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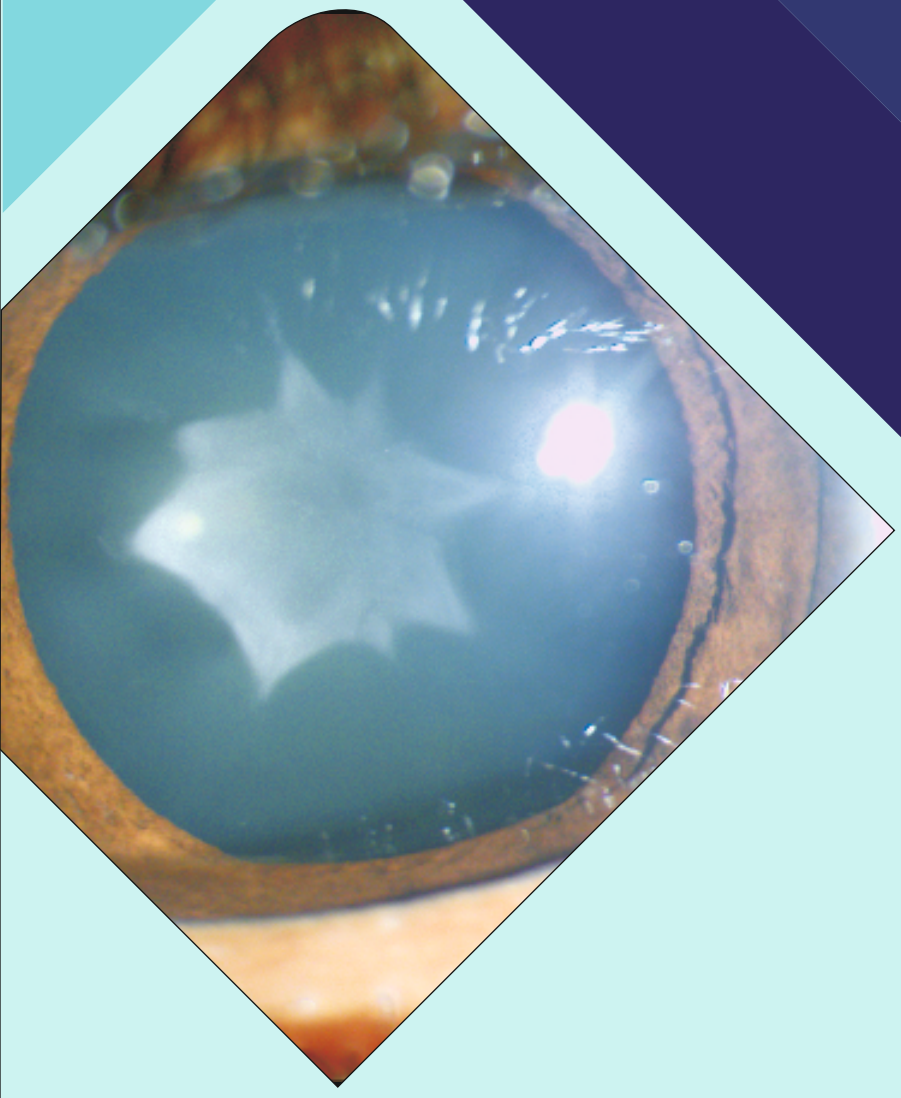

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UP JOURNAL OF OPHTHALMOLOGY

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Cover Photo

Rosette Cataract due to Blunt Trauma.

Courtsey : Dr Shalini Mohan, Kanpur

"WHEN GOING GETS TOUGH, THE TOUGH GETS GOING"

" The art of living is neither careless drifting on the one hand nor fearful clinging to the past on the other.It consists in being sensitive to each moment, in regarding it as utterly new and unique,in having the mind open and wholly receptive."

Above mentioned words of Alan W. Watts hold so much wisdom and guidance especially in this current chaotic covid era, which has not only burdened the medical resources but has also severely impacted our mental health. Indeed seeking knowledge and learning proves to be an effective tool in combating the fear of unknown and brings in cultivating faith and hope of a peaceful future.

I congratulate the editor Dr, Shalini Mohan for her sincere efforts in improving the sccientific material and for the upcoming issue of UPIJO.

Wishing all the readers a great scientific treatise and an insightful learning experience. Happy learning.

Dr. Shrikant, MS

President, UPSOS
Former Professor & Head, Regional Institute of Ophthalmology
Institute of Medical Science BHU, Varanasi
Presently Prof. & Head Department of Ophthalmology
Heritage Institute of Medical Sciences, Varanasi

Dear Members

'Ethics has been called as “Religion of Science”……..'

Edwin Grant Colwin

Ethics are bestowed on to us with the 'Hippocratic oath” as we finish the medical school. By definition '**Ethics**' is the philosophical discipline concerned with what is morally good and bad and morally right and wrong. Ethics is the soul of medical profession and the soul is the purest. The biggest strength in medical practice is being ethical in your diagnosis, investigations, treatment and in research. And if one is confident about his moral values no one can in this world can challenge his righteous principles.

When we see around, the faith and belief of the people about medical profession seems dwindling. It is not just common people but also we see the colleagues unethical and malpractices that keeps on surfacing and resurfacing. All these form an cloud of dismay and dissatisfaction. But we really need to introspect…….

Happy to present before you another issue of UP Journal of Ophthalmology having articles of great interest for the readers. I invite constructive criticism to improve upon it. Thanks to President Dr Shrikant, Secretary DrMohita Sharma, CSC Dr Deepak Mishra and whole editorial board along with executive body members for their constant support.

Wishing you all a very Happy and Prosperous New Year.

Dr Shalini Mohan

MBBS (Gold Medalist), MS, DNB, MNAMS, FCGP
Editor, UP Journal of Ophthalmology
Associate Professor of Ophthalmology, Kanpur
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Ex Consultant, Sir Ganga Ram Hospital, New Delhi.

Secretary Message

Dear all

Welcome to COVID times !! In an era where almost everything seems to have come to a standstill the UP Journal of Ophthalmology continues to come out and impart scientific knowledge to all its members. This journal signifies that life can never stop and scientific upgradation is a continuous process.

As a State society UPSOS is committed to imparting scientific knowlegde. On behalf of the whole executive committee my Congrarulations to the editor and the editorial team.

As doctors we took the hippocratic oath and promised to serve humanity. My appeal to all our dedicated members is to continue to woek whole heartedly and simultaneously be updated about all precautions which can keep you safe. Take special care of yourself especially from the ophthalmology point of you. And since workload is less ours being mostly an elective branch,spend more timing reading good journals like this one. You also have the opportunity to do what you never got the time to do. In this one important thing is studies and publishing. So let's get started on this academic front too. And hope to see more contributions of structured study results from our own state of Uttar Pradesh.

Stay safe, stay healthy and stay working

Dr. Mohita Sharma
General Secrtary UPSOS
Director
Tirupati Eye Centre, Noida

My Life Journey

Dr. Jignesh Taswala
Senior Eye Surgeon, Darshan Eye Care Centre, Hon. President MOS, Member Advisory POS, Member Scientific MOS

Many thanks Dr Shalini Mohan Editor of UP Journal of ophthalmology.

She had requested me to write an editorial of my choice and now that I am 60 plus I would definitely like to share my life journey which will be an inspiration to the young generation.

Usually nobody wants to share their personal life

In my case lot of issues in my personal life had to be balanced with my professional life. During my childhood I had to face a lot of hardships so much so that we use to have one meal a day .My dad earnings were so meagre that we use to manage a pair of clothes & shoes for atleast 3 yrs.Passed out my 10th n 12th std with flying colors inspite of all d hardships.

12th marks were good so I got admission in both 1st in Engineering n den Medical Stream ..I opted for the latter.

On the 1st day of MBBS my father was in hepatic coma.He was the sole bread earner.Chances of his survival way back in 1977 was 0.01%.So in fact the Dr in charge had given up & told us to prepare for the final rites. Dad was in admitted in General ward.Mom totally illiterate had not a pie to pay.She herself was crying inconsolably.All our relatives were in Surat & with no phones ..landline or otherwise so it was difficult to contact.Some how thru common family friends could contact my uncle & grandfather who came & salvaged the situation somehow by helping with finances

My paternal uncle on his way back come came to know his brother is seriously ill.He reached the hospital & told us not to loose heart & take a 2nd opinion.Miracales happen & he came out unscathed & was discharged on 5th day of his hospital admission. As he survived he could pay my tuition fees & I got MBBS admission to BJMC ,Pune else would not be able to pursue.That was my destiny.

For my Post graduation M.S Seat I had to fight with Pune University in Mumbai High court..To fight the case I took up a job in Mumbai hospital & paid to the Lawyer n won the case .Pune university had to create an extra seat for me ..So the next big hurdle was crossed.

Then I got a call from Sankara Netralaya for an interview for my fellowship in VR Surgery.Dr S S Badrinath Sir took it.I had passed out from an institute were slit lamp was a luxury & lo here i was being interviewed at a premier institute but Sir was impressed & I got selected & completed 1year inspite of the pyramidal system (in which you were told to leave inbetween if you are not upto the mark) Infact Sir was impressed with my work & offered 3 months extra research fellowship in Diabetic Retinopathy.

After completing my VR Fellowship I came back to Pune to settle.My dad had just retired.He had put all his retirement corpus in my elder brother's buisness who did not fare well n went bankrupt with 3yrs of starting n went into severe depression with 2 children & wife to look after.I got married & unfortunately for me she had Schizophrenia which was not revealed to me as she was already on antipsychotic drugs as her mother was a dr & use to cal her home fortnightly n clandestinely give her injections.She conceived & had twins but during pregnancy she aborted one.After repeated sonology who assured that 2nd twin was fine.

But on delivery ..he had multiple congenital anamolies

n need sequential 8 surgeries continued to partly rectify one of which was open heart surgery

I was the sole earner now with no support whatsoever

I started my rental practice with just a refraction box n an ophthalmoscope n used to go to periphery of pune around 50km daily collecting a meagre 20rs as consulting going from each patient that to door to door of General practicitoner. End of the day earning was just enough to cover up my expenses

On my personal front false court cases where filed against me which I had to fight n culminated in divorce..lot of mental agony n financial drain..Child custody was given to my wife as he was minor.

My 2nd marriage also didn't work out as she had severe bipolar depression & had to go seperation & litigation in family court which till todate I am fighting n paying heavy alimony that too back dated from 2003.

On the professional front I became the joint secretary of POS n later secretary & then VP & President of POS in 2010.

My father fell down had fracture neck femur & was bedridden for almost 5yrs as after the surgery he developed sepsis & lost him in 2005.

My brother due to his temper tantrums use to create lots of discord in family n whole society so had to admit him in mental hospital repeatedly & lost him in 2015.In the mean time my son started staying with me.He had gone into deep depression so I had to get counselling done as he was pursuing law but flunked 4 times so I shifted him to optometry & trained him..he passed out wd flying colors

My mother in the mean time fell ill & was bedridden for 7yrs .

Lot of mental stress as all by myself single handedly was looking after my aged diabetic overtly obese mother with 24hour servants n also my depressed child .Immense financial drain as had to manage court matters, permanent alimony , medications for my mother & child,24hrs servants ,cook & also expenses of clinic..

Still I did not loose heart fighting on all fronts solo climbed up the ladder to become joint secretary in 2013 of MOS & then Secretary ,MOS in 2014 .Then I had to fight an election for VP ,MOS but was forced to withdraw respecting our senior in favour of junior member 20yrs less my age.Then fought an election the following year for the same post of VP& lost & then again fought in opposition's home ground & won with a handsome margin to be VP & now President MOS .Also did my AIOS Leadership Development program way back in 2012.

Now before I took over as MOS President,I made 21 committee's with a theme of OVERALL WELLNESS OF MOS MEMBER'S that too well in advance (2months prior)before I took over.This is also 1st time in history of MOS

So it has been a monumental struggle all my life both on Professional & Personal life.This is not to get browny points or to gain sympathy but want young collegues of mine to take a leaf out it & vouch never to give up in life inspite of all odds.Thats my reason to share my life journey.

Dr. Jignesh Taswala

To Study the Outcome of Different Modalities for the Treatment of Astigmatism During Phacoemulcification

Nirupma Gupta, MBBS, Lokesh Kr. Singh, MS
Sandeep Mittal, MS, Alka Gupta, MS
LLRM Medical College, Meerut

Cataract affects approximately 20 million people world wide and this figure is expected to reach 50 million by the year 2020.

It is currently among the most performed planned surgical procedures world wide positively impacting over patients qualify of life.

