid medications.

However, the fundus examination shows advanced disc damage in the RE, the LE also shows 0.6 -0.7 CDR with a suspicious superior notching and dilatation of the vessels. There is a decrease in the RNFL thickness on OCT in BE. Perimetry in the LE was normal at this point of time. The CCT is WNL, and the IOP in the LE is 10 without Tt.

I feel, as the RE has advanced damage, we need to watch the LE carefully. The disc and OCT arouse a suspicion of early damage, not yet picked up by peimetry. Even if the IOP is WNL now, a diurnal variation of IOP might be done. Field examined at regular intervals should be done to pick up any damage. SITA Std as well as SITA SWAP strategies can be employed for this.

RE can be continued with the same 2 medications which are keeping the IOP at 10.

TW : Young female 33 yr, unilateral optic nerve changes ,vision CF 2 ft in RE suggest possibility of orbital or neurological pathology. (pupillary reaction and colour vision necessary in clinical exam).

Investigations required :MRI (brain + orbit) with contrast (to rule out suspect neurological problem) & Carotid Doppler study to rule out right carotid artery stenosis

Management will depend on the reports of the above investigations.

If the above investigations are normal and no specific cause for less vision in Right eye ,then continue with antiglaucoma treatment. this patient is receiving travoprost + Betoxolol ,IOP is 10 mm of hg which is quite satisfactory and keep follow up ,observe for progression

RN : I would assess for associated systemic risk factors including hypotension and Raynauds.

Diurnal IOP would help. Despite presence of diffuse NFL thinning on OCT LE red free and perimetry not correlating which I would repeat.A Neuro evaluation is necessary.

Monitor FDT and OCT 4monthly and continue topical medication.

SS : In this young female there is unilateral low vision and cupped out disc. OCT is similar in both eyes. Was she ever on steroids ? Has the IOP in the left eye ever been high? Maybe we can stop the drugs one by one to see if they are really needed. This is looking more like unilateral glaucoma (since we've ruled out PACG). If the fields stay normal in the left eye, maybe there is no glaucoma. Right eye can be followed by serial OCT & fields, for progression.

MP : There is no correlation between OCT findings and perimetry findings in left eye. Hence even if she had a history of high IOP recorded 5 years back we highly suspected that some other pathology was responsible. We stopped her antiglaucoma medication, first travaprost and after 15 days betoxolol. Her IOP remained 10 mm Hg in both the eyes. In between we investigated her.

MRI brain with contrast was normal. Also a neurologists opinion was sought who advised ANA antibodies which was positive and revealed high titre. Vitamin B12 serum levels were very low [67 pg/ml : normal levels are 219 – 911 pg/ml]. Based on these findings the neurologist started her on steroids 20mg /day and vitamin B12 supplements. After 20 days he re-evaluated and diagnosed her as having collagen disorder. He continued her systemic steroids and started her on immunosuppression. [T Azoran 50 mg OD]. Her IOP after 6 months is still 10 mm Hg in both the eyes with vision remaining same.

The diagnosis here can be neuromyelitis optica which is seen in patients with Multiple Sclerosis. These patients initially show normal MRI brain but MRI of optic nerve and spinal cord reveals demyelination.

MORE ABOUT COLLAGEN DISORDERS / CONNECTIVE TISSUE DISORDERS

Females are more likely to be affected than males. The prime age is 30-50 years but people of all ages may be affected. Most of the autoimmune connective tissue disorders affect multiple tissues, and the blood vessels are the organs most often affected. Many of the specific autoimmune connective disorders tend to overlap or appear in conjunction with other connective tissue disorders. The autoimmune connective tissue disorders may develop slowly over many years or they may present abruptly and show rapid progression, and they're typically characterized by alternating periods of remission and flares.

Read more at : Connective Tissue Disorders: Collagen Disorders and Their Causes http://autoimmunedisease.suite101.com/article.cfm/connective_tissue_disorders#ixzz0jOsUmkv0

CASE 3 CLINICAL DATA -

62 yrs. Male has H/O Glaucoma for past 12 years (highest IOP recorded elsewhere was 20.6mm Hg by ST). He has H/O Hypertenstion for past 5 years.

He was on following anti-glaucoma medications in Right eye- Pilocar2%, Latanoprost+Timolol (HS) & Dorzolamide (tid)

O/O/E- BCVA, OD-6/9, PH, NI, N6 (+1.00@170); OS- 6/9, PH 6/6,p, N6 (+1.00 DS)

