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Journal of Ophthalmological Society of Assam

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November 2018, Vol 2, Issue 1

Editorial

Towards a firm foundation

Dr Madhurjya Gogoi, MD
15 November, 2018
Guwahati, Assam

The *J*ournal of *O*phthalmological *S*ociety of *A*ssam (JOSA) was launched by OSA in its Golden Jubilee year 2017. As completes its first year, we take a look at the year gone by and highlight areas of priority.

The submissions have been varied, and several were based on work done in the North Eastern part of India . It is encouraging to note that 'Original articles', are being submitted. The review process has been a priority to ensure that the content of articles remains high. The inaugural editorial mentioned bi-annual issues, but this was not possibly for the first year because submissions were fewer and about 25% of articles have not cleared the review process so far.

Over the last year, certain areas of concern have been noted. All submissions received so far have been based on studies of a non-regulatory nature. Certain requirements that are now mandatory, such as registered Ethics Committees (EC), and require a declaration to the journal, number less than 10 in the region. Approved Centres for certain regulatory trials, such as Bioavailability and Bioequivalence, are nonexistent. Likewise, the ICMJE recommendations seem to be unfamiliar, and there is a need to discuss its nuances for the benefit of potential authors, editorial board members and reviewers alike. The inescapable conclusion is that, barring few exceptions, the research foundation in the region is nascent, and this may be expected to reflect in the articles published in JOSA in the near future.

JOSA is committed to make the journal available to all OSA life members. JOSA shall continue with 'Open Access' policy. Articles can be submitted at any time by email only at journal.osa@gmail.com. Authors shall not be charged, other than for including more than a permissible number of colour photographs. To enhance access through the internet, basic information on JOSA is available on the website of its parent organization OSA. At this time, JOSA doesn't quite have the resources to be a fully electronic journal.

JOSA would like to gratefully acknowledge all contributors, readers, editorial board members, reviewers and well wishers, but for whose continued support and goodwill, JOSA would simply not exist. Inadvertent errors of omission and commission may have crept in; and for that the editor takes responsibility.

Suggestions / feedback are welcome at: journal.osa@gmail.com
Website: www.osa.ind.in
Facebook group 'Ophthalmological Society of Assam'

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1. Nayak B.K. Ophthalmic Research in India: Are We on the Right Track? JIMSA July-Sep2010 Vol 23, No.3
2. International Committee of Medical Journal Editors (ICMJE) -*Dec 2016*

About the Journal

Journal of Ophthalmological Society of Assam (JOSA), is the official scientific publication of Ophthalmological Society of Assam (OSA). It is a peer-reviewed open access semiannual online journal. The journal's full text is available online at http://www.osa.ind.in/journal.htm. The journal allows free access (Open Access) to its contents and permits authors to self-archive the final accepted version of the articles on any OAI-compliant institutional/subject-based repository.

Scope of the Journal

Journal of Ophthalmological Society of Assam covers all aspects of clinical, experimental, basic science, interdisciplinary, multidisciplinary and translational research studies related to ophthalmology and vision science, with a preference for articles of applied interest.

Preparation of Manuscripts

Manuscripts must be prepared in accordance with "Uniform requirements for Manuscripts submitted to Biomedical Journals" developed by the International Committee of Medical Journal Editors (October 2008). The uniform requirements and specific requirement of Journal of Ophthalmological Society of Assam are summarized below. Authors are requested to check for the latest instructions the website of the journal (http://www.osa.ind.in/journal.htm).

Journal of Ophthalmological Society of Assam accepts manuscripts written in American English.

The Editorial Process

- A manuscript will be reviewed for publication subject to the Author's declaration
- The journal requires a corresponding author who will be responsible for all communication with the Journal related to the manuscript.
- All manuscripts received are duly acknowledged and given a Manuscript Number.
- On submission, all submitted manuscripts undergo a screening prior to a formal review. Manuscripts with insufficient originality, serious shortcomings, or outside the scope of JOSA may be rejected at this stage itself.
- Peer Review is undertaken by two or more subject expert reviewers. The contributor may suggest two or three qualified reviewers; such reviewers should not be affiliated with the same institutes as the author/co-authors. The selection of reviewers is at the sole discretion of the editorial board.
- The Journal follows a double-blind review process, wherein the identity of reviewers and authors are concealed from each other.
- The reviewers' comments (acceptance/ rejection/ amendments in manuscript) are conveyed to the corresponding author. The author is requested to submit a revised version of the manuscript incorporating a point by point response to reviewers' comments. This process may be repeated till reviewers and editors are satisfied with the manuscript.
- Manuscripts accepted for publication are copy edited.
- Page proofs are sent to the corresponding author. The corresponding author is advised to return the corrected proofs within 7 calendar days. It may not be possible to incorporate corrections received thereafter.
- *All* communication shall be by *email only* until further notice.

- The journal publishes articles online as 'Ahead of Print' on acceptance

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- Registration of clinical trials should conform to norms as updated by regulatory authorities in India from time to time. Kindly visit http://www.cdsco.nic.in
- For studies conducted in India, registration is required at http://www.ctri.in/
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- Other acceptable trial registers are http://www.actr.org.au/; http://www.clinicaltrials.gov/; http://isrctn.org/; http://www.trialregister.nl/trialreg/index.asp, http://www.umin.ac.jp/ctr. Kindly consult editorial board, JOSA for any clarification
- For clinical trials that have begun enrollment of subjects before June 2008, retrospective registration with clinical trial registry is admissible.

Authorship Criteria

JOSA strongly recommends prospective authors to adhere to guidelines of the International Committee of Medical Journal Editors (http://www.icmje.org)

Authorship credit should be based only on substantial contributions to each of the three components mentioned below:

1. Concept and design of study or acquisition of data or analysis and interpretation of data;
2. Drafting the article or revising it critically for important intellectual content; and
3. Final approval of the version to be published.

The order of naming the Authors should be based on relative contributions towards the study and writing of the manuscript. Once submitted, the order cannot be changed without written consent of all the contributors. JOSA prescribes a maximum number of 6 (six) authors for manuscripts. If the number of authors exceeds six, a justification signed by all authors should be submitted.

Acknowledgement is permissible for members responsible for acquisition of funding, collection of data, technical support, and general supervision.

Contribution Details

Each contributor should have participated sufficiently in the work to take public responsibility for appropriate portions of the content of the manuscript. Contributors should mention the contributions made by each of them towards the manuscript. The nature of contribution could be: concept, design, definition of intellectual content, literature search, clinical studies, experimental studies, data acquisition, data analysis, statistical analysis, manuscript preparation, manuscript editing and manuscript review. At least one author should take responsibility for the whole work and he/she shall be designated as 'guarantor'.

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All authors of must mandatorily submit the **conflict of interest form along with the main manuscript submission,** disclosing any and all possible conflicts of interest they may have with regard to the manuscript. The conflict of interest form is separate from the copyright transfer form

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Types of Manuscripts

- Editable checklists for reporting guidelines can be found on the EQUATOR Network site (www.equator-network.org)
- The following table lists the type of articles accepted by JOSA

TYPE of ARTICLE	Abstract	Word Limit (Max)*	Combined Maximum of Tables and Figures	Maximum References	Type of study	Checklist As per study Available on respective websites
1. ORIGINAL ARTICLES	Structured: Aim Methods, Results, Conclusion Maximum 250 words	3000 words	5	40	Randomized Controlled Trial (RCT), Prospective, Retrospective Observational / Interventional Study, Non-randomised Trial, Descriptive Data, Cost Analysis, Animal Studies	CONSORT,STROBE ,RECORD, TREND, COREQ, SRQR, CHEERIES, STARD, REMARK, TRIPOD, CHEERS, ARRIVE, REFLECT
2. REVIEW ARTICLES	Unstructured maximum 250 words	5000 words	10	100	Review of Observational studies, Systematic review, Meta-analysis, Qualitative Data By editorial invitation only. All review articles are subject to peer review	MOOSE, ENTREQ, PRISMA
3. CASE REPORT and CASE SERIES	Unstructured maximum 100 words	900 words	Maximum 4 tables or figures	Maximum 10	Structure: Introduction, Case report and discussion. Must add to existing knowledge. Proper	CARE

					documentation required. Case series must contain 3-10 cases.	
4. LETTERS TO THE EDITOR AND LETTERS IN RESPONSE	No abstract required	300 words	Maximum 2 tables or figures	Maximum 5	Should be either a response to a specific article published within the last 6 months, or introduction of a new issue. Letters in response are invited by Editorial board	Not applicable
5. GUEST EDITORIAL	No abstract required	1000 words	Maximum 4 tables or figures	Maximum 20	By invitation from Editorial board only	Not applicable
6. RESEARCH METHODOLOGY	Unstructured maximum 250 words	3000 words	Maximum 5 tables and 5 figures	Maximum 40	Scientific writing, statistics, legal, ethical aspects	Not applicable
7. OSA MEETING PAPERS	All papers that receive an award at an OSA meeting are required to be submitted to JOSA w.e.f. Golden Jubilee Conference 2017					As above

- * Excluding title, abstract, tables and figures, legends and references.

Ethics

When human subjects are involved in India, authors are required to declare compliance with ICMR 'Ethical guidelines for biomedical research on human subjects (http://www.icmr.nic.in/ethical_guidelines.pdf) /Clearance from Ethics Committee/Institutional Review Board in accordance with the Helsinki Declaration of 1975, as revised in 2000. Patient's identity should not be revealed, especially in illustrative material. When reporting experiments on animals, indicate compliance with applicable regulatory requirements on use of laboratory animals.