Aproach to the surgical correction of astigmatism at the time of cataract surgery

One way to plan surgical correction of astigmatism is to initially assess the refraction and the keratometry simultaneously. If good correlation exists as to the amount of cylinder and axis, the surgical planning for astigmatism correction during cataract surgery is fairly straightforward. If, however, there is poor correlation (even though keratometry should be more reliable), surgical correction can be less predictable, even with corneal topography. This is where the "art" of astigmatism correction applies. The surgeon needs to also judge the relative reliability of the astigmatic information. If, after careful consideration, there is doubt as to a reasonable surgical plan, the astigmatism correction should be postponed until after cataract surgery and an adequate time for incision healing rather than astigmatism "correction" or "treatment" is an important subtlety. The terms In some instances, a patient's astigmatism will be worse after surgery than it was before, but hopefully the unavoidable increase will be minimized or paired peripheral corneal relaxing incisions (PCRIs), and toricIOL implantation. For less than 1 D of corneal astigmatism, the phacoemulsification incision is placed in the steep axis. For 1 D can be utilized. We prefer LRI for astigmatism from 1 D to 1.5 D and toriclOLs for astigmatism above 1.5 D. ToriclOLs and LRI are used . The need for LRI is amarket and the availability of toric corrections in accommodating and multifocal lOLs.

Stepladder Approach to Management of Astigmatism at Time of

Cataract Surgery

Comeal Astigmatism	*Treatment Approach*
<1 D	incision on steep axis
1 D to 1.5 D	LRI incisions
1.5 D to 2.5 D or more	Toric intraocular lens

Limbal relaxing incision

Limbal relaxing incisions (LRIs) can flatten astigmatism as an adjunct to cataract surgery. LRI's have been demonstrated to predictably alter the corneal curvature by flattening the cornea in the meridian in which they are placed and allowing for a commensurate amount of steepening 90 degrees away

A number of nomograms are available to determine the arc length and number of incisions for a certain amount and axis of astigmatism, such as the Donnenfeldnomogram

Nichamin Nomogram

When performing LRIs alone, the number and length of incisions are determined according to the using nomogram.

This nomogram, which titrates surgery by length and number of LRIs, should be considered a starting point. The goal is to reduce cylinder power and absolutely avoid overcorrecting with-the-rule cases because we want to minimize against-the-rule astigmatism. In cases with 0.5 D or less of cylinder, only an astigmatically neutral cataract incision is used.

TABLE 1

Nichamin Nomogram for Limbal Relaxing Incisions to Correct Astigmatism With Phacoemulsification

	Degrees of Arc to be Incised—Against-the-Rule Astigmatism						
	Age (y)						
Cylinder (D)	30 to 40	41 to 50	51 to 60	61 to 70	71 to 80	81 to 90	>90
0.75 to 1.25	55	50	45	40	35		
1.50 to 2.00	70	65	60	55	45	40	35
2.25 to 2.75	90	80	70	60	50	45	40
3.00 to 3.75	90*	90*	85	70	60	50	45
	Degrees of Arc to be Incised—With-the-Rule Astigmatism						
	Age (y)						
Cylinder (D)	30 to 40	41 to 50	51 to 60	61 to 70	71 to 80	81 to 90	>90
1.00 to 1.50	50	45	40	35	30		
1.75 to 2.25	60	55	50	45	40	35	30
2.50 to 3.00	70	65	60	55	50	45	40
3.25 to 3.75	80	75	70	65	60	55	45

*Optic zone to 8 mm.

Toric intraocular lens

The first toric intraocular lens model was approved by the FDA in 1998. Widespread use of toric IOLs would not come until later, however, with approval of a foldable toric IOL in September 2005. Toric IOLs have the advantage of being able to correct large amounts of astigmatism. Alcon has published a web site for their toric IOL calculation which is very useful and takes into account induced astigmatism from the wound

(http://www.acrysoftoric-calculator.com)

There are 3 important prerequisites to success with toricIOLs. First, the surgeon must be able to perform astigmatically neutral surgery or be able to accurately estimate his or her surgeon-induced astigmatism so that the lOL's cylindrical power and axis can be selected based on the preoperative keratometry measurements. Second, the axis of the toricIOL must be properly aligned during surgery. Finally, the lens must not rotate out of alignment postoperatively.

Aim And Objectives

1. To assess astigmatism prior to cataract surgery.
2. To study the results of different modalities used during phacoemulsification for astigmatism treatment.
3. To assess final visual outcome.

Materials And Methods

This prospective, parallel, cohort, non-randomized study was performed at the Upgraded Department of Ophthalmology, LLRM Medical College, Meerut.

Total 60 patients were selected from OPD and undergone a complete ophthalmic examination that included uncorrected visual acuity (UCVA), refraction under cycloplegia, slit lamp biomicroscopy, tonometry, indirect ophthalmoscopy, automated keratometry and Ultrasonic biometry was used to calculate IOL power using the SRK-T formula in all study groups. The study was conducted from 2018 to 2019.

Inclusion Criteria :

All patients with age related cataract with preexisting astigmatism of less than 4 D where included in the study.

Exclusion Criteria :

1. Patients having irregular astigmatism.
2. Astigmatism due to pterygium.
3. Previous history of any surgery in the same eye.
4. The same Eye having Corneal Opacity 5.Those having traumatic or complicate cataract

6. Sever dry eye.
7. Macular degeneration or Retinopathy.

Three groups were formed on the basis of astigmatic error

Group 1-Patients were selected with astigmatism of < 1D

Group 2- Patients were selected with astigmatism of 1.00D to <2.00D

Group 3- Patients were selected with astigmatism of 2-4D.

Surgical Management in Three Groups-

GROUP 1- Patients were underwent clear corneal incision at steep axis during phacoemulsification.

GROUP 2- Patients were underwent for limbal relaxing incision using Nichamin nomogram during phacoemulsification.

Group 3- Patients were underwent for implantation of ToricIOL by phacoemulsification.Toric IOL axis and power were calculated by online acrysof Toric IOL calculator.

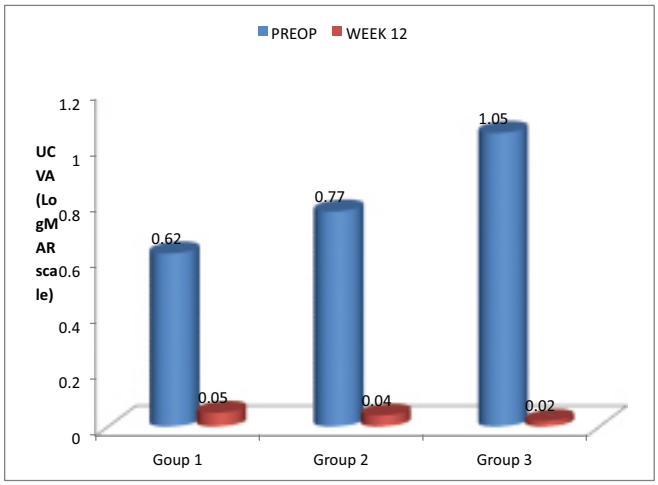
Observations And Results-

TABLE 1-Preoperative UCVA and postoperative UCVA at week 12-

UCVA(logMAR) mean±SD	Goup 1	Group 2	Group 3
PREOP	0.62±0.30	0.77±0.36	1.05±0.25
WEEK 12	0.05±0.03	0.04±0.02	0.02±0.02
P value	0.003	0.003	0.001

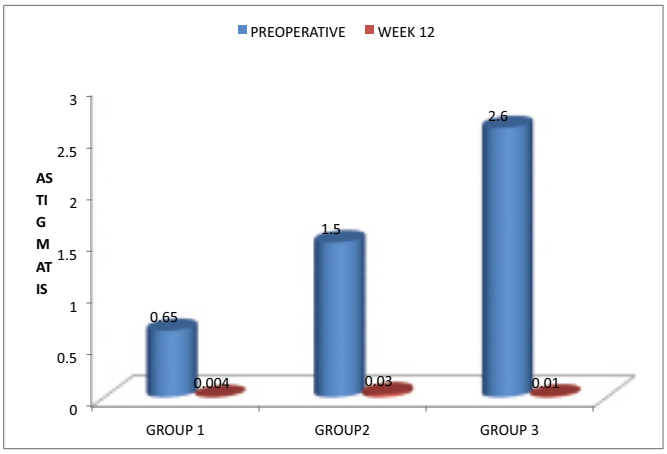
On applying non parametric WILCOXON SIGNED RANKS TEST based on positive ranks the average difference in UCVA (log MAR) AT week 12 postoperatively from preoperatively were all

Statistically significant ,taking p value <0.05 as statistically significant. P value in all the three groups were significant.



ASTIGMATISM (MEAN±SD)	GROUP 1	GROUP 2	GROUP 3
PREOPERATIVE	0.65±0.19	1.5±0.30	2.6±0.57
WEEK 12	0.04±0.08	0.03±0.13	0.01±0.02
P VALUE	0.001	0.001	0.002

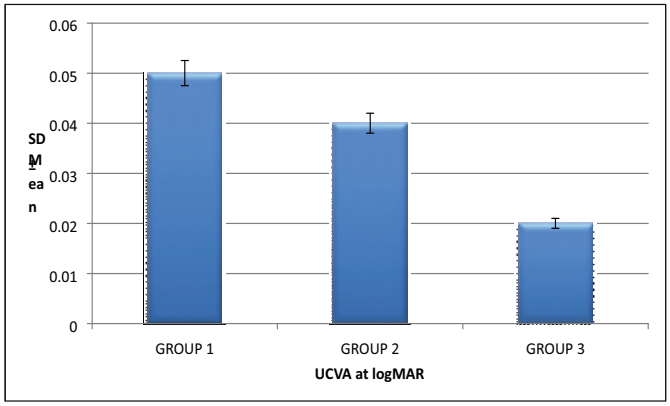
On applying non parametric WILCOXON SIGNED RANK TEST based on positive ranks the difference in Astigmatism at week 12 postoperatively were all statistically significant ,taking p value<0.05, as statistically significant



Comparison between three groups for improvement in UCVA at week 12

UCVA(MEAN ±SD)at logMAR	GROUP 1	GROUP 2	GROUP 3
AT WEEK 12	0.050±0.03	00.04±0.02	0.03±0.02

THE UCVA (logMAR) in postoperative group was analysed after applying KRUSKALL WALLIS NON PARAMETRIC TEST and taking p value <0.05 as statistically significant .H statistic is 7.719. -P value is 0.02. and this was considered to be statistically significant.

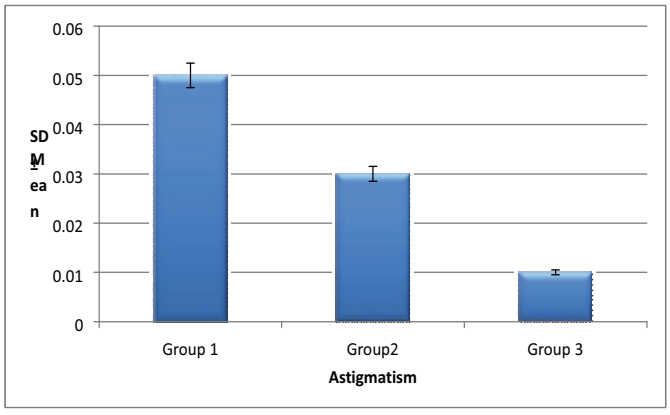


Comparison between three groups for the improvement in astigmatism between three groups at week 12

Astigmatism (mean ±SD)	Group 1	Group2	Group 3
at week 12	0.05 ±0.13	0.03 ±0.15	0.01 ±0.12

The Asmatigmatism in postoperative patients was analysed after applying Kruskall Wallis non parametric test and taking p value <0.05 as statistically significant

The p value is 0.012 and this was considered to be statistically significant.



Discussion

This study represents 60 patients visiting to our OPD confirmed inclusion criteria as laid down with corneal astigmatism of more than 0.50(D) to 4(D)

A study done by Hossein mohammad –Rabei and his colleagues in 2016 The results of this study showed that UCVA 20/40 at week 24 was achieved in 97% of eyes in thee EOAI group, 82.4% of eyes in the LRI group, and 76.2% of eyes in the tIOL group.

In a similar study done by carvalho et al who found LRI as a safe approach to correct astigmatism during phacoemulsification reported a postoperative UCVA of 6/12 in 75% of cases. my result is better than this result in LRI group.[10]

The result of our study showed that UCVA 6/9 at week 12 was achieved in 90% of eyes in clear corneal incision group 1,85% of eyes in group 2,and 95% of eyes in group 3.

Change in astigmatism at day 1 in all the three groups were significant.but there were a large change in astigmatism at day 1,0.14 ±0.22 in clear corneal incision group as compared to two other groups. Manpreet Kuar at al.in a study done on patients undergoing cataract surgery and toric IOL implantation with pre-existing high astigmatism.and concluded that toric IOL demonstrate good visual outcome with UCVA better than than 6/12 in 97% to 100% Of patients. and residual astigmatism lower than 0.50D in 38-78% of patients.our results are approximately same.[12]

Similarily in our study in toric IOL group 90% Patients improved to UCVA of ≥ 6/9 and residual astigmatism lower than 0.50 D in 15% patients.[12]

Dr K. Ravikumar at al done a study on efficacy of limbal relaxing incisions in correcting corneal astigmatism along with clear corneal phacoemulsification .in his study done on 50 eyes of 37 patients the preoperative UCVA was 1.0 with a standered deviation of 0.4.The post operative UCVA at 4 weeks 0.0 with standered deviation of 0.15 in logMAR units. In our study in mean UCVA in limbal

relaxing group at 4 week was 0.07 with standered deviation of 0.14.