SLE : OU : Clear Cornea, Deep AC

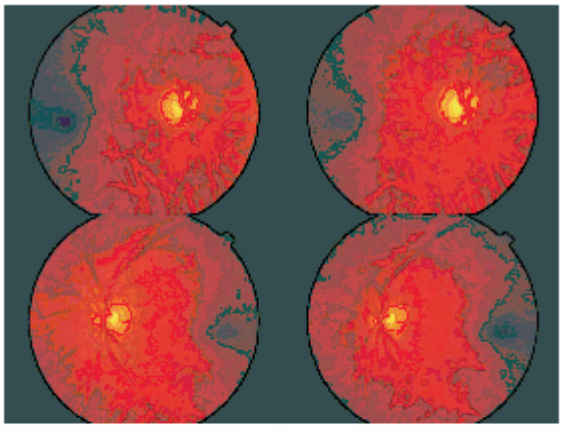
AT 16/15 mm Hg

Gonioscopy OU: 360 degree open

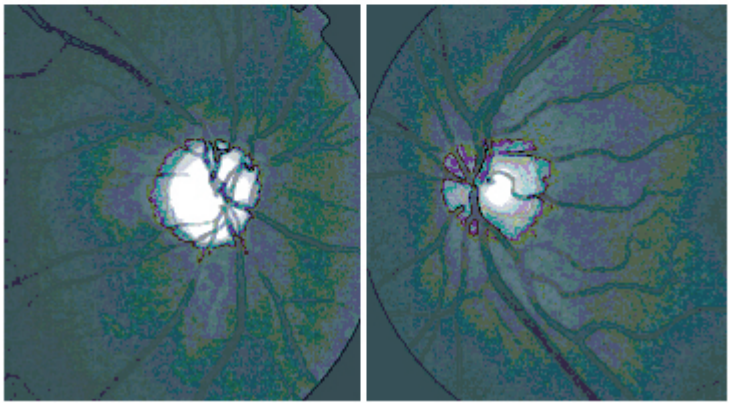
CCT : 489/ 510 microns

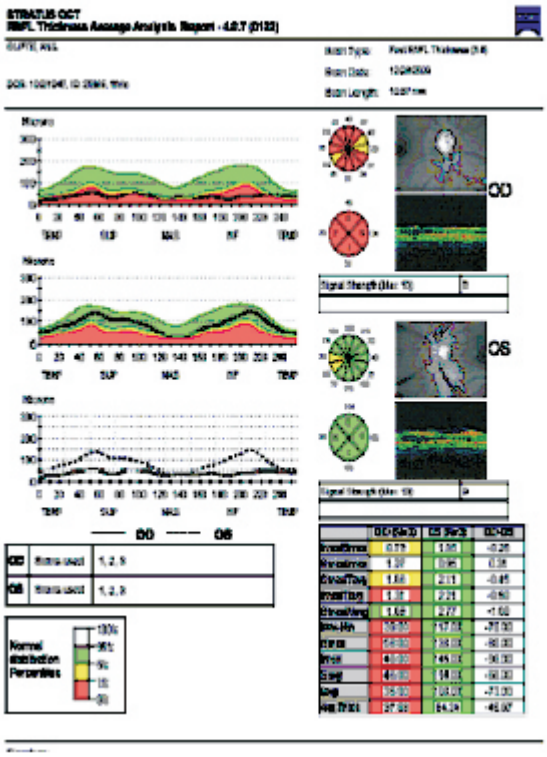
Perimetry : OD- Generalized depression, nasal paracentral defects ; OS-Normal

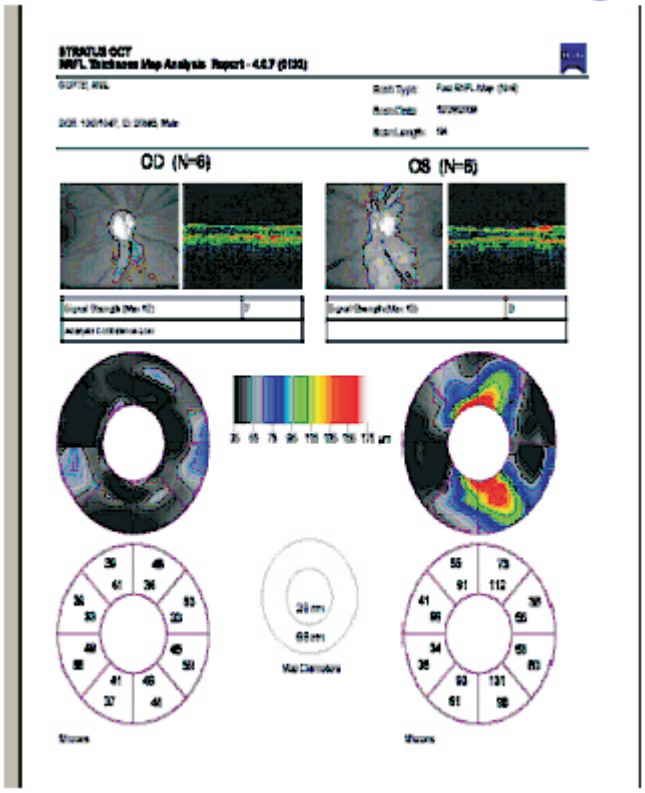
ONH Images



OCT







Questions to be answered

* Will you order any further investigations?
* Will you continue with all the three eye drops?
* Any other observations.

TW : This patient presented with low IOP because of less corneal thickness, so no need for investigations for NTG but considering age of patient (62 years) I will advise physician check up to rule out circulatory, cardiovascular and neurological problem.

We can not continue pilocar eye drops as 62 yr patient will likely to have nuclear sclerosis and pilocarpine will compromise his vision.

B-blocker can cause nocturnal hypotension so I prefer morning dose of timolol maleate and evening dose of latanoprost instead of latocom. Dorzolamide can be continued.

Target IOP for RE should be less than 14 mm hg considering low CCT, low IOP on presentation and OCT findings s/o NFL thinning in all quadrants. IOP of LE after treatment is 16 mm of hg which is satisfactory.