References

JOSA recommends formats used by the National Library of Medicine (NLM) *in Index Medicus* (https://www.nlm.nih.gov/bsd/uniform_requirements.html). References should be *numbered* consecutively in the order in which they are first mentioned in the text (not in alphabetic order). Identify *references in text,* tables, and legends by Arabic numerals in superscript with square bracket after the punctuation *marks. References cited only* in tables or figure legends should be

numbered in accordance with the sequence established by the first identification in the text of the particular table or figure. Names of non-indexed journals should be mentioned in full. Avoid using abstracts,"personal communication", and "unpublished observations" as references.

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- Tables should be self-explanatory and should not duplicate textual material.
- Each table should be limited to maximum of 5 columns and 20 rows.
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- Explanatory notes, credit lines to be placed in footnotes.
- Explain in footnotes all non-standard abbreviations that are used in each table.
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- Tables with their legends should be provided at the end of the text after the references in the manuscript file, not as separate file
- The table number should be cited at the relevant place in the text.

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- Upload images **as separate files in JPEG** only format. *Do not* inserted images in the body of the manuscript. Each file size should be within 2MB with minimum resolution of 300 dpi or 1800 x 1600 pixels. Rename file with - MANUSCRIPT NUMBER_FIGURE NUMBER, e.g., JOSA_2017_17_Fig 1
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1) Files should not be zipped.
2) Figures should be numbered consecutively according to the order in which they have been first cited in the text.
3) Labels, numbers, arrows, letters and symbols should be clear and of uniform size and good contrast. The lettering for figures should be large enough to be legible after reduction to fit the width of a printed column.
4) When graphs, scatter-grams or histograms are submitted the numerical data on which they are based should also be provided.
5) Legends for illustrations: word limit 50, excluding the credit line, using double spacing, and tagged with Arabic numerals. Place legends **after** the References Section / Tables in the article file.
6) The Journal reserves the right to edit illustrations.

Videos

- Video facility is not available presently. This section shall be updated in due course.

Protection of Patients' Rights to Privacy

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The revised manuscript should be submitted by email quoting the manuscript number, incorporating point to point clarification to the reviewer's remarks, highlighting the changes in the article. The "First Page" or "Covering Letter" file may be omitted while submitting a revised version.

Reprints and Proofs

A complimentary copy is offered to all Authors and Co-Authors whose articles are published in JOSA. The Journal provides no free printed reprints. To purchase reprints, kindly contact the editorial office.

Publication Schedule

JOSA is in the process of bringing out semiannual issues.

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4) Article File
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Presentation and Format

- Double spacing
- Margins 2.5 cm from all four sides
- Page numbers included at bottom
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- Abstract page contains the full title of the manuscript
- Key words provided (3-5)
- Manuscript title should be in Sentence case
- Remove 'Track Changes'

Language and Grammar

- Uniformly American English
- Write the full term for each abbreviation at its first use in the title, abstract, keywords and text separately unless it is a standard unit of measure.
- Numerals from 1 to 10 spelt out
- Numerals at the beginning of the sentence spelt out
- Check the manuscript for spelling, grammar and punctuation errors
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- Species names should be in italics

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Editorial and Ethics Policies of Journal of Ophthalmological Society of Assam conform to recommendation of the International Committee of Medical Journal Editors (http://www.icmje.org)

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(One signed copy to be submitted with the manuscript)

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Journal of Ophthalmological Society of Assam (JOSA)

JOSA is inviting original articles, case series and case reports related to visual sciences for its forthcoming issues.

For instructions for authors, kindly visit www.osa.ind.in/journal.htm. it is mandatory to submit the following

1. ICMJE Form for Disclosure of Potential Conflicts of Interest
2. Authors' Declaration and Copyright Transfer form

Both 1 and 2, above, can be downloaded from www.osa.ind.in
Presently, all submissions may be made by email only at journal.osa@gmail.com.

PEER REVIEW PROCESS

All submissions shall be peer reviewed. The peer review process is designed to assure that JOSA publishes only original, accurate, and timely articles that contribute to knowledge in the Vision Sciences.

The Editor shall make a preliminary assessment on whether the manuscript meets the requirements of the journal and is worth sending out for thorough review.

If so, it is then assigned to three reviewers, presently by email only, that may include any member of the Editorial Board and/or other experts in relevant fields, as selected by the Editor- for review, preferably in a double blind process.

Reviewers are asked to assess submissions based on depth of original research, accuracy, appropriate documentation, readability, and suitability of content.

Reviewers shall make One of Four Recommendations:

1. Acceptance
2. Provisional Acceptance With Revision
3. Provisional Non-Acceptance
4. Rejection

For points 2, 3 and 4, reviewers are required to include comments explaining the recommendation.

Authors may expect to know the results of the manuscript peer review within four weeks from the date of submission.

Authors shall receive the reviewers' comments and be advised to revise their manuscripts in line with the reviewers' and/or editor's suggestions.

If the revised article is accepted for publication, the editor then determines the journal issue in which it will appear. All efforts are made publish an accepted article in the next issue of the journal.

Recent advances in diagnosis and management of Herpes simplex virus (HSV) epithelial and stromal keratitis

Swapnali Sabhapandit, MS

Abstract:

Herpes simplex keratitis is a common disease entity with no definite treatment till date. In developing countries, there is a lack of data on incidence and prevalence of the disease. The epithelial and stromal form of disease presents different clinical forms and management strategies. Although the antiviral drugs have adequate potency, recurrence of infection and rise of antiviral resistance are major challenges in the eradication of the virus from the host. The availability of a preventive vaccine is awaited as clinical trials are in progress.

Key words:

Herpes simplex keratitis, cornea, acyclovir, resistance, vaccine.

Introduction:

Herpes simplex keratitis is a corneal infection caused by the Herpes simplex virus (HSV) type 1[1,2]. This is a ubiquitous DNA virus that affects any body part and any age group[1]. The type 1 virus involves the mouth and eye, rarely the brain. Type 2 virus involves the genitalia commonly[3]. The HSV virus is responsible for nearly 30-45,000 cases of severe visual impairment in developing countries per year[4, 5]. The disease usually occurs unilaterally, bilateral cases being in only approximately 1.2-10% of cases[6]. People with atopy or an immune deviation may be predisposed to bilateral involvement[7,8]. Any part of the eye can be affected, commonest being the corneal epithelium and stroma[7,9].

A major causative factor for the universal spread and high ocular morbidity rate of this form of keratitis is the tendency for latency of the virus. This has implications in corneal transplantation, eye banking and antiviral resistance[10,11,12]. This updated review will focus on the various challenges and developments in management of HSV keratitis.

Pathophysiology of HSV keratitis:

HSV is a double-stranded DNA virus belonging to Alpha herpes virinae, a subfamily of the

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E-mail: drswapnali@lvpei.org
Support – Hyderabad Eye Research Foundation (Intramural support)
Conflict of Interest: Nil, Permissions: Nil , Prior Publication – Nil
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Herpes viridae family[8,13]. The subfamily consists of Herpes simplex virus type-1, Herpes simplex virus type-2 and varicella zoster virus. Primary infection results with HSV entry via direct contact with mucous membrane of the host[14]. After primary infection, the virus is transported via sensory neurons to establish latency in trigeminal ganglia where it remains asymptomatic until reactivation of the virus leads to secondary or recurrent infections**(ref)**. The host cell's nuclear DNA polymerase is used by HSV to transcribe and replicate[15,16,17].

The HSV keratitis occurs in 3 forms- a) Epithelial b) Stromal c) Endothelial (Table 1). There may be overlap of features due to variation in presentation between different subtypes.

Clinical features of HSV keratitis:

a) ***Epithelial keratitis***: This form of keratitis accounts for 70-80% of cases[18]. It begins as superficial coarse punctate lesions, that may coalesce to form a stellate erosion and, finally, a dendritic or geographic ulcer[18,19]as shown in Figure 1.

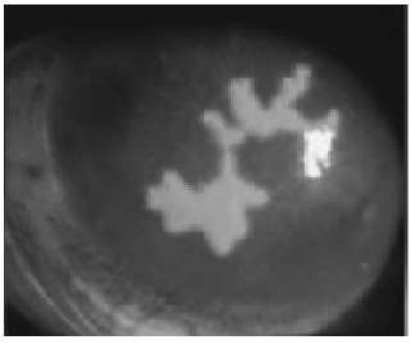

Figure 1: Dendritic ulcer after staining with fluorescein

b) The host's immune system helps in clearing the corneal epithelium of the virus. However, the virus remains in the trigeminal ganglion until reactivated. Marginal ulcers may also be seen involving the limbus and occasionally spilling over to conjunctiva[18].

c) ***Stromal keratitis***: It is commonly seen in recurrences, nearly 20-60% as per studies [20]. Both direct viral effects and CD4+ T cell destruction by body's inflammatory response causes the stromal disease process [20,21,22].This immune response may linger sub clinically long after viral activity is no longer found. The severity of disease may increase with each subsequent episode[20,23]. The stromal form is further subdivided into non-necrotizing and necrotizing keratitis. Non-necrotizing stromal keratitis presents with a localized pattern of corneal edema and is often self-limited (Figure 2),

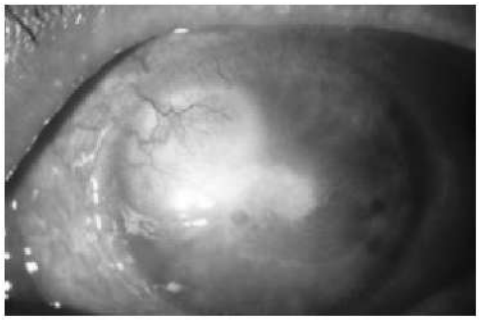

Figure 2: Recurrent non-necrotizing stromal keratitis with corneal neovascularization

d) whereas necrotizing stromal keratitis is a rapidly destructive corneal melting process with stromal infiltrates and dense inflammation (Figure 3).