A study done by Nick Mamalis in may 2019 concluded that there were statistically significant difference when looking at the mean residual astigmatism with toric IOL resulting in less cylinder than CCI.But in our study all the three modalities are equaly effective when comparing there preoperative and postoperative at week 24

The results of the present study should be interpreted in the context of its limitations. The study was not randomized which explains why preoperative astigmatism was significantly higher in the tIOL group.

All the three methods used in our study are effective , safe and predictable method.

However drawbacks of clear corneal incision is that it can't correct high astigmatism, while limbal relaxing incision and toric IOL implantation can correct moderate to high astigmatism .

Conclusion

Limbal relaxing incision is cost effective alternative to toric IOL and can be used in conjunction with cataract surgery to reduce moderate astigmatism .

Implantation of acrysof toric IOL is apredictive and safe method to `correct high astigmatism ,the stability of the lens in the capsular bag has been excellent .These lenses also appears to slow if not prevent the development of dense posterior subcapsular opacification

Outcome after toric IOL implantation are influenced by numerous factors ,right from the preoperative case selection and investigations to accurate intraoperative alignment and post operative care.

With the limitation of our study that is study was not randomized can't conclude that which study is better or which is best ,all the three methods to correct astigmatism during phacoemulsification give good results.

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Ophthalmology News:

Dry Eye? New Nasal Spray May Increase Tear Production

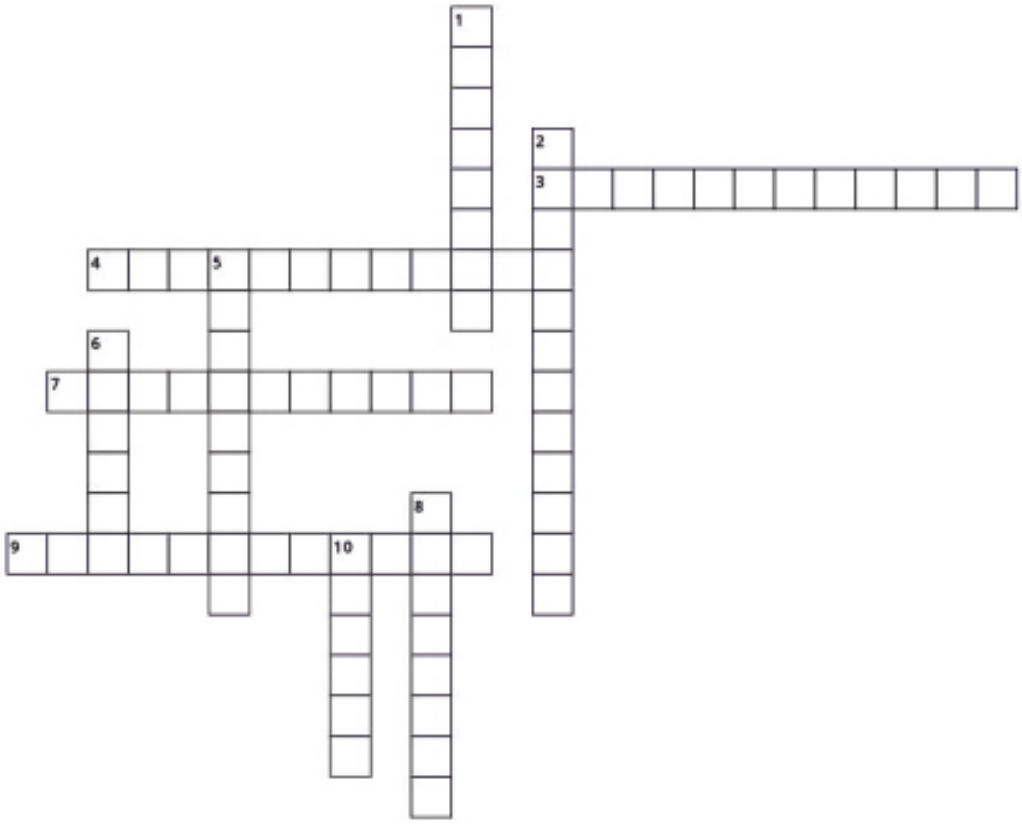
Varenicline (Tyrvaya, Oyster Point Pharma) is a cholinergic agonist that demonstrated an effect on tear production within five minutes of dosing in clinical trials. The nasal spray can restore the tear film homeostasis early on and, hopefully, prevent a lot of patients from having a chronic hyperosmolarity state that leads to more chronic inflammatory issues. The clinical trial results that led to FDA approval of varenicline last month. The study, with more than 1,000 participants, showed statistically significant increases in tear production after four weeks of twice-daily dosing with the nasal spray. The most common adverse reaction was mild sneezing, in 82% of participants. Other adverse events, also mild, were cough, throat irritation, and instillation-site (nasal) irritation, in 5% to 16%

Treat Presbyopia With an Eye Drop

The big news in this area came, when the FDA approved VUITY, a new eye drop medication developed by Allergan, an AbbVie company -- the first medication ever approved for the treatment of presbyopia.This drug has a rapid onset of almost 15 minutes and an exceptional duration, so this is a durable drug out to 6 hours relative to controls that improved distance-corrected near visual acuity almost three lines without affecting distance vision

Puzzle

Dr. Anchal Tripathi
Army Hospital, New Delhi



Across

3. Most common secondary tumor in retinoblastoma patients.
4. this drug is used in newborns with suspected chlamydial conjunctivitis.
7. this drug helps blepharitis patients by changing lipid viscosity.
9. Smokers should avoid this

Down

1. This phenomenon is considered to be an illusion.
2. MRI sign in patients with progressive Supranuclear palsy.
5. Paralysis of this nerve is the most common cause of vertical ocular misalignment.
6. a progressive study design.
8. Associated with PAX6 gene
10. In the collaborative normal glaucoma treatment study, progression was reducted by nearly three fold by reduction of IOP___%.

Endophthalmitis in a Vitrectomised Eye - An Unexpected Visitor

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Abstract

Purpose: This case report aims to highlight the rare incidence of endophthalmitis after pars plana vitrectomy and to elucidate the causative factors, implicated microorganisms, clinical features, prophylaxis, and treatment of this rare entity.

Methods: A 67-year-old male presented with complaints of a black spot in front of his right eye for 4 months. BCVA was CF@3m in RE and 6/9 in LE. On examination, RE had IMSC with a large macular hole, while LE was pseudophakic with an old macular tributary occlusion. The patient underwent Cataract surgery with 25G PPV + ILM Peeling + C3F8 in RE. 15 days later the patient developed endophthalmitis in the operated eye. The patient underwent a vitreous lavage with intra-vitreal antibiotic injections. One week later, the patient developed Retinal Detachment in the same eye.

Results: The patient was operated for macular hole and later on treated for endophthalmitis and RD. His final visual acuity was CF@2m.

Conclusion : Endophthalmitis following Pars Plana Vitrectomy has limited reports in the literature and is relatively uncommon. This case report highlights the factors which could lead to such incidences and discusses how to treat and prevent its occurrence.

Keywords : Endophthalmitis, Pars Plana Vitrectomy, Macular Hole

A 67-year-old male presented to our opd with complaints of a black spot in front of his right eye for the past 4 months.

Vision in the right eye was FC@3m and 6/9 in the left eye. With pinhole, vision in the left eye improved to 6/6 while there was no improvement in the right eye. The patient was a known hypertensive, controlled on medication.

On examination, the right eye was found to have a posterior subcapsular and cortical cataract, while the left eye had a PCIOL in the bag.

Fundus examination revealed a large macular hole in the right eye and the left eye had an old macular tributary occlusion with non centre involving cystoid macular edema (Figure 1), (Figure 2) (Figure 3.).

Figure 1. Fundus picture showing full thickness macular hole in the right eye and supero-temporal sclerosed vessel, suggestive of old macular tributary occlusion, with macular edema in the left eye.

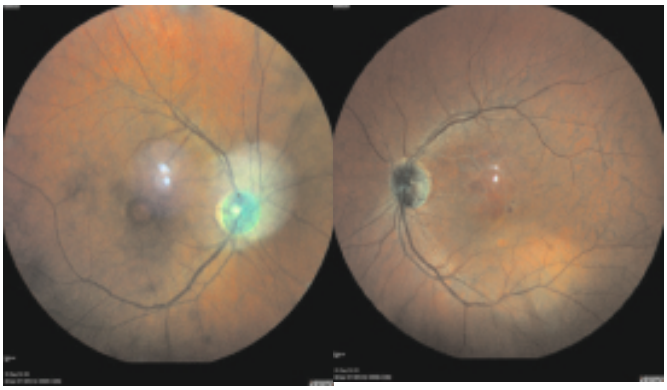


Figure 2. OCT of the right eye showing a large full thickness macular hole.

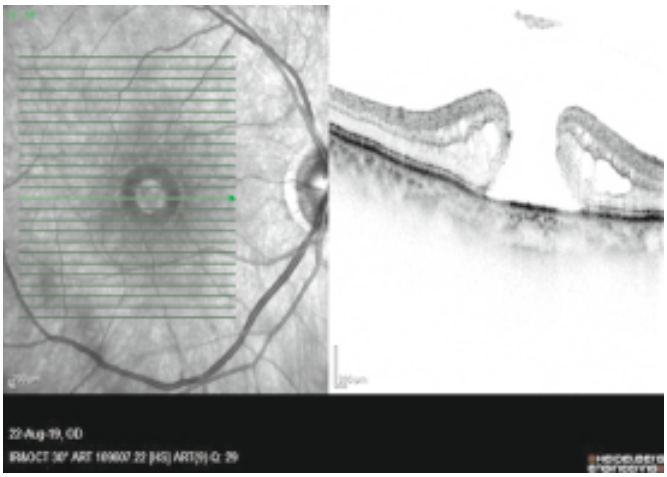
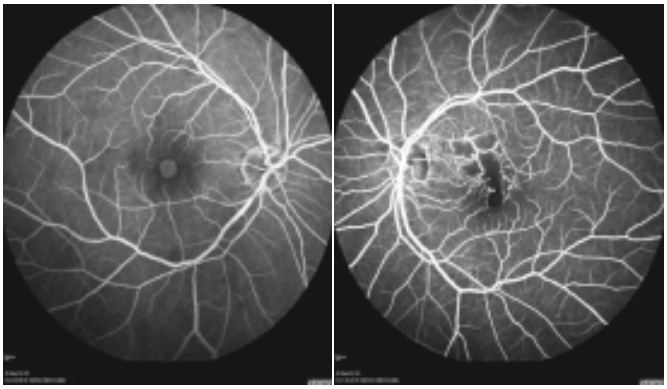


Figure 3. FFA of the right eye showing hyperfluorescence due to window defect owing to the macular hole and the left eye showing staining of the wall of the macular tributary vessel and capillary non perfusion area within the macula.

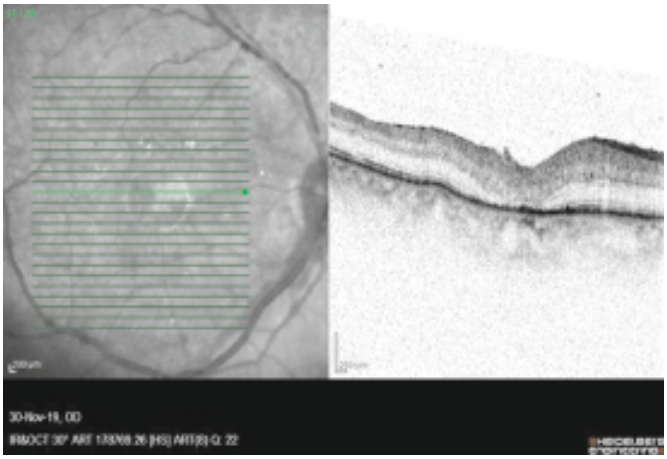


The patient underwent cataract surgery with monofocal IOL with 25G Vitrectomy + ILM Peeling + C3F8 Gas under local anaesthesia.

On post operative day 1, the right eye had mild corneal stromal keratopathy, PCIOL in the bag, and healthy looking retina with an 80% Gas Fill.

1 week later, the patient's vision in the right eye was FCCF. The gas bubble was at the level of superior vascular arcade (Figure 4).

Figure 4. Right eye OCT one week after surgery.



15 days after the surgery, the patient came with complaints of swelling, pain, and watering in the right eye for 1 day. The vision had dropped to HM in the right eye. The right eye had AC CELLS 1+ / Flare 1+ along with vitreous haze and severe exudation in the vitreous cavity. The patient was diagnosed with endophthalmitis. The patient underwent a Vitreous Lavage + intravitreal injection (Vancomycin (1mg in 0.1ml)+Ceftazidime(2.25mg in 0.1ml)+ Dexamethasone (0.4mg in 0.1 ml)) under local anaesthesia. The patient was also started on Tab. Ciprofloxacin 750 mg BD, fortified Vancomycin and Ceftazidime eye drops, on hourly basis, and eye drop Prednisolone Acetate qid.