Diurnal variation will help to decide target IOP.

RN : Significant asymmetry in Cupping and NFL –OD worse with arteriolar narrowing more in RE- I would consider Carotid Doppler

I feel pilocar can be discontinued-even the Timolol part of latocom if patient is on oral beta blocker. Medication discontinued one at a time with reassessment after 10 days would like to have target IOP at <16 RE, <18 LE (CCT thinner side) and repeat OCT/ Perimetry after 4-6 months.

SS : Again unilat open angle Glaucoma ! No Rx for the left eye. Right eye – I'd stop the Pilo and use bromonidine tds. Considering the age, also get a cardiovascular evaluation done. Though the highest IOP is 20.6, it was Schiotz, this could still be NTG, especially with the pericentral field defects

and the poor response to 3 drug regimen. If the IOP doesn't come down or progression occurs at this level, surgery may need to be considered.

SP : In this patient I would like to do a diurnal control to see if IOP is under 24 hr control with the given drugs. I would also like to remove pilocarpine as he might require cataract surgery in the near future and this will hinder his surgical results. I would rather put him on alpha adrenergic drugs for further IOP reduction .

MP : As this patient presented with advanced unilateral glaucoma [loss of RNFL in all quadrants in right eye] we advised carotid Doppler and serum vitamin B 12 levels. Carotid Doppler was normal. Serum B12 very low [49pg/ml]. Pilocar was discontinued. Patient was shifted on Travatan and Dorzolamide. After 15 days IOP was 14 mm in both eyes. IOP was checked at different timings on different days which revealed variation of 2mm Hg [14 -16]. Physicaians opinion was sought for low B12 levels who started him on injectable supplements. Incidently he underwent systemic examination which revealed IHD and had to undergo coronary angioplasty.

CASE 4 CLINICAL DATA -

48 yrs. Male, came for regular checkup. He had undergone Cataract Surgery OD in 1982 & OS in 1976.

He was diagnosed to have in left eye glaucoma in the year 2001.

He has H/O DM & HTN for last 12 yrs.

He is on OU Combigan (bid) & Careprost (hs).

O/O/E : BCVA, OD- 6/18, PH 6/12,p, N12 (+6.00/-0.50@130), OS-6/9, PH NI, N6 (+9.50/-1.50@25)

SLE : OU -Clear Cornea, Deep AC. OU- Aphakia

OS- Small pupil, eccentric, inferotemporal PAS from papillary margin to cornea with cortical remnants. Iris atrophy.

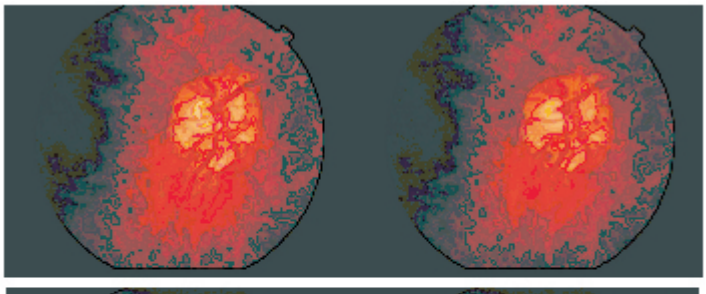
AT- 14-16/ 18-22 mm Hg (Initial IOP not known)

Gonio- OD – Open angles, Pigmented (+2 to +3), Iris processes + + : OS- Open only in nasal quadrant, rest chronic angle closure.