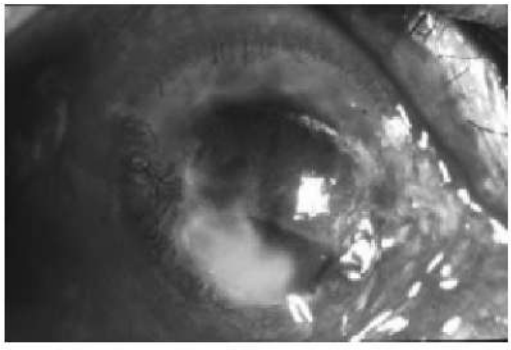

Figure 3: Necrotising stromal keratitis with inferior stromal melt

e) The end result of stromal keratitis is corneal neovascularization and scarring, with corneal blindness in untreated cases [18,24,25].

f) Endothelial keratitis: It manifests as a rejection line-like keratic precipitates with overlying stromal edema (Figure 4).

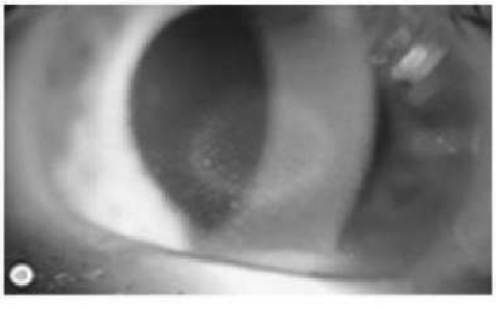

Figure 4: Disciform endothelitis with keratic precipitates underlying area of stromal edema

The cornea may show a linear, diffuse or disciform pattern of involvement. The stromal edema is due to HSV-mediated endothelial dysfunction in the absence of stromal inflammation or neovascularization[18].

Diagnosis of HSV keratitis:

HSV keratitis is mainly diagnosed by its clinical presentation on the slit-lamp examination [9]. Symptoms include redness, photophobia, irritation, pain and watery discharge. In the self-limiting epithelial form, symptoms begin to subside after the first **2** weeks.

Epithelial keratitis presents on the slit lamp as coarse punctate spots or dendritic lesion with a terminal bulb, swollen borders, and intraepithelial cell infiltration [20,26,27]. The lesions are best visualized by staining with either lissamine green or rose bengal dye [28,29]. Immune suppression, atopy or indiscriminate corticosteroid use can lead to geographic ulcers [18]. Newer methods such as Giemsa stain, polymerase chain reaction (PCR), tear collection and immuno fluorescence antibody assay (IFA)have been used to confirm HSV keratitis in atypical cases[30,31,32,33].The positive predictive value of real time PCR in epithelial disease can be as high as 81% [30,33]. However, PCR has high predictive value in cases which are not on antiviral therapy [18,35].The sensitivity of IFA and PCR is about 78.6% and 81.2%, respectively[30,33].Giemsa staining of corneal scraping for multinucleated giant cells shows a sensitivity of around 48%.[33]A triad of Giemsa staining, PCR and IFA gives a positive predictive value of nearly 95-100%[31,33].

Like epithelial keratitis, stromal keratitis is also primarily a clinical diagnosis. The greyish white opaque lesion with surrounding edema is pathognomonic, which may be accompanied by stromal necrosis and abscess formation in the necrotizing variety. Recurrent cases frequently show neovascularization, both deep and superficial. Reduced corneal sensation is also seen in majority of cases. The need for Giemsa staining of stromal tissue smear, PCR and IFA in stromal form of the disease arises when necrotic changes or superadded infection confounds the clinical picture[31,33].

Although viral culture is the gold standard for the detection of HSV, the time consuming and expensive methodology restricts the use of this tool by clinicians[31,34,35].

Management of HSV keratitis:

The management of HSV keratitis can be challenging. Although disease episodes can be self-limiting, timely and proper treatment is necessary to shorten disease course, reduce viral replication and risk of latency and prevent ocular morbidities like corneal opacity, perforation and blindness[18].

The management of HSV keratitis can be medical or surgical.

1. ***Medical management of HSV keratitis***: The commonest method of treatment is by antiviral therapy. There are mainly two groups of drugs, both DNA polymerase inhibitors[18,36]–

a. Purine analogues- Acyclovir, valacyclovir, ganciclovir, famcyclovir, cidofovir, vidarabine

b. Pyrimidine analogues- Idoxuridine, trifluoridine.

Acyclovir is the primary antiviral drug used for treating HSV keratitis. The drug prevents nucleoside chain elongation as it gets phosphorylated by the viral thymidine kinase enzyme and blocks DNA polymerase activity after being incorporated into the viral DNA. Thus, viral replication by DNA elongation is inhibited. The host DNA is not affected, hence side effects to the patient are reduced. The drug protects fresh host cells from getting infected, but does not cure host cells that are already infected[37]. The newer drugs such as valacyclovir, famcyclovir and ganciclovir have similar mode of action, but better bioavailability and hence lesser dosing than acyclovir [39,40,41]. Cidofovir has more side effects and is not commonly used. Trifluoridine is not popular in India commercially though it is FDA approved for HSV keratitis treatment.

Vidarabine and idoxuridine have poor bioavailability and higher risk of side effects; hence they are no longer used for HSV keratitis therapy [42].

Antiviral therapy may be by oral and topical route.

a. ***Topical therapy***- Acyclovir 3% eye ointment is available widely and is commonly used. The corneal penetration is excellent as proven by aqueous humor level being higher than median effective dose (ED50) [43]. Ganciclovir eye gel 0.15% is also available, however the ED50 is achieved only with a concentration of 3% which is not commercially available [44,45]. The dosing regimens given in Table 2.

b. ***Oral therapy***-Acyclovir tablet is widely used in stromal and endothelial forms of the disease. Various studies have proven equal efficacy of topical and oral acyclovir therapy for epithelial keratitis, hence topical route is preferred in this form of keratitis [37,46,47]. Studies have shown efficacy of valacyclovir and famcyclovir in preventing recurrences [18,41,42,48]. All three oral antiviral drugs are used in prophylactic doses for recurrence prevention. The dosing regimen of these drugs is shown in Table 3.

Role of corticosteroids in medical therapy- Corticosteroids are potent suppressors of inflammation and play a major role in management of stromal and endothelial form of HSV keratitis. Various studies have proven the efficacy of combined use of antiviral and steroid therapy in such cases [49,50]. The tapered dosing of topical or oral steroid should be more than 10 weeks for adequate inflammation control [50]. However, steroids are contraindicated in the epithelial form of HSV keratitis as it is proven to increase viral latency and risk of recurrence [51,52,53].

2. ***Surgical management of HSV keratitis***- Surgery is required in a low percentage of cases having HSV keratitis. Debridement was practiced earlier for reducing the viral load in epithelial form of the disease. However, multiple studies have shown that topical antiviral therapy is more effective in clearing viral load on the epithelium. Hence, debridement is no longer popular in treating HSV keratitis [53,54,55,56]. Necrotising form of stromal keratitis may need use of tissue adhesive with bandage contact lens if excessive corneal melt or impending perforation is noted. Alternatively, emergency corneal patch graft is an option. Recurrent cases may have dense stromal scarring with vascularization as an end result. Once the recurrence is controlled with prophylactic antiviral therapy, elective corneal transplant can be planned based on the degree of vascularization with antiviral cover [57,58].

Recurrence of HSV keratitis

In absence of prophylactic therapy, risk of recurrence is approximately 20% by 2 years, 40% by 5 years, and 67% by 7 years [59]. Latency of the virus and its DNA in host cells, commonly neurons and ciliary ganglia, followed by reactivation is responsible for recurrent episodes of epithelial disease and stromal inflammation. Several factors can aid in the latency and reactivation mechanism, which include

a. ***Altered immune system***- This can occur due to multiple reasons such as extremes of age (ref), fever, altered lifestyle with mental stress (ref), menstruation, atopic diseases, immunosuppressant use, HIV infection, measles, vaccination, diabetes mellitus and organ transplant patients[60,61,62].
b. ***Ocular immune causes***- This includes use of drugs such as topical corticosteroids, prostaglandin analogues and angiogenesis inhibitors [63,64,65]. Trauma to the eye due to injury, contact lens use, surgical procedures or any laser therapy can also elicit a

recurrence [66-69].

Every recurrence leads to corneal scarring, risk of melt and vascularization of the cornea, which in turn causes corneal blindness and higher ocular morbidity in the population. The actual incidence of corneal blindness due to HSV keratitis in developing countries has not been studied yet. In developed nations, the rate of severe visual loss due to recurrent HSV keratitis is around 0.015[70,71,72]. This rate is expected to be much higher in the developing world.