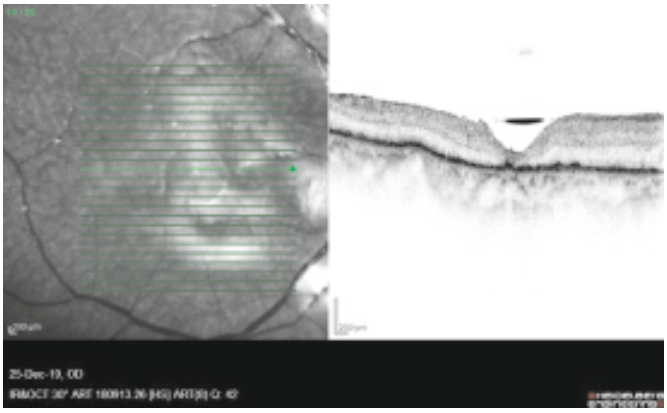
Culture reports suggested Coagulase Negative Staphylococcus Aureus. Anterior Chamber cells and flare and vitreous exudation didn't improve 2 days later and he was given a repeat dose of intravitreal (Vancomycin (1mg in 0.1ml)+Ceftazidime (2.25mg in 0.1ml) +Dexamethasone (0.4mg in 0.1 ml)).

3 days later, Anterior Chamber reaction and exudation had reduced and fundus glow had returned, with a hazy view of the disc. 5 days later, the vision had improved to 2/60. Fundus view was better and peripheral exudation in the vitreous cavity was noted (Figure 5.)

3 days later the patient came with diminution of vision in the same eye and he was noted to have a retinal detachment in the right eye. The patient underwent a

repeat vitrectomy + endolaser with silicon oil injection in the right eye.

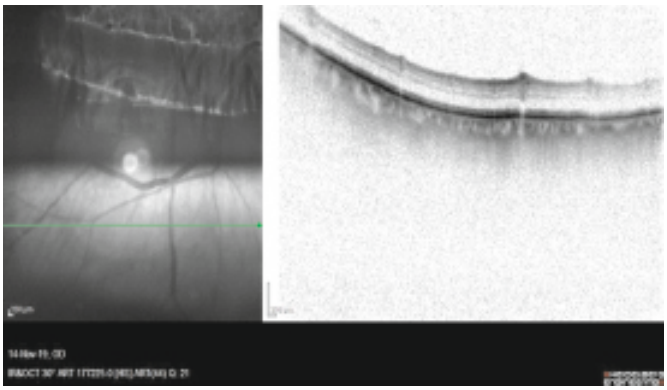
Figure 5. RE OCT after 10 days of vitreous lavage.



On Post-Op Day 7, his vision was CF@2m and retina was well attached. The patient was continued on topical steroid, antibiotic, and IOP lowering drugs.

The patient was examined 2 months later with a vision of CF@2m in the right eye, with retina well attached all around (Figure 6).

Figure 6. RE OCT after RD surgery with silicon oil in situ.



Discussion -

Endophthalmitis in most of the cases is seen after cataract surgery or after intravitreal injections. The incidence of endophthalmitis, as reported in the literature, is 0.07%-0.4% after cataract surgery, and that after intravitreal injection, ranges from 0.04%-0.07%. Endophthalmitis is also common after trauma and after filtration surgeries for glaucoma.

Endophthalmitis after pars plana vitrectomy (PPV) is a rare entity as vitreous is like a nutrient-rich growth medium for microorganisms, and sans the vitreous after PPV, chances of developing endophthalmitis are very bleak.

Being a rare entity, the reporting of such cases in the literature is quite scarce.[1-5]

The factors which are purportedly responsible for causing endophthalmitis post pars plana vitrectomy are as follows[6,7] :

1. Post-surgery leaking wound

Leaking sclerotomy sites after PPV may allow the microorganisms in the conjunctival cul-de-sac to gain access inside the vitreous cavity causing endophthalmitis. The incidence of endophthalmitis increases dramatically, if there is a tag of vitreous exuding from the site of sclerotomy, especially in a wound that has been left unsutured. Microorganisms use this vitreous strand as a scaffold to enter inside the vitreous cavity. This is known as the vitreous wick phenomenon.

2. Intraocular tamponading agents

Air, silicon oil, and expansile gases like C3F8, SF6 do not support the growth of microorganisms. They also seal the sites of sclerotomies well owing to the differential surface tension of these agents.[8] This provides good wound integrity.

The balanced salt solution, on the other hand, does not seal the site of sclerotomy as well as the above-mentioned agents, and therefore its use may facilitate the entry of microorganisms inside the vitreous cavity.

3. Associated pharmacotherapy

The use of subconjunctival antibiotics after PPV has shown to keep the incidence of endophthalmitis after PPV under check. Some centres do not follow this practice and it may be responsible for endophthalmitis after PPV.

Use of intravitreal anti-vegf injections, triamcinolone acetonide, dexamethasone implant concurrently with PPV may also be responsible for causing endophthalmitis following PPV.

4. Surgeon's learning curve

Increased operating time is considered to be one of the causative factors of endophthalmitis post PPV. Therefore, cases being operated by young surgeons might be at a slightly higher risk of developing

endophthalmitis after the surgery. Poorly constructed wounds might also be responsible for leaking wounds post operatively, thereby inviting infections.

5. Diabetes mellitus

Patients with uncontrolled diabetes mellitus and high blood sugar levels are naturally predisposed to develop infections in the post-operative phase. Diabetics are also likely to have a concurrent cataract, which would increase the operative time. There might also be the presence of complex tractional retinal detachment, which would again increase the operating time and would also require complex handling, both of which could lead to increased chances of developing endophthalmitis after the surgery.

Microbiological spectrum

A wide variety of microorganisms have been held responsible for causing endophthalmitis post vitrectomy. These include coagulase-negative staphylococci, ***Pseudomonas***, ***Propioni-bacterium***, enterococci, and ***Bacillus*** species.[9] Coagulase-negative staphylococci is the most common organism causing endophthalmitis after PPV.

Clinical features

Endophthalmitis following PPV, closely resembles endophthalmitis due to any other cause, in signs and symptoms.[10] Patients generally present with acutely painful, red-eye associated with/without watering and mucopurulent discharge. The presence of lid edema strongly raises the suspicion of endophthalmitis. These symptoms are accompanied with blurring/diminution of vision. Patients typically have hypopyon and dense vitritis, with exudation in the vitreous cavity. Although in some cases, the features may not be very marked, or there may be a delayed presentation, due to the absence of vitreous, which acts as a growth medium for microorganisms.

Treatment

Clinicians need to carry a high level of suspicion when a patient presents with symptoms of diminution of vision, pain, redness after surgery. It is always advisable to err towards the diagnosis of endophthalmitis when in doubt. Endophthalmitis should be treated as an ocular emergency.

Intravitreal antibiotic injections need to be administered as soon as possible once the diagnosis is made. Antibiotics most commonly used are Vancomycin(1mg in 0.1ml) and Ceftazidime (2.25mg in 0.1ml). Other broad-spectrum antibiotics(Cefazolin, Amikacin, Moxifloxacin, Imipenem,

Piperacillin/Tazobactam) may also be used. Intravitreal Dexamethasone(0.4mg in 0.1ml) is also given concurrently to counter the inflammation inside the eye.

If based on clinical examination, fungal etiology is suspected, then intravitreal Voriconazole950-100ug in 0.1ml)/Amphotericin-B(5ug in 0.1ml) is injected and the steroid is contraindicated.

Before injecting, a sample from the vitreous cavity should be taken and sent for culture and sensitivity. If media is clear and visibility is good, vitrectomy may be done to clear the bacterial/fungal load as much as possible.

If there is gas/silicon oil in the vitreous cavity, then the sample may be taken from the anterior chamber. Fortified topical antibiotics and oral antibiotics are included in the post-injection regime. Topical steroids may be included if fungal etiology has been ruled out and the cornea is healthy.

The patient needs to be called for frequent follow-up visits (preferably every 3 days) and intravitreal injections need to be repeated as per the need. Antibiotics may be altered as per culture and sensitivity reports.

Prevention

Preoperative asepsis is a must. 10% povidone-iodine should be used to clean the lids and lashes prior to every surgery.[11] A few drops of 5% povidone-iodine solution should be instilled in the conjunctival cul-de-sac and left for a few minutes. This povidone-iodine should be thoroughly washed and then the surgery should be commenced.

This practice is known to reduce ocular flora considerably and has shown to drastically reduce the chances of developing endophthalmitis after surgery. Lid speculum and adhesive surgical drape should be used to keep the eyelashes away from the field of surgery.

Patients who have any signs of ocular/periocular

infection (such as stye, blepharitis, dacryocystitis, etc) should be treated for the infective etiology first and the elective surgery should be taken up only after the infection has completely subsided.

Some surgeons prefer to mix antibiotics in the saline infusion fluid. Although this practice is controversial and not universally followed.

While making sclerotomy, it is advised to displace the conjunctiva with a swab before making an entry with the trocar. This ensures that the conjunctival and scleral entry wounds are not in the same line, which thereby decreases the chances of microorganisms gaining an entry inside the vitreous cavity.

The entry of the trocar should be in a beveled manner and not perpendicular to the sclera. This makes the wound self-sealing and reduces the chances of a leaking wound post-surgery.

After the surgery, the proper closure of the wounds needs to be ensured and the wounds may be sutured if at all there is a doubt of leaking sclerotomy.

Visual outcomes

Visual gain after the treatment of endophthalmitis post-PPV is quite varied. In most of the cases, the gain of vision post-PPV endophthalmitis is quite poor which may also be attributed to the primary retinal pathology.[12,13] As per several study reports, the visual outcomes after post-PPV endophthalmitis are poor as compared to endophthalmitis after cataract surgery.

Conclusion

Endophthalmitis after PPV is a rare entity but it may have grave consequences. It usually carries a very poor prognosis despite aggressive treatment and best efforts from the treating physician. Prevention is the best cure for this entity and every measure needs to be taken to ensure that chances of infection post PPV are minimal.

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Acute visual dysfunction following Phenytoin induced Toxicity

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Abstract

Aim: Acute Visual Dysfunction may be caused by Acute Phenytoin Toxicity

Method: An 18 year old female with prior generalized tonic-clonic seizures developed blurred vision, diffuse corneal opacity (OD), and ankyloblepharon after Phenytoin administration for seizures. Colour vision was found to be normal with Ishihara pseudo-isochromatic charts and visual fields (Humphrey's automated perimeter) show gross concentric constriction in both eyes. Fundus examination revealed increased CD ratio in both eyes. Patient also developed multiple cutaneous maculo-papular lesions and Steven-Johnson syndrome like exfoliation of skin around lips and perioral area. Serum free-phenytoin concentration measured reveals toxic levels of Phenytoin with no other prior co-morbid retinopathy or optic nerve defect.

Results: Phenytoin was withheld, and Leviteracetam administered for seizures and the patient experienced a partial recovery in skin lesions with administration of Prednisolone and in conjunction with reducing serum levels of Phenytoin. Two weeks later the skin lesions have partially subsided, perioral area healed but Bilateral Ankyloblepharon persists and surgical release may be advised.

Conclusion: Phenytoin toxicity may cause acute visual dysfunction as previously unknown phenomenon.

Keywords: Acute phenytoin toxicity, Ocular manifestation, Symblepharon, SJS-Steven Johnson Syndrome.

I. Introduction

Since its discovery in 1908, phenytoin has become one of the well-studied anticonvulsants. With an average monthly cost of $30, it has also become one of the most widely used anticonvulsants, listed on the World Health Organization's List of Essential Medicines. However, with its narrow therapeutic index and its pervasive daily use, considering potential phenytoin overdose or toxicity from chronic use is key to early management and prevention of further toxicity.[1][2][3].

Phenytoin is a commonly prescribed antiepileptic drug. Due to its saturation (zero-order) pharmacokinetics, phenytoin carries a special risk of dose-related toxicity that is an important issue in emergency medicine. Excessive self-medication, misunderstanding of the prescription order, and probable drug interaction were the three leading causes of acute phenytoin intoxication. Unsteady gait, dizziness/vertigo, nausea/vomiting, general weakness, and drowsiness were the most common presenting symptoms. Although acute phenytoin intoxication causes no mortality and has a good outcome, the unsteady gait increases the risk of injuries caused by falls. The management of acute phenytoin intoxication includes temporary withdrawal of phenytoin and supportive care.[4]

We are reporting a case of a patient who received phenytoin therapy for generalized tonic clonic seizures and developed ocular manifestations borne of acute toxicity.