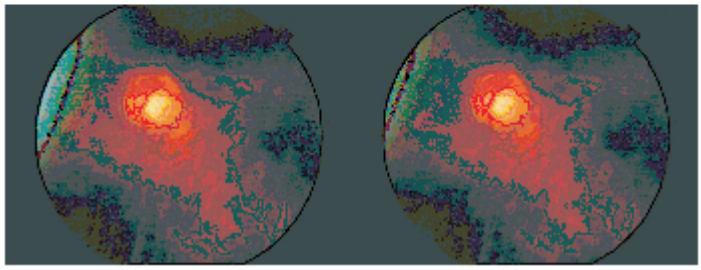
CCT : 602/ 586 microns

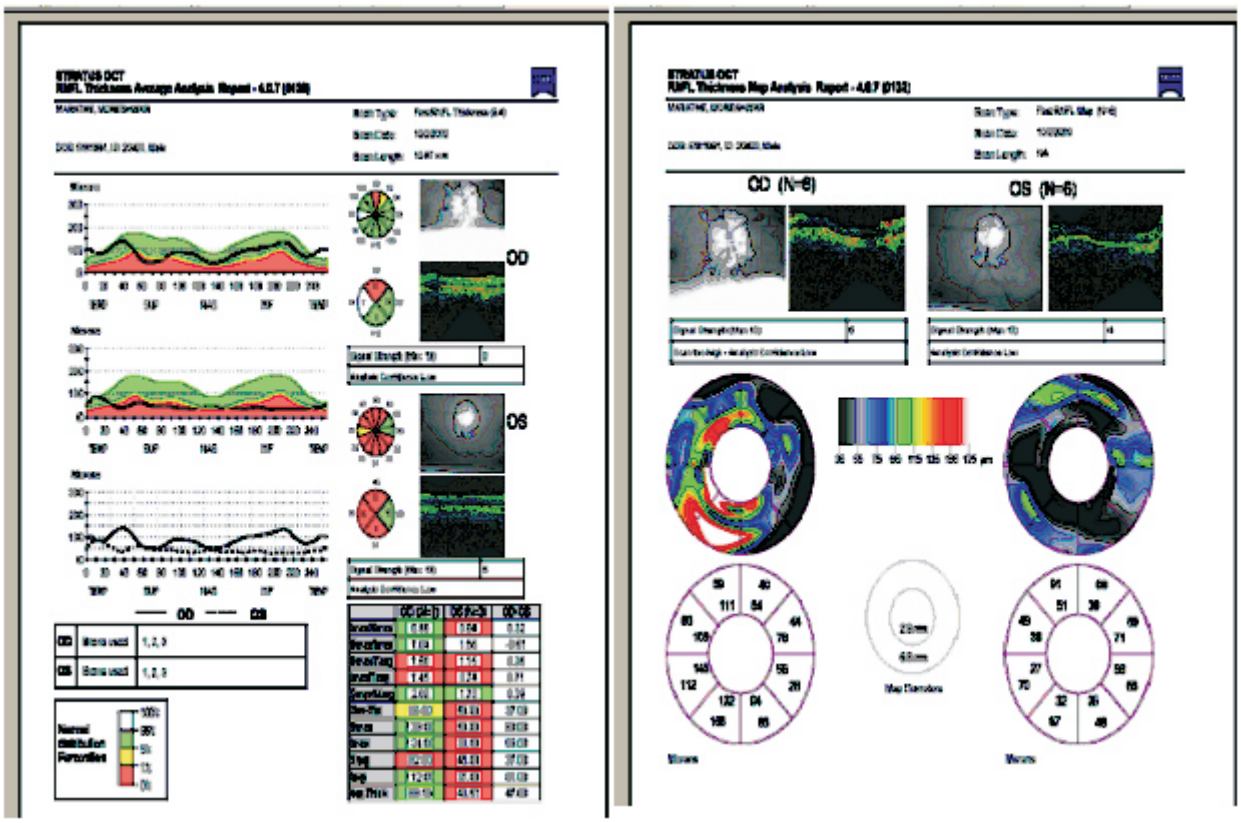
Perimetry : OD- Inferotemporal paracentral defects; OS- Central tubular field

ONH Phot



OCT





Questions to panellists -

* Can we continue with Careprost in Left eye?
* Is IOP reduction enough?
* Any other comments.

TW : Careprost should be avoided, reasons : Aphakia (prostaglandin can cause CME), Patient also has H/o DM for 12 years (hence risk of developing macular edema is high) & he has signs s/o past inflammation, PAS, remnant cortex (PG can aggravate inflammation)

IOP reduction in right eye-14-16 mm of hg is enough considering High CCT-(602microns), optic nerve and perimetry changes(inferotemporal paracentral scotoma), OCT s/o only sup.NFL thinning.

In LE 18-22 mm hg IOP is not enough considering tubular field, OCT s/o NFL thinning in 3 quadrants and optic nerve changes. Target IOP should be in the range of 16-18 mm of hg to reduce the rate of further progression.

SS : Aphakic glaucoma with myopia to complicate the picture! The left eye definitely needs a lower IOP – surgery may be good for this patient, may be even a shunt considering the aphakia. That would eliminate the need for Prsotaglandins too!

SP : A young male with bilateral aphakia, secondary angle closure left eye with advanced visual field defect and IOP ranging between 18-22 mmhg and advanced glaucomatous optic neuropathy with OCT changes corresponding to ONH changes definitely will benefit from further reduction of IOP. Careprost is not enough to reduce iop to target IOP levels which should be in lower teens. Addition of a prostaglandin would be helpful.

RN : In view of glaucomatous damage yes continue careprost-may omit if snellen vision drops or OCT shows macular edema. Inspite of thick cornea would prefer IOP to be lower maybe we can add Dorzolamide

VC : He is a middle aged man, BE aphakic with glaucomatous changes in BE, LE more advanced.

He is on 3 medications in BE. And IOP in LE seems to be a bit high.

He has both DM and systemic HTN.

He has an open angle with increased TMW pigment in the RE. LE has a closed angle 270 degrees.

The disc in the RE shows an inferotemporal notch with peripapillary atrophy and LE shows advanced damage with pallor of the disc. There is marked tessellation of the fundus in Be .

Here, we do not know if he had POAG before cataract surgery, which is unlikey as he was qiute young when cataract surgery was done. There is also absence of positive family history.

The RE diagnosis could be secondary open angle glaucoma, and LE secondary closed angle glaucoma.

The RE fields show early damage, OCT appears WNL. The LE fields show advanced damage and OCT shows marked decrease in RNFL thickness. Hence the target IOP in the LE needs to be lower. He is already on 3 medications in both eyes !

I feel the RE medications can be reduced to one or 2 drugs with close monitoring of IOP .

Regarding the LE, which has advanced damage and not so well controlled IOP, adding a Carbonic anhydrase inhibitor (oral) would bring down the IOP for now. For long term pressure control,adding topical CAI may be considered, with monitoring of IOP. With 4 drugs also, I fear the IOP may not be acheived to desired low levels. The next alternative is filtering surgery with adjuant mitomicin C.