Resistance to antiviral drugs

There have been multiple reports of resistance to acyclovir therapy in both developed and developing countries. This is more prevalent in immunocompromised patients and in long term use of acyclovir for prophylaxis [73-76]. Mutation or deletion of the thymidine kinase gene, which may be difficult to interpret due to gene polymorphisms, is believed to be the cause of the increased resistance [74,75].Such patients are resistant to valacyclovir, ganciclovir and famcyclovir also. Recently, alarming reports of acyclovir resistance as high as 6.5% in immuno competent patients have been seen, especially in recurrent infection cases[77]. The mutation has been seen in the DNA polymerase gene, which includes resistance to both purine and pyrimidine analogues of antiviral drugs [76,77].

Vaccination for HSV keratitis prevention

Due to increase in the incidence of antiviral resistance, there have been efforts to prevent HSV infection through adequate immunoprotection. The role of a vaccine to prevent HSV infection has been investigated. Greater focus has been given on developing a vaccine for HSV 2 due to its association with HIV infection and risk of genital herpes[78]. The high level of homogeneity in the genetic pattern of HSV1 and HSV 2 may allow protection for both types with vaccination. The two prominent vaccines under trial were Herpevac trial and VCL-HB01 trial. Both vaccines failed to offer protection against HSV 2, so presently there is no protection for patients against HSV infection[80,81]. Therapeutic vaccines for patients already infected with the disease are under trial[81,82]. The complex DNA structure, latency and low level of detection by the host's immune system are the challenges in preparing a vaccine for this ubiquitous virus.

Conclusion

HSV keratitis is a disease which is still lacking a permanent cure through either medication or surgery. Seroprevalence in the population is undetected in most developing countries, hence the exact burden of the disease on healthcare is not known. A national level surveillance program is needed for detection of incidence and prevalence of this disease. Better diagnostic tests, newer potent antiviral therapy and vaccination of protection are the need of the hour for winning the battle against HSV keratitis and its complications.

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Table 1: Clinical presentation of HSV keratitis

Corneal layer involved	Nomenclature
Epithelium	Punctate epithelial keratitis
	Dendritic ulcer
	Geographic ulcer
Stroma	Non-necrotizing keratitis
	Necrotising keratitis
	Immune stromal keratitis
Endothelial	Linear endothelitis
	Disciform endothelitis
	Diffuse endothelitis

Abbreviations: HSV- herpes simplex virus

Table 2: Topical regime of treatment for Herpes Simplex Virus (HSV) keratitis

Type of keratitis	Antiviral agent	Dosing regime	Corticosteroid therapy
Punctate epithelial	Acyclovir 3% oint.	5 times daily X 3 weeks	Not recommended
	Ganciclovir 0.15% gel	5 times daily X 3 weeks	
	Trifluridine 1% eyedrop (not available in India)	9 times daily X 1 week, then 5 times daily X 2 weeks	
Dendritic	Acyclovir 3% oint.	5 times daily X 3 weeks	Not recommended
	Ganciclovir 0.15% gel	5 times daily X 3 weeks	
	Trifluridine 1% eyedrop	9 times daily X 1 week, then 5 times daily X 2 weeks	
Geographic	Acyclovir 3% oint.	5 times daily X 3 weeks	Not recommended
	Ganciclovir 0.15% gel	5 times daily X 3 weeks	
	Trifluridine 1% eyedrop	9 times daily X 1 week, then 5 times daily X 2 weeks	
Non-necrotizing	Acyclovir 3% oint.	5 times daily X 3 weeks	Prednisolone 1% tapered weekly from 6-8 times daily till over 10 weeks
	Ganciclovir 0.15% gel	5 times daily X 3 weeks	
	Trifluridine 1% eyedrop	9 times daily X 1 week, then 5 times daily X 2 weeks	
Necrotizing	Acyclovir 3% oint.	5 times daily X 3 weeks	Prednisolone 1% tapered weekly from 4 times daily till over 10 weeks
	Ganciclovir 0.15% gel	5 times daily X 3 weeks	
	Trifluridine 1% eyedrop	9 times daily X 1 week, then 5 times daily X 2 weeks	
Endothelitis	Acyclovir 3% oint.	5 times daily X 3 weeks	Prednisolone 1% tapered weekly from 6-8 times daily till over 10 weeks

Table 3: Oral antiviral treatment regime of Herpes Simplex Virus HSV keratitis

Type of keratitis	Oral antiviral agent	Dosing regime
Punctate epithelial	Not recommended	
Dendritic	Acyclovir 400 mg tablet	5 times daily X 7-10 days
	Valacyclovir 500 mg tablet	2 times daily X 7-10 days
	Famcyclovir 250 mg tablet	2 times daily X 7-10 days
Geographic	Acyclovir 800 mg tablet	5 times daily X 14-21 days
	Valacyclovir 1 gm tablet	3 times daily X 14-21 days
	Famcyclovir 500 mg tablet	2 times daily X 14-21 days
Non-necrotizing *	Acyclovir 400 mg tablet	2 times daily till steroid therapy is completed
	Valacyclovir 500 mg tablet	Once daily till steroid therapy is completed
	Famcyclovir 250 mg tablet	2 times daily till steroid therapy is completed
Necrotizing *	Acyclovir 800 mg tablet	5 times daily X 7-10 days
	Valacyclovir 1 gm tablet	3 times daily X 7-10 days
	Famcyclovir 500 mg tablet	2 times daily X 7-10 days
Endothelitis *	Acyclovir 400 mg tablet	5 times daily X 7-10 days
	Valacyclovir 500 mg tablet	2 times daily X 7-10 days
	Famcyclovir 250 mg tablet	2 times daily X 7-10 days

*To be used in conjunction with topical corticosteroids as mentioned in Table 2.

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Hypertensive Retinopathy A Forgotten Entity

Gitumoni Sharma

Purpose: Report cases with blurred vision diagnosed as hypertensive retinopathy secondary to accelerated hypertension.

Materials and methods: Retrospective observational cohort from 1st Jan16- 31st Dec17. Included were cases presented with blurred vision, referred for retinal diseases and blood pressure on doubt, recorded. Accelerated hypertension found. Finally, diagnosed hypertensive retinopathy. Patients' demography analyzed. Referral diagnosis noted. Grading of hypertensive retinopathy done. Fundus photo documentation done. Blood pressure recorded. Patient referred to physician. Statistical analysis**:** Microsoft excel sheet.

Results: 58 patients, 38(65%) were newly diagnosed hypertensives at the time of presentation.Known hypertensives >1 month history 20(34.48%).Detected hypertensive retinopathy upon funds examination 25(65.78%).Grade III 26, Grade IV 32.Referred as Neuroretinitis 2, Ischemic CRVO 2, Papillodema 2, Eales' 1, posterior uveitis 1.18 (31%)in the age group of 2040 yrs. 20 (34%)in the age group of 40 60yrs. 20(34%) in the age group of >60yrs. BCVA >20/200, 38 (65%), < 20/200,20 (34%).BP of <180/100, 30(52%) and >180/100, 8(48%).Walk in patients for blurred vision 25(65.78%). All patients improved with control of their systemic hypertension.

Conclusion: There is insufficient evidence to recommend a routine ophthalmoscopic consultation for all patients with hypertension. Patients with blurring of vision, typical fundus finding of retinal haemorrhage, soft exudates and macular star, and accelerated Hypertension should be considered and recording of BP is mandatory not to miss a single case of hypertensive retinopathy.

Keywords: Hypertensive retinopathy, Hypertensive proliferative retinopathy, Accelerated Hypertension.

Introduction:

The American College of Cardiology (ACC) and the American Heart Association (AHA) have released a new guideline on hypertension with a new definition that will call 130 to 139 mm Hg systolic or 80 to 89 mm Hg diastolic stage 1 hypertension.[1]

Hypertensive retinopathy is the most common manifestation, and its presence is predictive of

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stroke, congestive heart failure and cardiovascular mortality.[2]

Inspite of the seriousness, blood pressure remains the most important public health problem in developing and developed countries. It is common, asymptomatic, readily detectable, usually treatable, and often leads to lethal complications if left untreated.

Hypertension has profound effects on various parts of the eye. Hypertensive retinopathy is among the vascular complications of essential hypertension. It is known that the auto-regulation of retinal circulation fails as blood pressure increases beyond a critical limit. [3]

Hypertensive retinopathy was first described by Marcus Gunn in the 19th century in a series of patients with hypertension and renal disease.[4]

Signs of hypertensive retinopathy are common in age more than 40 yrs even without history of hypertension. Its prevalence 2-15% and incidence is over 5-7 yrs is 6-10%.[5]

Classically, elevated blood pressure results in a series of retinal microvascular changes called hypertensive retinopathy, comprising of generalized and focal retinal arteriolar narrowing, arteriovenous nicking, retinal hemorrhages, microaneurysms and, in severe cases, optic disc and macular edema.[6] So, these conditions may mislead to optic neuropathy and macular oedema due to other causes if blood pressure is not checked. In addition, several retinal diseases such as retinal vascular occlusion (artery and vein occlusion), retinal arteriolar emboli, macroaneurysm, ischemic optic neuropathy and age-related macular degeneration may also be related to hypertension.

Literature search also shows, over the years, recently only few articles related to hypertensive retinopathy are available, where in reality there are many coming to ophthalmic clinic for blurring of vision who otherwise are also on dialysis etc. due to hypertensive renal failure and that too in young, which could have been prevented if detected early.