II. Case Study

An 18 year old female presented to the Medicine OPD with prior 3 year history of generalized tonic-clonic seizures under herbal medication for the condition and was prescribed Phenytoin (300mg HS/PO) for the condition, with instructions for regular

follow-up. 15 days later the patient presented in the emergency department with (Figure 1) severe maculo-

Figure 1 : Maculo-popular Rashes over lipe & face

papular rash with crust over face and extremities and perioral lesions with haemorrhagic eruptions over lips and oral ulcers and redness of both eyes and blurring of vision. She was immediately institutionalised for further treatment. She denied any history of prior episode of any such reaction in the past, with any other drug intake with no over the counter prescription and had no vision abnormalities in the recent past. She also denied of any substance abuse in the recent past. Neither she nor her family has any history of diabetes, hypertension and negative history of ocular trauma in the recent past.

On investigation, biochemical parameters show mild eosinophillia (5%) with moderate Anaemia (10.6 g/dL) which improved over 5 days (11.5 g/dL) and slightly raised RDWA (60.6 fL). Liver function tests show raised bilirubin levels(0.3 mg/dl) raised SGPT (74.6 IU/L) and ALP (289 IU/L). Serum Phenytoin levels were 17.0 mcg/ml. Rest of the reports were within normal limits. Based on the patient's medical history, clinical presentations, and lab reports, a diagnosis of Phenytoin toxicity was considered.

Ophthalmic examination revealed uncorrected visual acuity of OD 20/125 (log mar 0.7) and OS 20/60 (log mar 0.5). Slit lamp examination revealed bilateral bulbar conjunctival congestion with diffuse corneal oedema with superficial stromal infiltration, with epithelial defect staining positive with fluoroscein, with bilateral inflammation near lateral Canthi with palpebral conjunctiva adherent to bulbar conjunctiva (Figure 2), and mild anterior Blepharitis in both eyes.

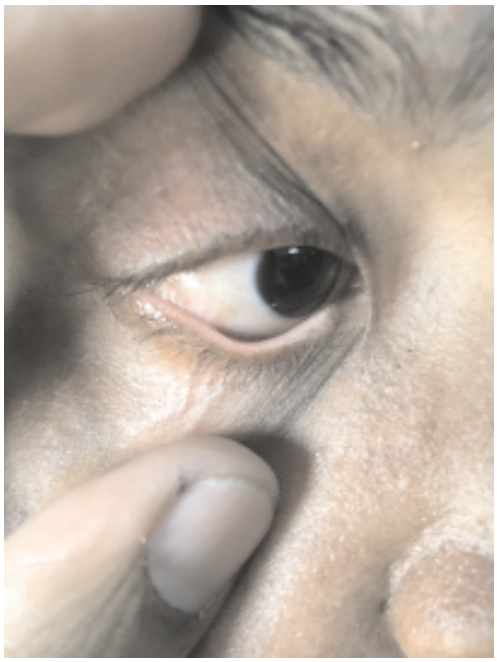

Figure 2 : Palpebral Conjunctiva Adherent at Canthi

Colour vision was found to be normal with Ishihara pseudo-isochromatic charts and visual fields (Humphrey's automated perimeter) show gross concentric constriction in both eyes. Fundus examination revealed increased CD (0.4-0.5) ratio in both eyes with normo-tensive IOP in both eyes (OD-14.6, OS-12.2).

Phenytoin was immediately discontinued and patient was maintained on oral Leviteracetam for seizures, along with supportive care for skin and perioral lesions with I / V Methyl Prednisolone for initial 3 days followed by oral Prednisolone for maintenance. For ocular condition patient was commenced on topical Moxifloxacin 0.5% QID along with CMC gel 1% BD for alleviate FB sensation, and oral Antioxidants, with advice for follow-up after 2 weeks.

After a fort night facial and perioral lesions have subsided, along with disappearance of corneal opacity, along with improvement in ocular symptoms and visual acuity. But the ankyloblepharon has not improved and may need surgical correction once the ocular inflammation has subsided.

III. Discussion

Phenytoin has a narrow therapeutic range of 10-20 mcg/mL.[5,6] At plasma concentrations below 10 mcg/mL, elimination follows first order. However, at higher concentrations, including those in the therapeutic range (10-20 mcg/mL.), the metabolic pathway becomes saturated and elimination shifts to zero order [5]. Half life of phenytoin varies between six and twenty four hours at plasma concentration less than 10 mcg/ml, but increases with higher concentrations[5,7]. As a result, plasma concentration rises disproportionally even with small increase in dose [8][6]. Toxicity generally correlates with the increasing plasma levels. The increased half life due to zero order pharmacokinetics can also result in prolonged duration of toxic symptoms[9].

The toxic effects seen with chronic treatment are primarily dose related cerebellar-vestibular effects.3 It may also cause other central nervous system effects, behavioural changes, increased seizure activity, gastrointestinal symptoms, hirsutism, gingival hyperplasia, osteomalacia and megaloblastic anaemia[7,8,6]. Chronic phenytoin ingestion leads to its accumulation in the cerebral cortex, resulting in atrophy of cerebellum, causing ataxia and nystagmus[10]. Signs of phenytoin toxicity usually manifest at phenytoin levels above 15 mcg/mL[11]. Our patient had serum phenytoin levels of 17.0 mcg/ml. Previous studies point out that phenytoin toxicity may develop over months to year after starting the drug[12]. These effects can be reversed by withdrawing or reducing the dose of Phenytoin[7,13].

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Study on Management of Ptosis by Different Surgical Modalities

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Abstract

Background

Ptosis is derived from the greek word for falling and is the medical terminology describing a drooping or abnormal lowering of an anatomical area. Ptosis that obstruct the pupil may interfere with the normal development of vision, resulting in amblyopia in children[2] . In adult it may impair the field of vision and interfere with activities of daily living Ptosis is broadly classified into congenital and acquired, based on age of onset of the ptosis. Ptosis that is present at birth or within the first year of life is called congenital ptosis. Ptosis that presents after the age of one year is termed acquired ptosis. The treatment of ptosis depends upon the underlaying etiology. Ptosis usually does not improve over time and nearly always require corrective surgery. Depending upon the severity of congenital ptosis, patients should be monitored every 3-12 months for sign of amblyopia due to congenital ptosis. In mild cases of congenital ptosis observation is sufficient, if no sign of amblyopia, strabismus and abnormal head posture are present.

Methods

This study was conducted in the Upgraded Department of Ophthalmology, LLRM Medical College, Meerut during 2018–2019.

Study design-A Population based Prospective interventional study was done on patients selected from OPD and camps during 2018-2019.

Plan & Work All patients with ptosis who are attending ophthalmology OPD and admitted in eye wards were included in the study provided they fulfill the inclusion criteria.

Results And Conclusion

The subjects in our study were more males 12(66.66%) than females(33.33%). In our study there were 12(66.66%) patients of myogenic ptosis, out of them 11(61.11%) had frontalis sling surgery and 1(5.55%) had levator resection surgery.

There was 2(11.11%) cases of neurogenic ptosis which was congenital in nature and in which 1(5.55%) is operated with frontalis sling &1(5.55%) with levator resection surgery. Most commonly performed surgery was frontalis sling 14(77.77%) followed by levator resection 2(11.11%) and 2(11.11%).

Key Words:

MRD-Marginal Reflex Distance, ePTFE-Poly Tetra Fluoro Ethylene, B/L-Bilateral, U/L-Unilateral, FS-, Fasanella Servat, FSling-Frontalis Sling, LR-Levator Resection.

Background

Ptosis is derived from the greek word for falling and is the medical terminology describing a drooping or abnormal lowering of an anatomical area.

It referes to the unilateral or bilateral drooping of the upper eyelid. It usually occurs from a partial or complete dysfunction of the muscles that elevate the upper eyelid: the levator palpabrae superioris and the muller's muscle[1].

Ptosis that obstruct the pupil may interfere with the normal development of vision, resulting in amblyopia

in children[2] . In adult it may impair the field of vision and interfere with activities of daily living[2-3].

Types of ptosis

Ptosis is broadly classified into congenital and acquired, based on age of onset of the ptosis. Ptosis that is present at birth or within the first year of life is called congenital ptosis. Ptosis that presents after the age of one year is termed acquired ptosis.

Ptosis may also be classified by etiology:

- Congenital
- Neurogenic ptosis which includes oculomotor nerve palsy, Horner's syndrome, Marcus Gun jaw winking syndrome, third cranial nerve misdirection.
- Myogenic ptosis which includes oculopharyngeal muscular dystrophy, myasthenia gravis, myotonic dystrophy, ocular myopathy, simple congenital ptosis, blepharophimosis syndrome.
- Aponeurotic ptosis which may be involutional or post operative.
- Mechanical ptosis which occurs due to edema or tumors of the upper lid.
- Neurotoxic ptosis which is a classic symptom of envenomation[4] by elapid snakes such as cobras[5], kraits, mambas and taipans.
- **Pseudoptosis due to:**

1. Lack of lid support: empty socket or atrophic globe.
2. Higher lid position on the other side: as in lid retraction.

Ptosis that obstruct the pupil may interfere with the normal development of vision , results in amblyopia in children. In adults it may impair the field of vision & interfere with activities of daily living. Thus the early diagnosis & treatment of ptosis is an important prognostic factor in its management.

Mortality/Morbidity

If congenital ptosis obscures any part of the paediatric patient's visual field, surgery must be performed to correct the problem early in life. Otherwise,a permanent loss of vision may occur as a result of amblyopia.

History

a. The onset of ptosis
b. Alleviating or aggravating factors
c. Family history of ptosis
d. History of trauma or ocular surgery are important clues to the etiology.
e. Family photographs can help determine onset or variability of the ptosis.
f. Providing photographs also gives the surgeon a chance to examine other family members. A patient with a strong family history of congenital ptosis may not need an extensive workup.
g. A history of fluctuating ptosis with strabismus may indicate myaesthenia gravis.A recent report has also suggested the development of myasthenia like syndrome resulting in ptosis with the use of inhibitors of 3- hydroxy -3- methyl-glutaryl-COA reductase as statins.They are used in the treatment of hypercholesterolemia33[6].
h. Metastatic or primary orbital tumors can result in malpositioning of the eyelid. A history of trauma with orbital wall fractures can result in pseudoptosis with enophthalmos. Additionally, third cranial nerve palsy from trauma may result in ptosis.
i. A history of difference in the size of the pupil may be helpful in diagnosing Horner syndrome. Patients with Horner syndrome have ptosis and miosis on the same side.
j. A detailed history of present illness includes asking about the onset, duration, variability, progression and severity of ptosis. Also investigate whether there is involvement of one eye or both eyes simultaneously.

Symptoms

Most patients presents with drooping eyelids, giving a sleepy or tired appearance.

Undiagnosed congenital ptosis may result in amblyopia.

On inspection, the patients speacialy children may assume a head tilt backwords and chin up position.

Signs

A comprehensive ophthalmic examination should be done in all cases.

The examination begins with a carefull external examination along with palpation of the eyelid and the orbital rim. Evaluate any clinical evidence of relative proptosiss or enophthalmos in each eye.

The patients head posture should be noted, document chin up position.

Eyelid measurement

To quantify the severity of ptosis, various eyelid measurements should be taken with the face held in the frontal plane and with frontalis

muscle relaxed.

Palpebrae fissure height(PF)is the distance between upper and lower eyelid margins at the axis of the pupil. Normal measurment is 9-12mm.

Marginal reflex distance (MRD-1) is the distance between the central corneal light reflex and upper eyelid margin with eyes in primary position while MRD-2 is the distance between the central corneal light

reflex and lower eyelid margin.

Levator function should be evaluated in all cases.

Other tests

Corneal sensitivity should be tested in all cases.

Bell's phenomenon should be tested to evaluate the risk of exposure keratopathy if surgery is being planned.

Jaw winking phenomenon a brisk upper eyelid retraction will be elicited when the patients is asked to open and close his/her eyes with protrusion or lateral movement of the jaw.

Treatment

The treatment of ptosis depends upon the underlaying etiology.

Ptosis usually does not improve over time and nearly always require corrective surgery.

Depending upon the severity of congenital ptosis , patients should be monitored every 3-12 months for sign of amblyopia due to congenital ptosis.

In mild cases of congenital ptosis observation is sufficient , if no sign of amblyopia, strabismus and abnormal head posture are present.

If the child has a strabismus together with ptosis surgery to correct strabismus is usually done prior to ptosis surgery.

Non surgical treatments also includes[7]

1. Lid crutches used to support a drooping lid mechanically.
2. Haptic contact lens with a shelf on which the margin of upper lid rests may be used.
3. Elevation of the lid by a mechanical force : a strip of highly magnetically metal is implanted in the upper lid and a magnet is placed behind the upper rim of frame.

Surgical care

Correction of ptosis in a child may often be delayed untill the patient is 7 year old, although consistent child up positions/complete ptosis may justify early surgery[8]

The method of repair depends on treatment goals, the underlying diagnosis, and the degree of levator function.

Surgical correction of ptosis can be undertaken at any age depending on the severity of the disease. Earlier intervention may be required if significant amblyopia or ocular torticollis is present.If intervention is not urgent, surgery is often delayed until age 3-4 years. Waiting until this age allows for more accurate measurements preoperatively.

1. Levator muscle resection

This procedure is the shortening of the levator-aponeurosis complex through a lid-crease incision. The skin incision is hidden either in the existing lid fold or in a new lid fold created to match that of the contralateral eyelid.