This choice though is not without risk. This is aphakic glaucoma,with advanced disc and field damage. Surgery carries high risk of wipe out of the remaining field.

MP : The left eye IOP control was not adequate. Topical dorzolamide was added. The patient was now on four medications inspite of which the IOP was still 17 – 20 mm Hg. I would like to mention that this patient had very poor compliance.

We know that PG anologues are contraindicated in these patients, still as he was on careprost for a very long time and did not develop CME, we switched him from Careprost to Travoprost in the left eye only and omitted Combigan. With this his IOP came down to 14 -16 mm Hg after 15 days. The reason behind changing from one drug to the other in the same class is that there may be certain patients who do not respond well to a particular molecule.

He still needs close watch for development of CME or any new signs of inflammation. He may need surgical intervention in left eye in future.

CASE 5 CLINICAL DATA -

62 yrs. Male, came for routine eye examination. He is a high myope. He had been diagnosed with glaucoma in January 2009. He was applying Timolol 'ocassionaly'.

He had undergone RD surgery of Right Eye in 1981. There are no H/O of systemic diseases

O/O/E : BCVA: OD- 6/24,PH6/12,P, N6 (-15.25/-2.50@80); OS- 6/18,P, PH 6/9,P, N12 (-15.00/-3.50 @70)

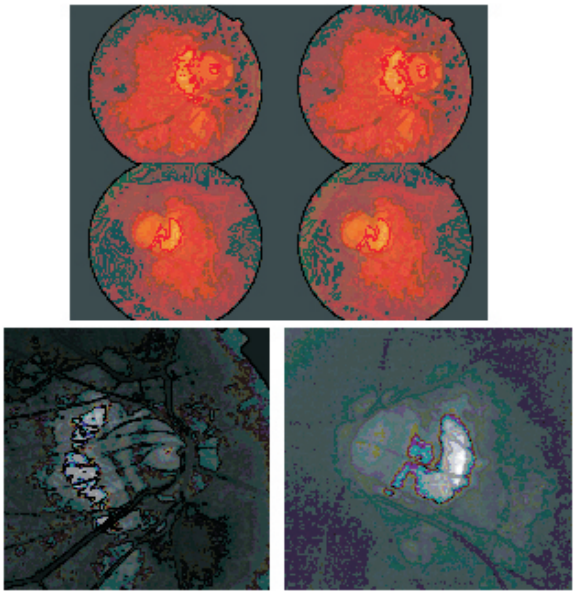
SLE OU : Cornea Clear, AC Deep, NS 2+

AT : 18/18 mm Hg

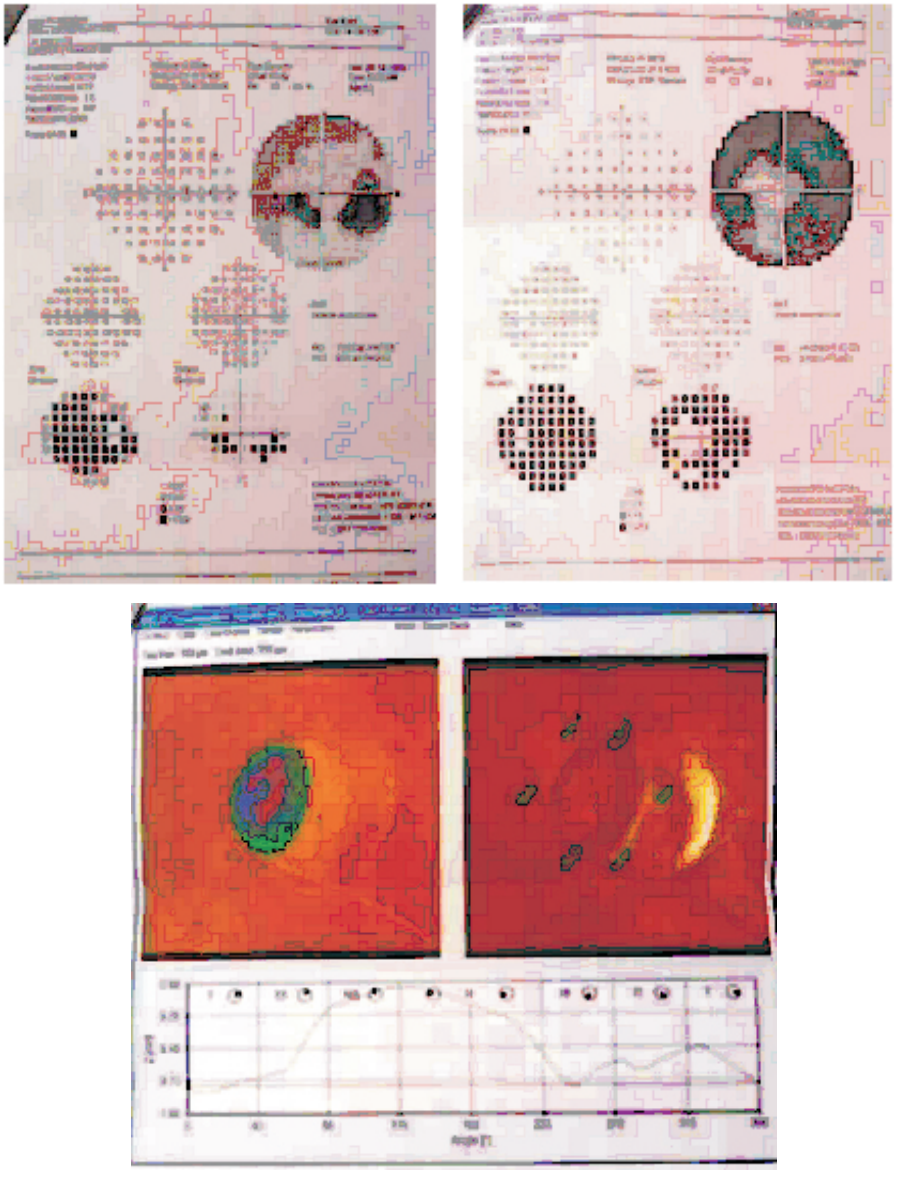
Gonio- OU open till SS

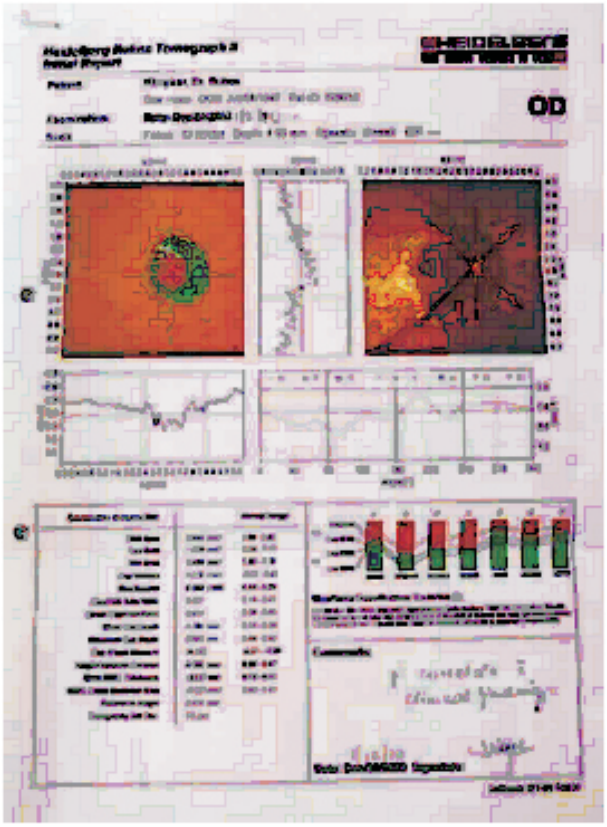
CCT-540/535 microns

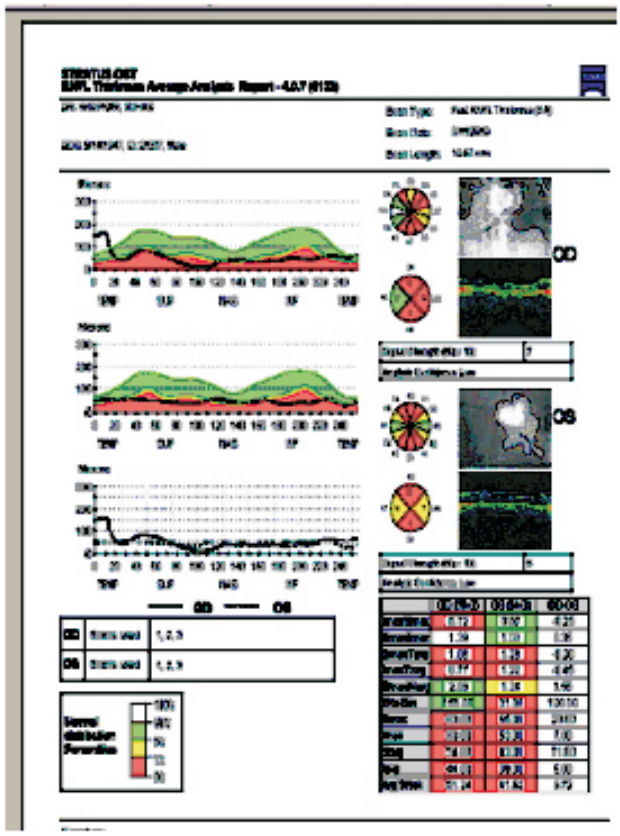
ONH Image

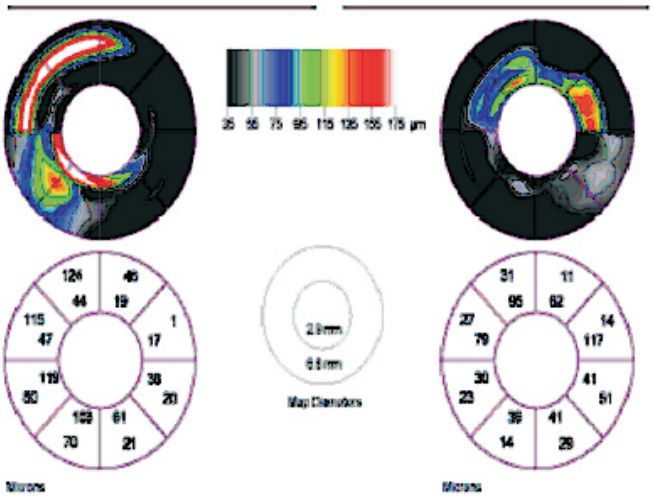


Automated Perimetry









Questions to panellist -

* Your comments on co-relation between HRT & OCT reports.
* What should the management be ?
* Any other comments.