Hence, this study was done to report series of cases who had presented or referred with blurred vision and were diagnosed as hypertensive retinopathy secondary to accelerated hypertension.

Methods:

Retrospective cross sectional analysis of the case sheets were done for 2 years from 1st Jan 2016- 31st Dec 2017. Institutional Ethics committee approval was taken to do the study. The cases included were who had presented with blurred vision3, with or without headache, referred for retinal diseases and blood pressure on doubt were recorded and accelerated hypertension was found. Finally, diagnosed as hypertensive retinopathy. Patient demography was analyzed. Referral diagnoses were noted.

A detailed ophthalmological examination included best corrected visual acuity(logMAR chart), anterior segment examination using slit lamp, intraocular pressure by Goldmann applanation tonometry and dilated posterior segment examination by slit lamp biomicroscopy using 90D, and indirect ophthalmoscopy with

+20D lenses followed by fundus photography was done Blood pressure on doubt were recorded in supine position [microlife automatic upper arm BP monitor] and accelerated hypertension was found. Finally the cases were diagnosed as hypertensive retinopathy. Staging of hypertensive retinopathy was carried out using Modified (Keith Wagner Barker) Scheie Classification 1953.[7] Fundus photograph was taken for each patient at each visit for documentation of the fundus. Patient was referred to physician for further management of systemic blood pressure. Patients were reviewed after 1 and 3 months of antihypertensive treatment.

Excluded were cases of blurred vision where final diagnosis was not hypertensive retinopathy only. The primary outcome measures were change in visual acuity, improvement of hypertensive retinopathy status following control of hypertension and the secondary outcome measures were duration of hypertension, amount of blood pressure elevation and hypertension grades.

Results:

There were a total of 58 patients, all had bilateral hypertensive retinopathy. Grade III was in 26 eyes (44.82%) and Grade IV was in 32 eyes (55.17%). Of these, 38 (65%) were newly diagnosed hypertensives at the time of presentation with hypertensive retinopathy. Figure I. 20(34.48%) were known hypertensives for more than 1 month history with antihypertensive treatment. Referred for retinal diseases were 08(13.70%) and walk in patients for blurred vision 25(65.78%).

There were 38(65%) male and 20(34%) female, who had presented in the mean age of 52+/-14(23-78), median 50.5years.18 (31%) were in the age group of 2040 years. 20 (34%) were in the age group of 4060 Years. 20(34%) were in the age group of >60yrs.**Table I**. BCVA of logMAR = /> 1.00 in OD 23 (39.6) and OS 09(15.5%) and logMAR =/< in OD 35(60%) and OS 49(84.48%).**Table II**. 8(13.7%) cases were referred. Referred diagnosis as Neuroretinitis in 2(3.4%), Ischemic CRVO 2(3.4%), Papillodema 2(3.4%), Eales disease 1(1.72%) and posterior uveitis 1(1.72%).**Table III.**

There were 25(65.78%) patients who were detected hypertensive retinopathy upon routine fundus examination. Blood pressure of less than 180/100 in 30 (52%) and more than 180/100 in 28 (48%).

All the patients were improved with control of their systemic hypertension in a follow up of 1 month. **Figure II.** BCVA in OD improved from mean logMAR VA = 0.393 to 0.171(p=0.00 at 1 month and p=0.65 at 3 months) and is OS mean logMAR VA = 0.634 to 0.257(p=0.00 at 1 month and p=0.06 at 3 months) showing statistically significant.**Graph I.** Lowering of mean blood pressure, was 184/110mmHg to 138/86mmHg at 3 months which was statistically significant (p=0.00). **Graph II.** Six representative cases where blood pressure were recorded and found to be systolic more than 200mmhg are shown in Figure. Two representative cases shown in Figure.II, where there is improvement in the signs in the follow -up fundus photograph after control of systemic hypertension. Figure.II.

Discussion:

Hypertensive retinopathy, first described as "albuminuric retinitis,"[6] has traditionally been referred to as a spectrum of "retinal vascular signs"caused by elevated blood pressure.[8]

Hypertension may lead to multiple adverse effects to the eye causing retinopathy, optic neuropathy, and choroidopathy. Hypertensive retinopathy includes two disease processes. The acute effects of systemic arterial hypertension are a result of vasospasm to autoregulate perfusion.[9] The chronic effects of hypertension are caused by arteriosclerosis and predispose patients to visual loss from vascular occlusions or macroaneurysms.[10] The arteriosclerotic changes of hypertensive retinopathy are caused by chronically elevated blood pressure, defined as systolic greater than 140 mmHg and diastolic greater than 90 mmHg. In this study, out of 58 patients in 2 yrs, 38 (65%)were newly diagnosed hypertensives at the time of presentation with hypertensive retinopathy and known hypertensives less than 1 month history with antihypertensive treatment were 20 (34.48%).

Hypertension is usually essential and not secondary to another disease process. Essential hypertension is a polygenic disease with multiple modifiable environmental factors contributing to the disease.

Grades of Hypertension	Fundus Changes
Grade 0	No changes
Grade 1	Barely detectable arterial narrowing
Grade 2	Obvious arterial narrowing with focal irregularities
Grade 3	Grade plus retinal hemorrhages, exudates, cotton wool spots, or retinal edema
Grade 4	Grade 3 plus papilledema

However, secondary hypertension can develop in the setting of pheochromocytoma, primary hyperaldosteronism, Cushing's syndrome, renal parenchymal disease, renal vacular disease, coarctation of the aorta, obstructive sleep apnea, hyperparathyroidism, and hyperthyroidism.[11] Many young patients with secondary hypertension may actually present to an ophthalmologist with bilateral vision loss due to serous macular detachment, bilateral optic disc edema, and exudative retinal detachment. In this study, patients referred for retinal diseases were 08(13.70%) and walk in patients for blurred vision were 25(65.78%).18 (31%) were in the age group of 20 40 years. 20 (34%) were in the age group of 40 60 Years. 20(34%) were in the age group of >60yrs. BCVA of 6/60 or better were 38 (65%) and 6/60 or worse were 20 (34%). Referred diagnosis as Neuroretinitis in 2(3.4%), Ischemic CRVO 2(3.4%), Papillodema 2(3.4%), Eales disease 1(1.72%) and posterior uveitis 1(1.72%).

Risk factors for essential hypertension include high salt diet, obesity, tobacco use, alcohol, family history, stress, and ethnic background. The major risk for arteriosclerotic hypertensive retinopathy is the duration of elevated blood pressure. The major risk factor for malignant hypertension is the amount of blood pressure elevation over normal. In this study, Blood pressure of less than 180/100 in 30 (52%) and more than 180/100 in 28 (48%) were found.

The signs of malignant hypertension are well summarized by the Modified (KWB) Scheie Classification of Hypertensive Retinopathy.[9]

Classification of Hypertensive retinopathy: Keith-Wagener-Barker (KWB) was based on the level of severity of the retinal findings; and Scheie (grade 0-4) attempted to quantify the changes of both hypertension and arteriosclerosis.

Grading of hypertensive Retinopathy included Grade III in 26 (44.82%) and Grade IV in 32 (55.17%) in this study.

The signs of chronic arteriosclerotic hypertension are also summarized by the Scheie Classification.[10]

Grade 4 Grade 3 plus papilledema

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The signs of chronic arteriosclerotic hypertension are also summarized by the Scheie Classification.[10]

Stages	Fundus findings
Stage 1	Widening of the arteriole reflex
Stage 2	Arteriovenous crossing sign
Stage 3	Copper-wire arteries (copper colored arteriole light reflex)
Stage 4	Silver-wire arteries (silver colored arteriole light reflex)

Of specific interest is the classification of hypertensive retinopathy by Wong and Mitchell.[12]

Grade	Stage	Ophthalmoscopic signs	Systemic associations
1	Mild retinopathy	One or more of the following arteriolar signs: -Generalised arteriolar narrowing. -Focal arteriolar narrowing. -Arteriovenous nicking. -Arteriolar wall opacity (silver wiring)	Modest association(risk and odd ratios of > 1 but <2) with risk of clinical stroke, subclinical stroke, coronary heart disease and mortality
2	Moderate retinopathy	One or more of the following arteriolar signs: -Haemorrhage (blot, dot, flame shaped) -Microaneurysm. -Cotton wool spots. -Hard exudates.	Strong association (risk and odds ratios of >2) with risk of clinical stroke, subclinical stroke, cognitive decline and cardiovascular mortality.
3	Malignant retinopathy	Moderate retinopathy plus optic disc swelling.	Strong association with mortality.

The treatment for hypertensive retinopathy is primarily focused upon graduated reducing blood pressure. The treatment for malignant hypertensive retinopathy is to reduce the systemic blood pressure below 140/90 mmHg. This can be accomplished by any of the armamentarium of medical treatments for hypertension. Medical treatment can only treat the acute changes of hypertension from vasospasm and vascular leakage. There is no treatment for arteriosclerotic changes of chronic hypertension. Follow up is dependent upon the degree of hypertension and resistance to medications. Close contact is essential between the ophthalmologist and the primary care physician for consistent follow up individually tailored to each patient. Graduated lowering of blood pressure is essential to prevent end organ ischemia and failure upon sudden lowering of blood pressure including the choroid and retina. In figure.1, there are six representative cases shown where malignant hypertension was recorded. The photo documentation also shows the follow up photograph of the same patient when hypertension is controlled. This shows that once hypertension is controlled the fundus picture also shows improvement. All the patients were improved with control of their systemic hypertension in a follow up of 1 month in this study. These rules out retrospectively the possibility of other pathologies causing blurring of vision.