If the levator function is greater than 4 mm but less than 6 mm,a levator resection of greater than or equal to 22 mm is recommended. If the levator function is 6-8 mm, a levator resection of 16 -18 mm is indicated. If the levator function is greater than 8 mm, a levator resection of 10-13 mm is indicated.

Contraindications: An external levator resection is not indicated when the levator function is less than 4 mm. In such cases, a long-term surgical outcome may result in undercorrection. Poor Bell phenomenon reduced corneal sensitivity, or poor tear production can produce exposure keratopathy.

2. Frontalis suspension procedure

This procedure is designed to augment the patient's

lid elevation through brow elevation. Frontalis suspension procedures produce lagophthalmos in most cases. Some surgeons prefer to perform a bilateral suspension procedure for severe unilateral congenital ptosis to obtain symmetry.

The procedure is indicated when the levator function is less than 4 mm. Relative contraindications are poor Bell phenomenon, reduced corneal sensitivity, or poor tear production, which can produce exposure keratopathy.If surgery is still indicated, these patients need close postoperative follow-up care to avoid corneal exposure,infection,corneal ulcer and amblyopia.

Several materials are available to secure the lids to the frontalis muscles.These materials include the following:

- Autogenous fascia lata: Autogenous fascia lata can be obtained from the leg of patients older than 3 years .
- Preserved (tissue bank) fascia lata
- Nonabsorbable suture material (eg, 2-0 Prolene, Nylon (Supramid) or Mersilene)Silicone bands, silicone rods ePTFE.
- Autogenous materials used less frequently include palmaris longus

  tendon and temporalis fascia[9-12].

3. Fasanella-Servat procedure

The upper lid is elevated by removing a block of tissue from the underside of the lid. This tissue includes the tarsus, conjunctiva, and Muller muscle. This procedure is not commonly performed for cases of congenital ptosis.

Further Outpatient Care

Patients who underwent surgery for ptosis are initially monitored every 2-4 weeks for signs of exposure keratopathy, infection, granuloma formation, and overcorrection and undercorrection.

Following the surgery, visual acuity, head posture, and refractive error should be carefully monitored.

Complications

Complications associated with the frontalis suspension procedure for congenital ptosis repair include the following:Granuloma, Lid asymmetry, Overcorrection with exposure keratopathy and dry eyes.

Undercorrection: Suspension materials may dissolve or break.

Methods

This study was conducted in the Upgraded Department of Ophthalmology, LLRM Medical College, Meerut during 2018–2019.

Study design-A Population based Prospective interventional study was done on patients selected from OPD and camps during 2018-2019.

Plan & Work

All patients with ptosis who are attending ophthalmology OPD and admitted in eye wards were included in the study provided they fulfill the inclusion criteria.

Inclusion Criteria:

- All age groups
- All types of ptosis(congenital and acquired)
- Patient who gives written informed consent for examination, treatment and surgery.
- Patient with different degree of levator action
- Mentally and physically fit up to a minimum level required to participate in study

Exclusion criteria:

- Not interested/unable to provide informed consent.
- Patient with uncontrolled systemic illnessses
- Patients with myasthenia gravis
- Patient not fit for general anaesthesia

All patients with ptosis were evaluated on the basis of detailed history seeking symptoms of disease itself and its complications such as history of drooping of eyelid, decrease in vision, amblyopia, fever, ocular, nasal, facial surgery recurrent infection or trauma in the past.After taking detailed history general physical examination of the patient was done.

After general examination ocular examination was done which includes

1. Recording of vision
2. Papillary reaction
3. Vertical palpebral fissure height
4. Action of levator palpabrae superioris
5. Marginal reflex distance 1& 2

6. Lid crease height
7. Bell's phenomenon
8. Corneal sensations
9. Schirmer's test

The cases were managed by standard ptosis correction surgical procedures in various describe indications.

Results

The subjects in our study were more males 12(66.66%) than females(33.33%).

Most subjects were in the age group of 4-21years.

Congenital ptosis 14(77.77%) was found to be most common types of ptosis. Most of the cases of congenital ptosis were myogenic 12(66.66%). Amongst the acquired cases of ptosis aponeurotic 4(22.22%) were the most common.

In our study there were 12(66.66%) patients of myogenic ptosis, out of them 11(61.11%) had frontalis sling surgery and 1(5.55%) had levator resection surgery.

There was 2(11.11%) cases of neurogenic ptosis which was congenital in nature and in which 1(5.55%) is operated with frontalis sling &1(5.55%) with levator resection surgery.

Of all the cases of aponeurotic ptosis -4(22.22%)were acquired in nature . 2 patients were operated using frontalis sling and 2 patients were fasanella servat operation.

Most commonly performed surgery was frontalis sling 14(77.77%) followed by levator resection 2(11.11%) and 2(11.11%).

No mechanical or traumatiic case was found in our study.

Complication were seen in 3(16.66%) cases but they could be managed by post operatively in six months follow up.

Complication like overcorrection 2(11.11%) and granuloma formation in 1(5.55%) were seen. Over correction was resolved after follow up in 6 months and granuloma formation was corrected after repeat surgery. Other complication like infection , lid assymmetry were not seen.

There was significant cosmetic improvement in the drooping of eyelids attributable to the surgery of ptosis and no progression of amblyopia in congenital cases.

Conclusion

The subjects in our study were more males than females.Congenital ptosis was found to be most common types of ptosis. Most of the cases of congenital ptosis were myogenic. Amongst the acquired cases of ptosis aponeurotic were the most common.Most of the myogenic ptosis had frontalis sling surgery and had levator resection surgery.All the cases of aponeurotic ptosis were acquired in nature,some patients were operated using frontalis sling and some were fasanella servat operation.Most commonly performed surgery was frontalis sling followed by levator resection and fasanella servat operation.

There was significant cosmetic improvement in the drooping of eyelids attributable to the surgery of ptosis and no progression of amblyopia in congenital cases.

Table 1 : Unilateral or Bilateral

	U/L	U/L(%)	B/L	B/L(%)
Congenital	12	66.66%	2	11.11%
Acquired	0	0%	4	22.22%
Total	**12**	**88.88%**	**6**	**11.11%**

Table 2 : Choice of operation

Pre op amount of ptosis	Levator function	Type of operation
Severe	</= 4mm	F Sling
Moderate	5-10mm	LR
Mild	>=11	F servat

Table 3 : Degree of ptosis & Surgery

S. No	Type		No of pt/%	Sx		
				F. Sling	F. servat	L.R.
1	Myogenic	Mild	0(0.00%)	0(0.00%)	0(0.00%)	0(0.00%)
		Moderate	2(11.11%)	1(5.55%)	0(0.00%)	1(5.55%)
		Severe	10(55.55%)	10(55.55%)	0(0.00%)	0(0.00%)
2	Neurogenic	Mild	0(0.00%)	0(0.00%)	0(0.00%)	0(0.00%)
		Moderate	2(11.11%)	1(5.55%)	0(0.00%)	1(5.55%)
		Severe	0(0.00%)	0(0.00%)	0(0.00%)	0(0.00%)
3	Aponeurotic	Mild	1(5.55%)	0(0.00%)	1(5.55%)	0(0.00%)
		Moderate	2(11.11%)	1(5.55%)	1(5.55%)	0(0.00%)
		Severe	1(5.55%)	1(5.55%)	0(0.00%)	0(0.00%)
4	Mechanical	Mild	0(0.00%)	0(0.00%)	0(0.00%)	0(0.00%)
		Moderate	0(0.00%)	0(0.00%)	0(0.00%)	0(0.00%)
		Severe	0(0.00%)	0(0.00%)	0(0.00%)	0(0.00%)
	Total		**18(100%)**	**14(77.77%)**	**2(11.11%)**	**2(11.11%)**

Table 4 : Types of operation & success rate

Type of Op.	Success rate					
	Good		Fair		Poor	
F Sling	No.	%	No.	%	No.	%
	10	55.55%	4	22.22%	0	0.00%
LR	2	11.11%	0	0.00%	0	0.00%
F Servat	2	11.11%	0	0.00%	0	0.00%
Total	**14**	**77.77%**	**4**	**22.22%**	**0**	**0.00%**

Table 5 : Complication of ptosis surgery

S. No	Type of operation	No of patients%	Complication
1	F Sling	3(16.66%)	Granuloma-1 Overcorrection-2
2	F servat	0(0.00%)	0
3	LR	0(0.00%)	0
	Total	**3**	

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Focal Dissection of Internal Carotid Artery Presenting as Horner's Syndrome : A Case Report

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Introduction

One of the most common cause of stroke in young adults is internal carotid artery dissection.[1] Patients with carotid artery dissection often present with a partial Horner's syndrome and have a high prevalence of fibromuscular dysplasia.[2] Patients with dissection-related carotid artery occlusion have a significantly increased risk of suffering a disabling stroke.[2] It is important to diagnose dissection as anticoagulation can prevent carotid thrombosis and embolism.[3]

Aim

To report internal carotid artery dissection presenting as Horner's syndrome

Case report

A 46 year old male presented with burning sensation on the right side of face with a complaint of decrease in vision in the right eye for 2 weeks. There was no history of decreased sweating in one half of the face. There was no weakness or decreased sensation in face or any limbs. There was no history of trauma. He was a known case of diabetes and hypercholesterolemia. On examination his best corrected visual acuity (by Snellen chart) was 6/6, N6 OU, on hirschberg test he was found to be orthophoric, on cover test there was no phoria. There was no head posturing or face turn .Extra ocular movements were full in both eyes. Lid examination revealed mild ptosis in the right eye. Palpebral fissure height in OD was 10 mm and OS 12 mm (as shown in Fig 1 and 2). Pupillary evaluation showed an anisocoria of 2 mm with right eye pupil smaller than the left eye pupil, anisocoria was more in dim light compared to the bright illumination (Figure 3 and 4) Both the pupils were briskly reacting to light

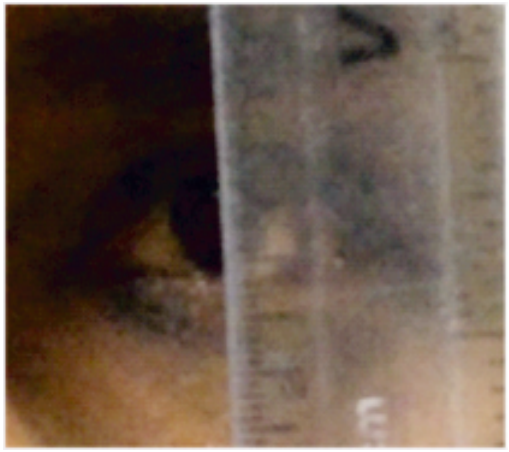

Fig. 1: Right eye palpebral fissure height -10 mm

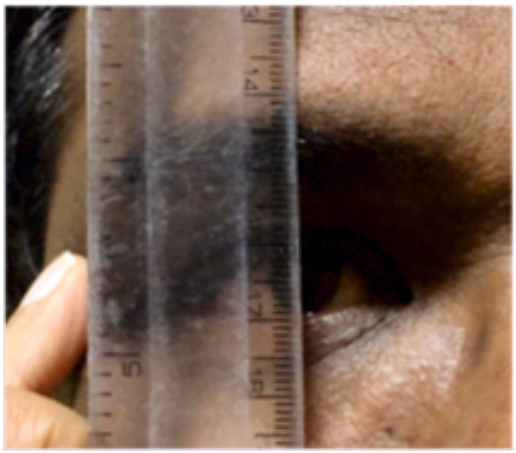

Fig. 2: Left eye palpebral fissure height – 12 mm

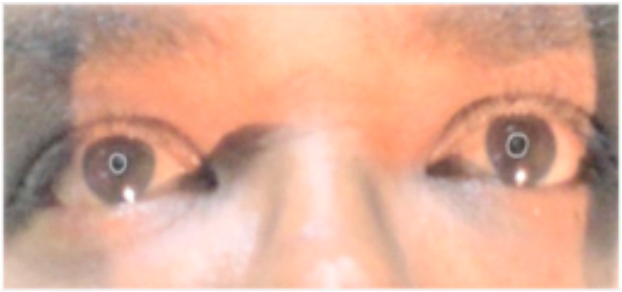

Fig. 3: Pupil size in bright illumination

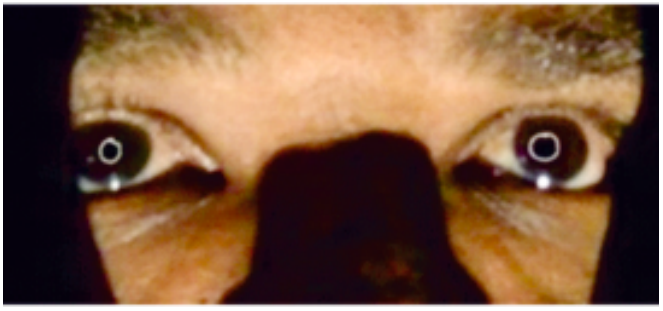

Fig. 4: Pupil size in dim illumination

with no relative afferent pupillary defect in either eyes. There was a mild decrease in the corneal sensation in the right eye. Rest of the anterior segment examination and intra ocular pressure as tested by applanation tonometry was within normal limits in both the eyes. Fundus examination was within normal limits in both the eyes. A diagnosis of right side Horner's syndrome was considered as the patient had the classic triad of the right eye mild ptosis, miosis and mild apparent enophthalmos though he did not have anhidrosis.