RN : In tilted disc with PPA the marking of disc margin often erroneous.

IOP definitely needs to be lower

Also rule out associated systemic problems-(without relying on history alone) and vaso dysregulation.

VC: 62 yr old male pt, with high myopia, early cataract and diagnosed glauoma in the left eye.

Iop is 18 & CCT are okay Fundus in RE shows a contour cup of about 0.7 -0.8:1 with sloping and a healthy NRR, peripapillary atrophy and marked tesselation. The LE shows more advanced damage with pallor of NRR.

Visual fields in RE shows inferior paracentral scotomas. THe LE shows advanced field loss.

In the HRT of RE: The patient is a high myope (-15 D). The normative database in the HRT-II is based on normal eyes with refractive error of less than 6 dioptres.and optic disc size between 1.2 and 2.8 mm x mm.Also the image quality(focussing)is affected in eyes with refractive error > +/-12 D.

The disc size is on the higher side. These factors should be taken into consideration when interpreting the report.

The reflectance image (on the right of the report) is divided into 6 scetors namely temporal,temp/ sup, temp/inferior nasal, nasal /superior and nasal/inferior for analysis. The RNFL thickness is analysed in each of these sectors as well as totally (globally).The report is given as WNL (green tickmark), borderline(yellow exclamation) and outside normal limits (red cross). In each sector.the RNFL thickness measured along the disc margin is shown opened up in the box below the reflectance image.

In this case there is a loss of the 'double hump pattern, especially in the superotemoral and superonasal sectors. The Moorfields classification shows the infero temporal sector having borderline damage, the rest of the sectors being WNL. In the inferior temporal sector the neuroretinal rim(top line of the green bar) falls between the 95and 99 % predicted interval, in such case this particular segment is classified as "borderline". If the top of the green bar lies above the 95 % prediction interval, then the disc segment is classifed as "WNL". If the top of the green bar lies below 99.9 % prediction interval, then the disc segment is classifed as "Outside normal limits".The most abnormal of the seven classifications (whole disc and six segments) gives the overall classification of the optic disc.

Abnormalities other than glaucoma, such as tilted discs, may cause measurements to fall outside the normal range. There is an overlap of measurements between normal and glaucomatous eyes, so that classifications such as 'within normal limits', 'borderline' and 'outside normal limits', are appropriate.

They provide measurement information that should be integrated with other clinical information, such as intraocular pressure level and visual field status.

The LE HRT should be interpreted with caution, as mentioned above. The Optic disc is classified as WNL ie green ticks in all sectors, which is misleading. Also, the image is not well focussed which may be due the high myopia as well as cataract.

Here lies the importance of correlating the clinical findings with the imaging technique result .The optic disc is showing advanced glaucomatous damage and the case should be treated based on our clinical interpretaion.

Regarding management, the present mediactions have not been mentioned. I might start Prostaglandin analogue in both eyes and have regular follow up.

SP : Patient is a myope .According to the stereophotographs, the patient has a tilted disc in the left eye but the right eye disc does not have this abnormality. Therefore objective tests in the form of HRT and OCT may not really be helpful but since they have been done follow up after a few months with these tests may be helpful in detecting progression of glaucoma. The patient' diurnal variation

should be done to see if there is any fluctuation of IOP. Treatment would preferably be based on IOP measurements. For the left eye 18 mmHg seems a little more considering his optic cup, therefore one more drug could be given to reduce iop.

SS : The HRT accuracy has been jeopardized by the tilted disc in the left eye, the peripapillary atrophy and the incorrect disc outline. The OCT shows definite thinning in both eyes. If the IOP is 18 without Rx, we need to do the systemic evaluation for NTG. I'd start with a beta blocker. Is there a maculopathy in the right eye? We need to consider prostaglandin add on therapy after deciding that.

MP : This case shows a how good clinical correlation plays an important role. HRT in this case is erroneously normal because of the tilted discs. This is a classic example of ordering the correct investigation in a given patient, considering his overall status, so as to avoid false positive or false negative errors.

OCT analysis shows thinning of RNFL. OCT in cases where the patient has high myopia shows thinning of RNFL and also shows thinning in macular map. The only thing which we should look for in OCT is correlation between myopia and RNFL thinning. This patient has myopia of -15 DS in both the eyes but he has RNFL asymmetry of 10 microns, left eye more thinning than in the right eye. This corresponds well with ONH findings and findings on perimetry.

This patient was also investigated for cardiovascular risk factors. He did not have any major CVS or CNS disease, but had microvascular infarcts (on MRI), which were asymptomatic & a marginally raised serum homocystein level. How this co-relates with the optic neuropathy is to be investigated.

We can start this patient on single medication initially. Periodic evaluation with OCT, as it gives us a direct measurement of RNFL and perimetry will be helpful for follow up of this patient.