Hypertension predisposes patients to many other retinal vascular diseases including central or branch retinal artery occlusion, central or branch retinal vein occlusion, and retinal arterial macroaneurysms. Ischemia secondary to vascular occlusions can cause neovascularization, vitreous hemorrhage, epiretinal membrane formation, and tractional retinal detachment. Hypertension also leads to more advanced diabetic retinopathy progression. Hypertensive optic neuropathy can cause chronic papilledema, leading to optic nerve atrophy and severe loss of visual acuity.[9] Recently a term 'proliferative hypertensive retinopathy' has been coined.[13]

Patients with severe hypertensive retinopathy and arteriosclerotic changes are at increased risk for coronary

disease, peripheral vascular disease, and stroke. Since arteriosclerotic changes in the retina do not regress, these patients remain at increased risk for retinal artery occlusions, retinal vein occlusions, and retinal macroaneurysms. Most retinal changes secondary to malignant hypertension will improve once blood pressure is controlled as is also seen in this study. Damage to the optic nerve and macula, however, could cause long term reductions in visual acuity.

Conclusion:

There is insufficient evidence to recommend a routine ophthalmoscopic consultation for all patients with hypertension. Patients with blurring of vision, typical fundus finding of retinal haemorrhage ,soft exudates and macular star, accelerated hypertension should be borne in mind and recording of blood pressure is mandatory, irrespective of age and sex. Treatment should be aimed at graduated lowering of blood pressure to prevent further retinal ischemia. Reduced vision to some extent may be permanent in some cases of late presentation.

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Tables and Figures

Table I: Demographic data

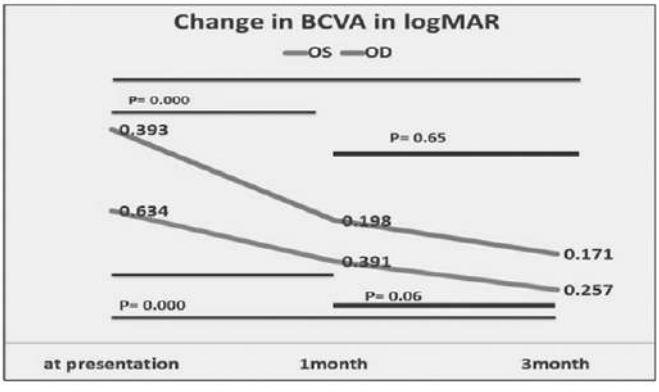
Gender	
Male:	38 (65%)
Female:	20 (34%)
Age in years	
Mean, SD & Range	52 ± 14 (23 - 78)
Median	50.5
Age Groups	**No. of cases**
20-40 Years	18(31%)
41-60Years	20(34%)
>60Years	20(34%)

Table II. Presenting BCVA in logMAR

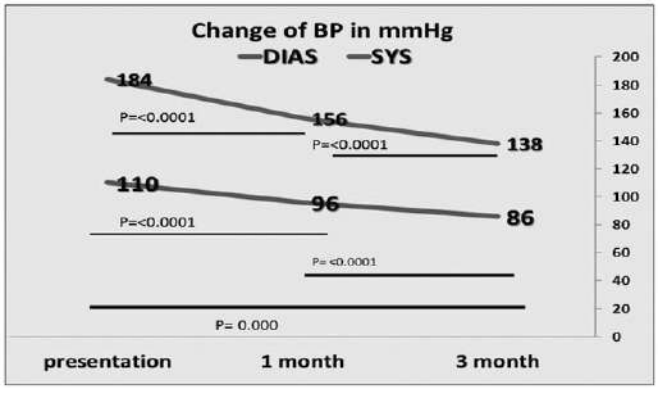
logMAR BCVA	≥ 1.00 or worse	< 1.00 or better
OD	23 (39.6%)	35 (60%)
OS	09 (15.5%)	49 (84.48%)

Table III. Referred Diagnosis

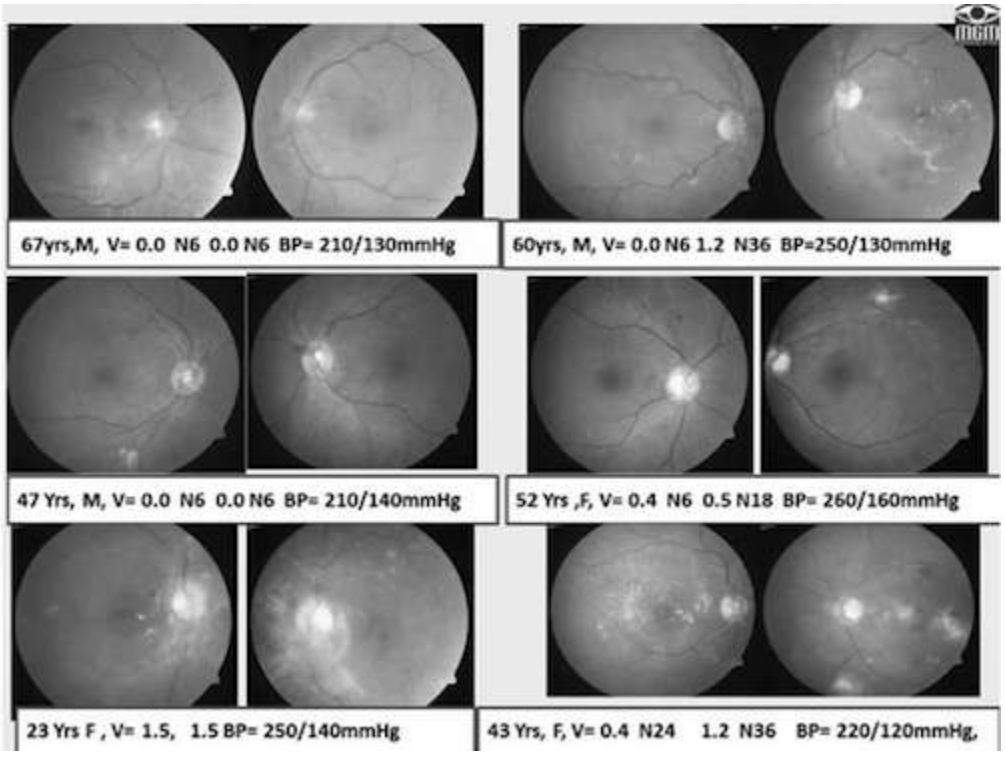
Referred Diagnosis	**No. of cases**
Neuroretinitis	**2**
Ischemic CRVO	**2**
Papilloedema	**2**
Eales' disease	1
Posterior uveitis	1
Total	**8(13.7%)**

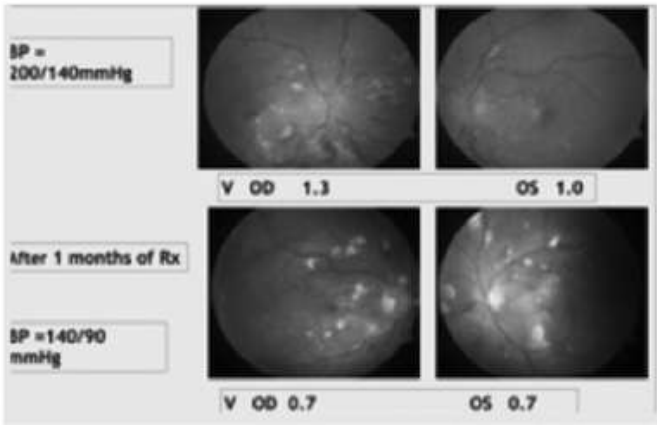


Graph I . Change in visual acuity at presentation, 1 month and 3 months shows improvement



Graph II. Change in Blood pressure at presentation, 1 month and 3 months shows improvement





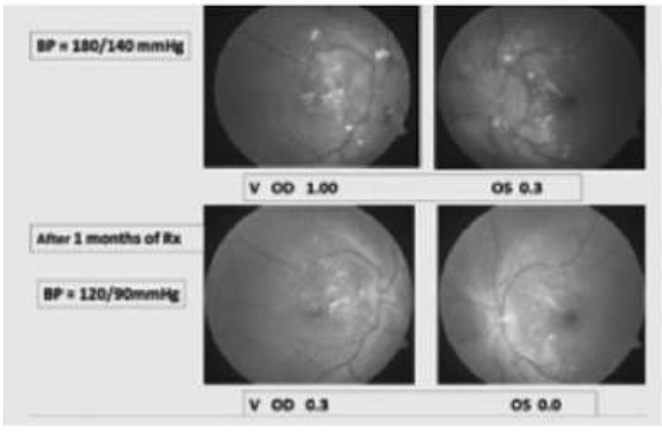


Figure I. Six representative cases with hypertensive retinopathy at presentation
Figure II. Two representative cases with hypertensive retinopathy at presentation and improved upon control of blood pressure at follow-up visit at 1 month.

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INCIDENCE AND RISK FACTORS FOR RETINOPATHY OF PREMATURITY IN A TERTIARY CARE CENTER

Shubhra Das, Madhura Madapaddy

Abstract

Introduction: Retinopathy of prematurity is a disease affecting the retinas of premature infants.ROP is unique in that the vascular disease is found only in infants with immature, incompletely vascularized retinas. The range of possible outcomes for patients with ROP extends from minimal sequelae with no effect on vision, in mild cases, to bilateral, irreversible and total blindness in more advanced cases.