Management: In view of sudden onset incomplete painful Horner's syndrome an magnetic resonance imaging (MRI) of brain and orbit along with magnetic resonance angiogram (MRA) was advised.MRA showed a focal dissection of the distal cervical internal carotid artery (Fig. 5) The patient was referred to an

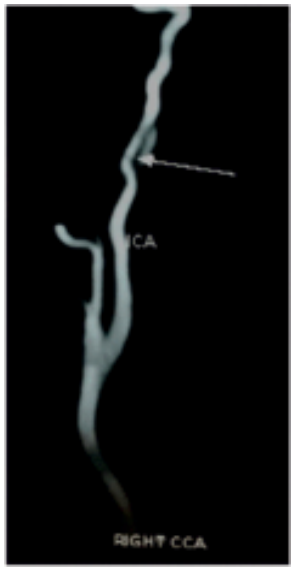

Fig. 5: MRA showing focal dissection of the distal part of the ICA.

interventional radiologist who started the patient on oral antiplatelet agents. At 2 weeks of follow up, the patient was asymptomatic and when a repeat MRA was done the focal dissection had resolved.At 1 month follow up, patient was still asymptomatic for any neurological complications.

Discussion:

The various ophthalmological manifestations of internal carotid artery dissection described in the literature (in order of the frequency) are painful Horner syndrome, transient monocular visual loss, ischemic optic neuropathy ,ophthalmic artery occlusion ,central retinal artery occlusion, ischemic ocular syndrome and oculomotor nerve palsies.[4]Two third of patients with ICA dissection have ophthalmologic symptoms or signs, which are the presenting features of the dissection in more than half of such cases.[4]Horner's syndrome is secondary to the damage to the sympathetic pathway distal to the superior cervical ganglion. Pain is presumed to be the result of ischemia (through the vasa nervorum) or compression (by the enlarged occluded carotid) of the pericarotid sympathetic fibers.[1]It is a referred pain to the orbital area originating from the internal carotid artery area. Pain has been reported in up to 90% of cases with ICA dissections. Such pain involves the head in 70% of cases, the neck in 20%, and/or the face in 10%.[4] According to this 1998 study looking at the complete spectrum of ophthalmic manifestation of the ICA dissection, ophthalmologic symptoms or signs preceded a non-reversible ocular or cerebral infarction in one third of patients, with the majority having a stroke within the first 2 weeks after the onset of ophthalmologic symptoms or signs.[4]This suggests that any potential preventive treatment should be initiated as early as possible after the onset of the first symptoms, and treatment initiation up to 1 month after the onset of symptoms have shown to be beneficial.[5]The ophthalmic signs and symptoms are not specific for ICA dissection and are also seen with carotid diseases like atheroma.[6]But, such symptoms occurring in a young patient with associated pain

should alert an ophthalmologist for early imaging and referral.

Conclusion:

Sudden onset painful Horner's syndrome needs an urgent radiological evaluation to avert a stroke.

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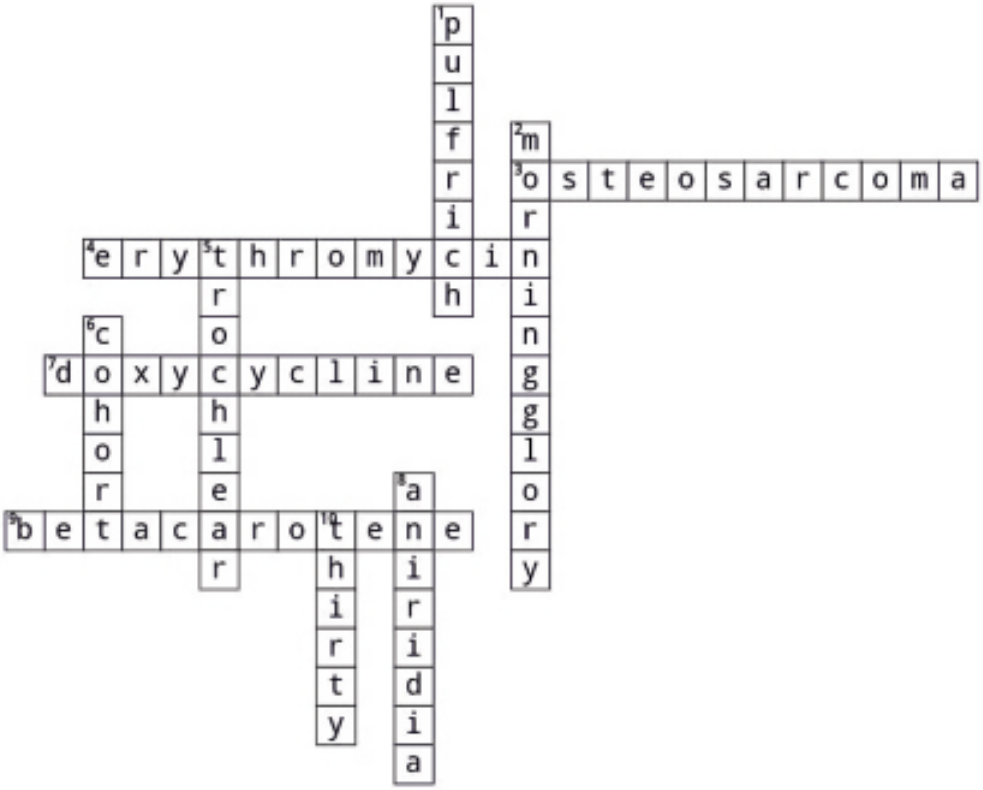
Puzzle Answer



A review of Rhino-orbitocerebral mucormycosis IN COVID

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Abstract

This is a review article. We have collected data from the pubmed, through the various articles published since the inception of COVID-19 in 2019 to October 2021. Rhino-orbital-mucormycosis (ROCM) is increasingly reported in COVID 19 patients, either during or after the recovery from the disease. It is a fulminating infection involving nasal mucosa, paranasal sinuses, further involving orbit and the brain. The major underlying pathology is the immunocompromised status of the patient and aggressive nature of the fungus. The patients present with spectrum of signs and symptoms depending on the stage of involvement. The diagnosis can be done by various microbiological tests and the treatment depends upon the stage of the disease. The mainstay of treatment involves reversal of the patient's immunocompromised state, aggressive treatment with systemic antifungals and surgical debridement. The prognosis is usually grave if diagnosis is delayed. We are still learning about the ROCM in COVID19, and have summarized the available information about the disease.'

Introduction

Mucormycosis (zygomycosis) is a known invasive fungal infection, often acute and extremely severe caused by opportunist and ubiquitous fungi belonging to class phycomycetes, subclass zygomycetes, orderMucorales, family mucoraceae. It is usuallycaused by following species- Absidiacorymbifera, Apophysomyces elegans, Cunning hamellaber tholettieae, Mucor rouxii, Rhizomucorpussillus, Rhizopus arrhizus and by species of genus saksenaea species. [1]

It is acquired by establishment or implantation of fungal spores in the oral, nasal and conjunctival mucosa (Rhino-orbito-cerebral), by inhalation (pulmonary), or by the ingestion of contaminated food (digestive). [1]

As they colonize in nutrients rich in simple carbohydrates being glucose its main energy source. [1]

Mucormycosis is an angioinvasive infection; diabetes mellitus is being reported as most common underlying condition and an independent risk factor of Rhino-orbito-cerebral mucormycosis, being Rhizopus species is the most common cause. [1]

Rhino-orbito-cerebral mucormycosis (ROCM) typically originates in the nasal or oral mucosa, spread to the paranasal sinus and enters the orbit via the ethmoid and maxillary sinuses or via the nasolacrimal duct. Intracerebral extension may occur from the orbit via orbital apex, orbital vessels or via the cribriform plate, carotid artery or possibly via a perineural route. [2]The fungus is angioinvasive and exhibits a remarkable affinity for arteries and grows along internal elastic lamina causing thrombosis and infarction. [3]

The pathogenic mechanisms implicated in the fungal aggressiveness are the decrease of phagocytic functions, ketoacidosis in diabetes offers advantage to this fungal invasion, acidicmilieu reduce binding of iron to transferrin, more available iron due to displacement of protons by transferrin in diabetic ketoacidosis and fungal heme oxygenase which facilitates iron uptake for its metabolism[1], also lack of a dialysable inhibitory factor in patients with diabetes offer favourable conditions for fungal multiplication.[2]

Mucorales have a ketone reductase enzyme, they thrive in hyperglycemia and diabetic ketoacidosis states associated with poor prognosis.[4]

Other common risk factors are immunosuppressive therapy, leukemia, neutropenias , patients with neutrophil dysfunction , hematopoietic stem cell transplantation , diabetic ketoacidosis, iron overload and HIV/AIDS are some identifiable risk factors.[3]

Rhino-orbital involvement is a time sensitive condition that must be recognized and treated promptly to avoid morbidity and mortality. [4]

Challenges in treating Rhino-orbito-cerebral mucormycosis is due in part to its underlying pathogenesis in which endothelial cell damage lead to vascular thrombosis decreasing the efficacy of systemic antifungal medication.[5]

Covid

Corona virus disease 2019(COVID-19) is a new disease caused by a novel corona virus (SARS-COV-2) that was first documented in china in December 2019 and has grown into a worldwide pandemic.[6] Pandemic was officially declared by World health organisation (WHO) on march ,11 ,2020.[7]

The severity of disease ranges from asymptomatic infection to respiratory failure and death.[8] It may progress to acute respiratory distress syndrome (ARDS), a condition that increases the susceptibility of pulmonary fungal coinfections.[9] Severe COVID-19 is associated with immune dysregulation affecting both T-helper cell(Th2) and Th1 responses, including the cytokine release syndrome, which contribute to lung pathology and promote pulmonary microbial proliferation and a subsequent infection.[7]Critically ill covid-19 patients have higher pro-inflammatory (IL1,IL2,IL6,Tumor necrosis alpha) and anti-inflammatory (IL4,IL10) cytokine levels, less CD4 interferon-gamma expression and fewer CD4 and CD8 cells. The immune dysregulation and altered cytokine profile increase the risk of invasive fungal infections (IFI),such as invasive pulmonary aspergillosis(IPA),rhino-orbital cerebral mucormycosis, invasive candidiasis(IC), or pneumocystis jirovecii pneumonia(PJP).[7]

Drugs like methylprednisolone and dexamethasone are believed to modulate inflammation mediated lung injury and thereby reduce progression of respiratory failure in COVID-19.There side effects include increased secondary infections, immune modulation, manifestation of latent diabetes mellitus, dizziness, weight gain, mood changes, insomnia, and muscle weakness.[2]

Although COVID-19 primarily affects the lungs, different disease complications affecting the whole body such as myocardial injury, arrythmia, thromboembolic events and immune dysregulation are reported. [4]

Rhino-orbitocerebral Mucormycosis More In Covid,why?

The second wave of the COVID-19 pandemic in India documented an increase in mucormycosis especially ROCM [10].Critically ill COVID-19 patients are candidates are at a high risk for ROCM and other fungal infections. [9]Most people affected by COVID-19 are old and have other predisposing conditions like type 2 diabetes mellitus.[4]In addition to these patients frequently receive broad spectrum antibiotics and corticosteroids.[4]Also, they are supported by invasive or non-invasive ventilation due to severe ARDS.[4]

Immune dysregulation associated with COVID-19 with reduced number of CD4+T and CD8+T cells, may alter innate immunity. [11] The risk of hospital acquired infections and systemic immune alterations of COVID-19 infection may lead to secondary fungal infections. [11]

On contrary, Clinical evidence suggests that the neutrophils monocytes and macrophages which play a predominant role in the primary host defence against Muckrakes are unaffected in COVID-19 infection, thus eliminating their role in the pathogenesis [12]. On the contrary, an increase in peripheral neutrophil number was noted in COVID-19 with an increased neutrophil lymphocyte ratio [13]. This is in fact beneficial as far as

immunity toward Mucorales is concerned. These neutrophils are very effective and readily inactivate the fungus by the generation of oxidative metabolites if the host is immunocompetent. Analyzing the existing literature lymphopenia seems to be the only significant immune cell defect detected in COVID-19 [12]. However, lymphopenia does not play any significant role in increasing the host susceptibility to Mucorales. Clinically, this can be explained by the lower incidence of mucormycosis in HIV-infected patients and other lymphopenic syndromes. A retrospective study has shown only 2 cases of mucormycosis in autopsy of 1630 patients died of AIDS-related complications from 1984 to 2002, signifying the rarity of incidence [14].