MCI REGULATION FOR DOCTORS

NEW REGULATION 2009, PART I- NOTIFICATION DT 10-12-2009

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Chairman, Medico legal cell, IMA Pune and Maharashtra State

The long awaited code of ethics, etiquettes and beahviour regarding interaction between doctor and pharma and allied industries (like instruments/light source/scopes etc and manufacturers, distributors and so on) has been recently published. It is on the same lines as has been encoded a couple of years back by American Medical Council.

A new code is added under a new clause 6.8, which now covers doctors and professional association of doctors in their relationship with pharma industry & allied healthcare sector industry are as follows :

(a) **Gifts** – no gifts are to be accepted- newer amendment is in offing, which will quantify the gifts and a gift below Rs 1,000/- will be exempted. A gift betwee 1 to 5 thousand will result in fine and beyond that, in temporary suspension of registration for 1 month - and so on. A gift beyond 100,000/- will result in suspension for 1 year!

(b) **Travel** : doctor is prohibited from receiving aid to attend conferences, seminars, workshops, pleasure trips etc. for himself and for family members etc. The rules for suspension are as above.

(c) **Hospitality** – no hospitality for doctor or his family member in form of hotel accommodation etc.

(d) **Cash/monetary grants** – doctor cannot receive any of these under any pretext.

Exception – funding for medical research or study can be received through an approved institution by modalities laid down by law/guidelines adopted by such approved institutions in a transparent way and it shall always be fully disclosed

(e) **Medical research-** A doctor may carry out/participate/work in a research project funded by pharma industry if following provisions are met:–

(i) Ensure that the concerned proposal (s) has due permission from the competent authorities.

(ii) Ensure that the project has clearance from National/State or institutional ethics committee/body

(iii) Ensure that it fulfills all the legal requirements prescribed for medical research

(iv) The source and amount of funding is publicity disclosed at the beginning itself

(v) If human volunteers are involved, ensure there are proper care & facility for them

(vi) Animals be involved only when needed and they be treated in scientific and humane way.

(vii) In the MOU [memorandum of understanding] for conducting research work or similar document, ensure that doctor shall have the freedom to publish the outcome of research in the greater interest of the society

(f) **No compromise over professional autonomy as autonomy and freedom of medical institution.**

(g) **Doctor may work for a pharma or allied healthcare industry in advisory capacity or as consultants, as researchers, as treating doctors, etc. with following provisions :**

(i) Professional integrity and freedom are ensured

(ii) Patient's interest is not compromised

(iii) Lawful

(iv) Fully transparent and disclosed.

(h) Endorsement

A doctor shall not endorse any drug or product of industry publicly. Study conducted on efficacy or otherwise of any product can only be presented through appropriate scientific bodies or publicized properly in a scientific journal.

Doctor is expressly prohibited from endorsing or promoting any drug or instrument or healthcare product. All types of promotional and tempting tactics like travel and hospitality, gifts and grants etc are banned. Research projects approved through due process of law and regulations are only allowed and that to after following certain norms.

A doctor should not be caught unaware and hence, the rules and regulations are published here with some explanations and comments.

After a special meeting with MCI of ficials, convened for elaboration of these amendments, in Mumbai on 28th Mar. 2010, following details have emerged-

These changes have been necessitated due to constantly demeaning the profession by pharma and healthcare industry. These will go a long way in upholding the dignity of the profession.

The MCI rules and regulations have the enforceability, even through courts, because Indian medical Council Act is a "delegated" legislation passed by the parliament. (Jc. Dhananjay Chandrachud, of Bombay High Court).

Do we need codified (written down) ethics? Yes. They have a definite role—it has both effects-corrective as well as deterrent. A wrong doer should not go unpunished, as well as, the action taken shows what may happen, to the next wrong doer.

An opinion was expressed in the seminar, that MMC should think seriously before putting in any new clauses, as courts will give literal meaning to the printed code.

Can medicine samples be accepted? Probably "yes". The answer from Hon.Gen. Secretary, IMA's headquarters in New Delhi, is affirmative.

**Next set of amendments is almost ready regarding compulsory updating through system of "credit hours' and the criteria are really stringent A draft copy is available with this author if any doctor is interested. I feel, due cognizance should be taken at this stage only, so that the professional organizations can adopt the changes rather smoothly.

**Another set of amendments regarding cross-speciality is also getting ready and about 75 specialities' fields have been defined.

In my opinion more stringent the rules made by MCI, more will be the difficulties we will have to face as practicing doctors. MCI is making path of qualified doctors more and more difficult. Instead, MCI seems non-competent in stopping cross-pathy, allowing only its registered doctors to practice allopathy, though it is clearly mentioned in its section 2.

The rules made by MCI are implemented through State Medical Councils—in Maharashtra, it is through MMC i.e. Maharashtra Medical Council. The elections took place last year in 2009 April, and 9 members from Maharashtra have been elected. But 11 Government (of Maharashtra)

Nominated members are not yet appointed, and hence, unfortunately, MMC is yet NOT functional.

Overall, the rules made are difficult to be implemented and the ingenious brains will find many ways!

Of course, the intentions are laudable and something was needed to be done in regards to gifts, travel, hospitality and sponsorships—only thing I pray is for its uniform and unbiased implementation. However, the updating (CME) amendments are difficult to implement and MCI should seriously consider its repercussions before implementing them. The spirit of amendments is worthy and we should try to adhere to them to the maximum possible.