Aim: To study the incidence and risk factors for retinopathy of prematurity in a tertiary care centre.

Materials and methods: This prospective observational study was conducted for a period of one year at a tertiary care center after obtaining ethical clearance and consent of parents. All the neonates fulfilling the inclusion and exclusion criteria were examined using indirect ophthalmoscopy after full mydriasis and followed up according to the schedule. Treatment was done according to the stages.

Results: Out of 109 neonates screened retinopathy of prematurity was seen in 20 neonates, with the incidence being 18.34%.The perinatal risk factors with significant association with ROP in this study were low birth weight ($p<0.001$),oxygen therapy(p value-< 0.001),sepsis (p value <0.002) and phototherapy(p value-<0.023). In our study maximum number of ROP cases detected were in the Stage of Stage 1 zone 3 (50%), 25% cases were in the stage of Stage 1 zone 2. 1 case was in Stage 2 zone 2. Plus disease was seen in 4 cases in the present study. 2 case with stage 3 zone 1 plus ROPand other two cases being Stage 1 zone 1 plus, stage 2 zone 2 plus.

Conclusion: ROP is known to be one of the leading causes of preventable blindness in children especially in the developing countries. Thus, a universal eye screening program with wide coverage is essential for early detection and treatment of the disease there by avoid severe retinal impairment in future.

INTRODUCTION

Retinopathy of prematurity (ROP) is a blinding disease of premature and low birth weight babies with abnormal proliferation of the immature blood vessels at the junction of vascular and the avascular retina[1].ROP begins to develop between 32 and 34 weeks after conception, regardless of gestational age at delivery.[2] Retinopathy of prematurity occurrence is usually associated with premature birth, but the risk for its occurrence represents a consequence of various interactive factors .Various studies have proved that early gestational age (GA)≤30 weeks and low birth weight ≤1500g are the most important risk factors in the development of ROP, but besides these there are other factors, such as poor weight gain, reduced IGF increase, the percentage of oxygen in the inhaled , hypoxia, respiratory distress syndrome, twin pregnancy, anaemia, blood transfusions, fungal infections, sepsis, intraventricular haemorrhage, etc.[3,4] A clinical study was done to evaluate the significance of possible risk factors, in all preterm (GA <32weeks) with birth weight under 1500gm and preterm infants who had unstable clinical condition with birth weight 1500-2000gm.

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MATERIALS AND METHOD

This prospective observational study was conducted for a period of one year between July 2017 and June 2018. The study was done in the Special Care Neonatal Unit of a Guwahati Medical College and Hospital, Guwahati which is a tertiary care center. All the babies admitted in Department of Neonatology were evaluated and those babies fulfilling the inclusion and exclusion criteria were undertaken for the study. The total of 109 babies was evaluated and the results were statistically analyzed.

Ethical clearance was obtained from the hospital ethics committee and informed consent of the parents was also obtained.

Inclusion Criteria[5]

1. Birth weight less than 2000 gm
2. Gestational age less than 34 weeks
3. Gestational age between 34 to 36 weeks but with risk factors such as: a) Cardio-respiratory support, b) Prolonged oxygen therapy, c) Respiratory distress syndrome, d) Chronic lung disease, e) Fetal hemorrhage, f) Blood transfusion, g) Neonatal sepsis, h) Exchange transfusion, i) Intraventricular haemorrhage, j) Apneas, k) Poor postnatal weight gain.
4. Infants with an unstable clinical course who are at high risk (as determined by the neonatologist or paediatrician).

Exclusion Criteria

1. All newborns with birth weight of more than 2000 gm and or gestational age more than 34 weeks with no history of cardiorespiratory support, ventilation, long duration oxygen therapy, apnea of prematurity, blood transfusion.
2. Lost to follow-up cases.

Examination Procedure

A detailed antenatal and perinatal history was taken from the mother. Neonates were examined in the neonatal unit itself under supervision of attending paediatrician/neonatologist.

Fundus was evaluated by dilating the pupil with Phenylephrine 2.5% and Tropicamide 0.4% one drop every 10-15 minutes starting 1 hour before the time of examination. Baby well clothed and wrapped. After instilling topical anaesthetic drop (0.5% Proparacaine), a sterile wire lid speculum was applied. Distant direct ophthalmoscopy done to note the red reflex. Retina was evaluated using indirect ophthalmoscope and 20D lens along with scleral indentor whenever required. Posterior pole as well as 360-degree peripheral retina was examined.

One drop of antibiotic drop was instilled in each eye at the completion of examination. The babies diagnosed with ROP were classified according to International Classification of ROP (ICROP). Follow up was done according to the schedule.

RESULT:

Out of 109 neonates screened retinopathy of prematurity was seen in 20 neonates, with the incidence being 18.34%

NEONATES	NUMBER	PERCENTAGE
ROP	20	18.34
NO ROP	89	81.66
TOTAL	109	100

TABLE 1: Incidence of ROP among screened neonates

The incidence of males babies having ROP was (12 out of 52) 23.07%, and females being (8 out of 57) 14.03%.The mean birth weight of ROP neonates being 1364g and mean period of gestation was 30.35 weeks. The type of pregnancy seen was singleton pregnancy being maximum with 80.7%, twin pregnancy with 15.6%, triplet pregnancy with 2.8%, and quadruplet pregnancy being minimum with 0.9%. Mode of delivery was LSCS in 65.1% and NVD in 34.9%. The perinatal risk factors with significant association with ROP in this study were low birth weight ($p<0.001$), oxygen therapy (p value-< 0.001), sepsis (p value <0.002) and phototherapy (p value-<0.023)

FACTORS (PRESENT STUDY)	p-value
Low birth weight	0.001
Oxygen therapy	0.001
Sepsis	0.002
Phototherapy	0.023

TABLE 2: Significant risk factors associated with ROP.

No significant association between modes of delivery, sex of the baby, multiple pregnancywas seen with ROP.

In our study ,50% neonates were detected with stage 1 zone 3 ROP, 25% neonates had stage1 zone 2 ROP,5% were in stage 1 zone 1 plus ROP,5% were detected with stage 2 zone 2 ROP, 5% were in stage 2 zone 2 plus ROP,10% were detected with stage 3 zone 1 plus ROP.

STAGES OF ROP	NUMBER OF NEONATES	PERCENTAGE
STAGE 1 ZONE 3 ROP	10	50%
STAGE1 ZONE 2 ROP	5	25%
STAGE1 ZONE 1 PLUS ROP	1	5%
STAGE2 ZONE 2 ROP	1	5%
STAGE2 ZONE 2 PLUS ROP	1	5%
STAGE 3 ZONE 1 PLUS ROP	2	10%
Total	20	100%

TABLE 3: Various stages of ROP.

DISCUSSION

a. Incidence of ROP

In the present study out of the 109 low birth weight neonates evaluated ROP was seen in 20 neonates i.e. the incidence being 18.34%.

In a study done by Archambault (1987)[6] with screening criteria of birth weight less than 2000gms showed incidence of 15%.

The incidence of ROP as by LIGHT ROP[7](1998) study was 58%. ET ROP [8](2003) also had similar incidence of 58%

In CRYO-ROP [9,10](1988) study incidence of ROP was 65.8% with birth weight of less than 1251g and in 82% of those with a birth weight of less than 1000 grams.
The difference in incidence could be attributed to the fact that earlier studies done by authors had different screening criteria which lead to different proportion of ROP babies. Though our study involved both preterm and term low birth weight neonates, the ROP babies detected were all preterm babies.

b. Birth weight

In our study the mean birth weight of the babies detected with ROP was 1346g which is accordance with other studies.[11,12,13]

c. Gestational age

In our study the mean gestational age of the ROP babies was 30.35 weeks.
The mean gestational age as per the study done by Charan et al (1995)[11] was 32.47 weeks.
In another study by Gopal et al (1995)[12] which included birth weight criteria <2000 grams had mean POG 32.4 and mean birth weight as 1477 grams.
In a study done by Vinekar et al (2007) [13]were in they had screened the premature babies BW > 1250g the mean birth weight was 1533.9g and the mean period of gestation was 30.9 weeks.
Our study also shows similar results in relation to low birth weight and lower gestational period. Thus, proving the fact that low birth weight and lower gestational age are the two important risk factors for development of ROP similar to other Indian studies.

d. Perinatal risk factors

The various perinatal risk factors among ROP neonates seen in our study was Low birth weight was present in 100% of neonates, history of prenatal leaking membrane in the mother was there in 15% neonates, postnatally 40% neonates were put on ventilator, 75% neonates were given oxygen therapy, 40% of them had thrombocytopenia, 45% neonates had anaemia, 35% required blood transfusion, 60% had postnatal sepsis that required antibiotic therapy and 60% had jaundice that required phototherapy.
In our study the predominant risk factors associated with neonates detected with plus disease were multiple gestation, blood transfusion, phototherapy and sepsis.
Gupta VP et al [14](2004) and Choudhuri et al[15](2009) found oxygen supplementation to be a risk factor associated with development of ROP which is in accordance with present study.
Schaljidelfos NE and Lucey JF et al [16] (2002) have reported phototherapy light being associated with development of ROP but this is yet to be proved.