A complex interplay of these multiple factors is probably responsible for increased incidence of ROCM. [11]

It is not necessary that only long term use of corticosteroids in covid patients leads to fungal infection many case reports recently proved that short course of steroids therapy causes mucormycosis[15] especially in people with DM[16,17].

HOW DO THESE PATIENTS PRESENT? [4, 18]

Signs and symptoms of ROCM depend on the stage of the disease at presentation. Honavar SG *et al* [18] have proposed a detailed classification for ROCM. (Table 1)

Proposed staging of RHINO-ORBITO-CEREBRAL MUCORMYCOSIS (ROCM) (Table - 1) [18]

Stage 1	**Involvement of the nasal mucosa**
1a	Limited to the middle turbinate
1b	Involvement of the inferior turbinate or ostium of the nasolacrimal duct
1c	Involvement of the nasal septum
1d	Bilateral nasal mucosal involvement
Stage 2	**Involvement of paranasal sinuses**
2a	One sinus
2b	Two ipsilateral sinuses
2c	>Two ipsilateral sinuses and/or palate/oral cavity
2d	Bilateral paranasal sinuses involvement or involvement of zygoma or mandible
Stage 3	**Involvement of orbit**
3a	Nasolacrimal duct, medial orbit, vision unaffected
3b	Diffuse orbital involvement(>1 quadrant or >2 structures), vision unaffected.
3c	Central retinal artery or ophthalmic artery occlusion or superior ophthalmic vein thrombosis; involvement of superior orbital fissure, inferior orbital fissure, orbital apex, loss of vision
3d	Bilateral orbital involvement
Stage 4	**Involvement of CNS**
4a	Focal or partial cavernous sinus involvement and/or involvement of the cribriform plate
4b	Diffuse cavernous sinus involvement and/or cavernous sinus thrombosis
4c	Involvement beyond the cavernous sinus, involvement of skull base, internal carotid artery occlusion, brain infarction
4d	Multifocal or diffuse CNS disease

If only nasal mucosal involvement i.e. patient is in stage-1 of ROCM, the presenting symptoms are nasal discharge, nasal stuffiness, foul smell, epistaxis and signs are like foul smelling sticky mucoid or black tinged or granular or hemorrhagic nasal discharge, nasal mucosal inflammation, erythema, violaceous or blue discolouration, pale ulcer, anaesthesia, ischemia and/or eschar formation.

When there is paranasal sinus involvement i.e. disease is in stage-2, the patient presents with signs and symptoms of stage-1 plus symptoms like-facial pain, facial edema, dental pain, systemic symptoms(fever, malaise) and signs include unilateral or bilateral localises or diffuse facial edema, edema localised over sinuses, localised sinus tenderness.

When there is orbital involvement i.e. disease is in stage-3, patient may also have pain in eye, proptosis, ptosis, diplopia, loss of vision, infraorbital and facial V1 and V2 nerve anaesthesia and signs include conjunctival chemosis, isolated ocular motility restriction ,ptosis, proptosis ,infraorbital nerve anaesthesia ,central retinal artery occlusion , features of ophthalmic artery occlusion and superior ophthalmic vein thrombosis.

Ophthalmic (V1) and maxillary (V2) nerve anaesthesia and features of 3, 4, 6 nerve palsy indicating orbital apex/superior orbital fissure syndrome may be seen in some patients.

Involvement of the CNS occurs most frequently (70%) due to contiguous spread from the paranasal sinuses and orbits. The remaining 30% are divided equally between isolated CNS infection (usually in intravenous drug injectors) and hematogenous spread from distant sites of infection. Intracranial fungal granuloma is a distinct clinical entity, with most cases to date reported from India. About half the cases are associated with fungal sinusitis, and half appear as isolated intracranial infections with no clinically apparent sinus disease. Aspergillus spp. are the most frequently recovered organisms, followed by Mucorales. Imaging (CT scan and MRI) shows multiple tumour-like masses with faint contrast enhancement and surrounding parenchymal edema. The frontal lobes are the most frequent location. Involvement of central nervous system causes bilateral proptosis, paralysis, altered consciousness, focal seizures and signs include V1 and V2 nerve anaesthesia, ptosis and features of 3,4,6 nerve palsy indicate cavernous sinus involvement. Bilaterality of these signs with contralateral orbital edema with no clinico-radiological evidence of paranasal sinus or orbital involvement on the contralateral side indicates cavernous sinus thrombosis.

Hemiparesis, altered consciousness and focal seizures indicate brain invasion and infarction.

DIAGNOSIS

The clinical diagnosis of mucormycosis by Smith and Krichner[19] criteria include:

(i) Black, necrotic turbinate's

(ii) Blood-tinged nasal discharge and facial pain, both on the same side,

(iii) Soft peri-orbital or peri-nasal swelling with discoloration and induration,

(iv) Ptosis of the eyelid, proptosis of the eyeball and complete ophthalmoplegia and,

(v) Multiple cranial nerve palsies unrelated to documented lesions.

The diagnosis can be done by microbiological examination of nasal mucosal biopsy and imaging. [4, 18]

Nasal endoscopy

Deep or endoscopy guided nasal swab, paranasal sinus or orbital specimen is collected. Direct microscopy of the sample using KOH mount and calcofluor white shows aseptate ribbon like hyphae, wide angle of non- dichotomous branching (more than or equal to 45-90^0) and greater hyphal diameter (6-25 micrometer).Direct microscopy has 90% sensitivity.

The sample can also be cultured on brain heart infusion agar, potato dextrose agar, or sabouraud dextrose agar with gentamicin or chloramphenicol and polymyxin B but without chlorhexidine, incubated at 30-37^{0}C.This is strongly recommended andcan help in genus and species identification and antifungal susceptibility testing.Rapid growth of fluffy white, grey or brown cotton colonies can be seen. The sample can also be used for molecular diagnosis by

quantitative polymerase chain reaction(75% sensitivity) and can be used for diagnosis confirmation.

Histopathology of the sample is done with haematoxylin-eosin, Periodic-acid-Schiff and Grocott-gomori'sMethanamine-silver special stain. Hyphae showing tissue invasion is confirmatory of invasive ROCM.[13]

Imaging – Contrast enhanced MRI preferred over CT scan

Nasal and paranasal sinus mucosal thickening with irregular patchy enhancement is an early sign of ROCM. Also, Ischemia and non-enhancement of turbinates manifests as an early sentinel sign on MRI-described as the Black turbinate sign. The fluid level in sinus and partial or complete sinus opacification signifies advanced involvement of paranasal sinus.Thickening of medial rectus is an early sign of orbital invasion.

Patchy enhancement of orbital fat,lesion in the area of superior and inferior orbital fissure and the orbital apex and bone destruction at the paranasal sinus and orbit are seen with advancedstages of the disease. The stretching of optic nerve and tenting of posterior pole of eyeball indicate severe inflammatory edema secondary to tissue necrosis.

MR Imaging and angiography

These help determine the extent of soft tissue involvement, intracranial extension, cavernous sinus involvement and ischemic damage to CNS.MRI with diffusion weighted imaging may also show us the cerebral infarcts.

MANAGEMENT OF RHINO-ORBITO-CEREBRAL MUCORMYCOSIS[4, 18]

Management depend on the stage of the disease and whether it is possible, probable or proven Rhino-orbito-cerebral mucormycosis. The treatment recommendations can be supported by the global guidelines for the diagnosis and management of mucormycosis in 2019 by European Confederation of Medical Mycology (ECMM) and Mycoses Study Group Education and Research Consortium.[20]

Possible ROCM: These patients havesigns and symptoms of ROCM and the risk factors including less than 6 weeks treated COVID-19, Diabetes mellitus, immunosuppression, use of systemic steroids or Tocilizumab, Mechanical ventilation or supplemental oxygen.These patients should be given.Supportive treatment and kept under observation. Nasal endoscopy is repeated after 24hour and with contrast enhanced MRI or CT scan after 72 hours.If patient improves and there is no evidence on endoscopy or imaging then just continue observation for 3 weeks. On the other hand,if there is evidence on endoscopy or biopsy then manage it as a case of probable ROCM.

Probable ROCM patients: They have signs and symptoms plus there is supportive evidence clinically and on diagnostic nasal endoscopy and/or contrast enhanced MRI/CT scan.

Proven ROCM patients: have signs and symptoms plus there is supportive evidence clinically and on diagnostic nasal endoscopy and/or contrast enhanced MRI/CT scan and also there is confirmation on direct microscopy or histopathology with special stains or molecular diagnosis.

For both possible and proven ROCM patients

Immediate induction therapy with intravenous Liposomal Amphotericin-B (5-10 mg/kg body weight) for a minimum of 4weeks followed by oral isavuconazole (200mg TDS on day 1-2 and 200mg OD from day 3) or oral Posaconazole (300mg BD on day 1 followed by 300mg OD from day 2).The oral drugs are given for a duration of3-6 months (minimum of 6 weeks).

If amphotericin B is not available, Amphotericin B lipid complex (ABCL)can be used but its side effects are more prominent. Also, if Amphotericin B is contraindicated, intravenous Isavuconazole (200mg TDS on day 1-2 followed by 200mg OD from day 3)or intravenous Posaconazole (300mg BD on day 1 followed by 300 mg OD from day 2) can be used.

With this if there is sino-nasal involvement, early and aggressive MRI/CT guided debridement of paranasal sinus is done (with or without Turbinectomy, palatal resection and medial orbital wall resection) with clean

margins. Alongwith this, Retrobulbar Amphotericin B (3.5mg/ml) and sinus irrigation with Amphotericin B (1mg/ml) can also be given.

If there is worsening of orbital component in less than or equal to 72hr, then orbital exenteration can be considered along with above measures.

If CNS is involved surgery is done if systemic condition permits (orbital exenteration+ aggressive debridement of paranasal sinus+ turbinectomy+ palatal resection + orbital wall resection)with clean margins along with supportive treatment.

And if surgery is not feasible then only supportive treatment is done.

In neutropenic patients, those with graft-versus-host disease or high risk factors, primary prophylaxis with posaconazole may be recommended(ECMM and Mycoses Study Group Education and Research Consortium).[20] However, no such recommendations have been givenfor severely ill COVID patients, and may be given by physician based on his assessment.

PROGNOSIS

Survival in mucormycosis depends on early diagnosis, alleviation of basic predisposing factors, aggressive debridement of necrotic tissues and appropriate systemic antifungal agents. [8]

Mortality rate in ROCM is as high as 90% [10]. Approximately 50% of cases of mucormycosis have been diagnosed in autopsy [10]. This prompted us to conduct a systematic review of published case reports/series of mucormycosis in people with COVID-19, to know its temporal associations in relation to comorbidities, association with drugs being used in COVID-19 and overall characteristics of patients with its outcome. We additionally postulated a mechanistic explanation as to why mucormycosis could be increasingly linked to COVID-19 and is being reported increasingly from India. Predisposing factors such as corticosteroid therapy should be discontinued and blood sugar should be controlled restrictively. [8] ROCM in case of brain involvement, mortality rises to 50%-85%. [8]

Prognosis is dependent on multiple factors and early initiation of treatment is an important element. [11]A delay of even 6 days is associated with a doubling of 30day mortality from 35%-65%.[10]Once the diagnosis is confirmed conservative management is started for the patient.[11]Orbital exenteration remains the most difficult decision in Rhino-orbital cases, due to concerns about disability and disfigurement. Although exenteration is the last resort, but can be life saving in a few cases. [11]

CONCLUSION

COVID-19 is associated with a significant increased incidence of fungal infections due to Immune dysregulation. Additionally, the widespread use of steroids/Tocilizumab/broad spectrum antibiotics for treating COVID-19 may lead to development/exacerbation of preexisting fungal infection.Diabetes which is the most important risk factor for ROCM, is exacerbated in COVID-19 due to steroid induced hyperglycemia.There is increased risk of fungal infection in patients with pre-existing risk factors.Early diagnosis and treatment should be done as it leads to subsequent reduction of morbidity and mortality due to rhino orbito cerebral mucormycosis and improves the prognosis.

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Allvar Gullstrand

Figure: Allvar Gullstrand, MD (1862-1930). Courtesy of Howe Library, Massachusetts Eye and Ear Infirmary, Boston.

Only one individual who practiced ophthalmology for a significant period has ever received a Nobel Prize, This was Allvar Gullstrand, MD (1862-1930). Today, Gullstrand is best known as the inventor of the slitlamp. When his slitlamp was combined with a microscope made by members of Zeiss Optical Works, Jena, Germany, it became the basis of the instrument that is still used in every ophthalmologist's office today. Gullstrand first demonstrated his slitlamp in 1911, the same year he received the Nobel Prize for his contributions to optics. It incorporated 2 important advances, far more intense light and sharp focus of the beam.

Gullstrand made another important contribution to ophthalmology, a reflexless ophthalmoscope. Ophthalmoscopy can be made difficult by the glare of reflexes formed from the cornea and other layers of the eye, which act like mirrors, reflecting light back at the examiner. Bright sources of illumination and small pupils are contributory factors. The solution to this problem is either to separate the systems of illumination and observation or to use polarized light. Gullstrand used the first of these 2 options.

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