Stages of ROP

In our study maximum number of ROP cases detected were in the Stage of Stage 1 zone 3 (50)%, 25% cases were in the stage of Stage 1 zone 2. 1 case was in Stage 2 zone 2. All these cases showed spontaneous resolution
Rui-hong Ju et al [17] noted that spontaneous regression was seen in 87% cases of Stage 1 and 57% cases with stage 2. They also noted 100% resolution of all those cases in zone 3, which is almost in accordance with our study.
Plus disease was seen in 4 cases in the present study. 2 case with stage 3 zone 1 plus ROPand other two cases being Stage 1 zone 1 plus, stage 2 zone 2 plus.

Treatment

1. 3 cases received intravitreal bevacizumab injection in both eyes i.e. stage 3 zone 1 plus ROP (2 cases), Stage 1 zone 1 plus (1 case).
2. 1 case received laser treatment i.e., stage 2 zone 2 plus ROP

Follow up

On subsequent follow up regression of vessels was noted in all 4 cases.
In a study done by Hellen et al [18] (2011) concluded thatIntravitreal bevacizumab

monotherapy, as compared with conventional laser therapy, in infants with stage 3+ retinopathy of prematurity showed a significant benefit for zone I but not zone II disease In another study done by ShanH et al [19](2015) wherein a total of 12 infants (24 eyes) with Type 1 ROP and birth weight > 1250 g were enrolled. All infants enrolled had plus disease and ROP in zone II retina.ROP regressed in 23 eyes (96%) following laser treatment. No severe involution sequalae or laser-related complications were recorded.

CONCLUSION

ROP is known to be one of the leading causes of preventable blindness in children especially in the developing countries. Incidence of ROP increases with decreasing gestation and birth weight. However, not all pretermneonates develop ROP. Important risk factors which increase the probability of developing ROP areoxygen therapy, anaemia needing blood transfusion, sepsis and apnoea. Nevertheless, a verypreterm extremely low birth weight neonate can develop ROP even without exposure to oxygen orpresence of these risk factors. Thus, a universal eye screening program with wide coverage is essential for early detection of the disease.

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A Study on the Intra-Operative Complications of Manual Small Incision Cataract Surgery done by Post-Graduate Trainee and their management in Silchar Medical College

Shibashis Deb, Nilanjan Kaushik Thakur

Abstract:

Manual Small Incision Cataract Surgery has been the gold-standard operating procedure for cataract surgery for the past 30 years. This procedure is simple, cost-effective and has an easy learning curve, which is suitable for post-graduate trainees. This procedure allows for the beginners to get familiar with the surgical anatomy of the anterior segment and the various instruments used in cataract surgery and their proper handling. It provides a good platform for the beginners to learn the management of various intra-operative complications during a cataract surgery. This study reviews the various intra-operative complications of manual small incision cataract surgery and their management done by the post-graduate trainee of Silchar Medical College.

Introduction:

Cataract remains the leading cause of avoidable blindness worldwide [1]. Cataract Surgery is currently the most commonly performed ocular surgery, with recent statistics showing that the incidence of cataract surgery is >1000 per 100,000 population per annum.

This tremendous increase in incidence of cataract surgery has been influenced by several factors, including – safer techniques, better day-01 post-operative outcomes, better cosmesis, faster post-operative recovery time and increased public awareness on the safer techniques. However, the safest, most effective and economical technique of cataract surgery remains debatable [1,2]. Over the past decade, manual small incision cataract surgery (SICS) has become an established surgical alternative to phacoemulsification. MSICS still is relevant in the setting of under-developed and developing areas [3].

MSICS, with its easier learning curve, less sophistication, independence from costly phaco machine and faster surgical time requirement – is still a viable alternative for phacoemulsificaton. Surgeons who have mastered MSICS also show a better learning curve for phaco, as the tunnel construction and capsulorrhexis are common to both [4]. Thus, among small incision surgeries, MSICS is ideal for developing countries and for beginners to master. It was propagated for high-quality, high-volume cataract surgery at the Aravind Eye Hospital, India [5,6] and in Nepal [7].

The post-graduate trainees in our institute are therefore started with MCICS first to familiarize

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them with the surgical aspects of the anterior segment, the proper handling of various instruments and the various complications that can occur and their appropriate management.

Objectives:

This study is focused to analyze the various intra-operative complications done during manual small incision cataract surgery (MSICS) by post-graduate trainees, their relative incidence and the management of these complications. This study will also review the various measures that can be taken to avoid these complications.

Materials and Methods:

a. Study Design:
This study is a retrospective, hospital-based study.

b. Setting:
The study is based on the hospital records of cataract surgery done by post-graduate trainees on patients admitted in the Dept. of Ophthalmology, Silchar Medical College. The records of the last 2 years have been studied from 01/01/2016 to 31/12/2017.

c. Participants:

Inclusion criteria:
The records of MSICS done by the post-graduate trainees were selected for this study. It included both cases which did not have any complications, as well as cases which had intra-operative complications.

Exclusion criteria:
All cases which had any pre-existing ocular pathology, except cataract, were excluded from the study. Those case which were not done by post-graduate students including – one-eyed patients, cases with corneal pathology, lens – induced glaucoma, pseudoexfoliation, subluxated lens etc. were also excluded.

Data source:
The data was collected from the case records of the Dept. of Ophthalmology as well as from individual records of the post-graduate trainee.

Study size:
A total of 100 patients were studied from the period 01/01/2016 to 31/12/2017.

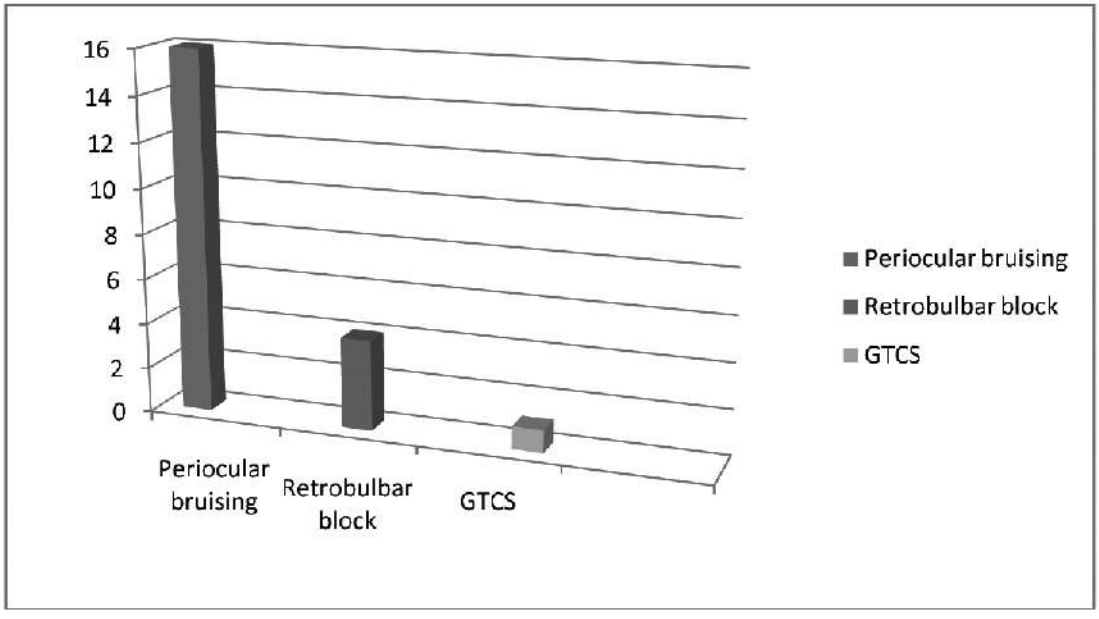
Bias:
There were few bias of the study which included – reporting bias and recall bias which could have been avoided with more robust record-keeping.

Results:
The study reviewed a total of 100 cases which fulfilled the inclusion and exclusion criteria. The study took into account all the complications that had taken place during cataract surgery –

including anesthesia related complications as well as intra-operative complications. The incidence of these complications has been discussed in a step-wise manner.

Of the 100 cases studied, there were total 20 cases which had anesthesia related complications. In our institute, peribulbar block is routinely used as the preferred method of anesthesia which is administered by the post-graduate trainee. Of the 20 cases which had anesthesia related complications, there were 16 cases with periocular bruising (16%); 4 cases of retrobulbar block which resulted in raised IOP (04%); and 1 case of generalized seizure following peribulbar block (01%). Periocular bruising did not require any additional treatment. In case of retrobulbar block with raised IOP, Tab. Acetazolamide (250mg, 1 tab. Stat) was given and O.T. was postponed. In case of generalized seizure, the O.T. was cancelled and the patient was shifted to ICU with ventilation given to the patient.

Table 1: Relative frequency of peribulbar block related complications.



Of the 100 cases, there have been 30 cases (30%) of irregular scleral incision and tunnel. If the tunnel is too shallow, it can lead to button holing, and if the tunnel is too deep it can cause premature entry. Of the 30 cases of improper scleral tunnel, there were 3 cases of button holing (03%) and rest 27 cases were of premature entry (27%). Irregular scleral incision resulted in higher degree of post-operative astigmatism which was managed with spectacle correction. Button-holing of the tunnel was managed by making a newer "frown" incision at deeper plane. In case of premature entry, a newer tunnel is made from the other end at a more superficial plane. Suturing of the tunnel was done at the end of the surgery in all the cases. There have been 2 incidences of iridodialysis associated with premature entry which were managed by iris suturing.

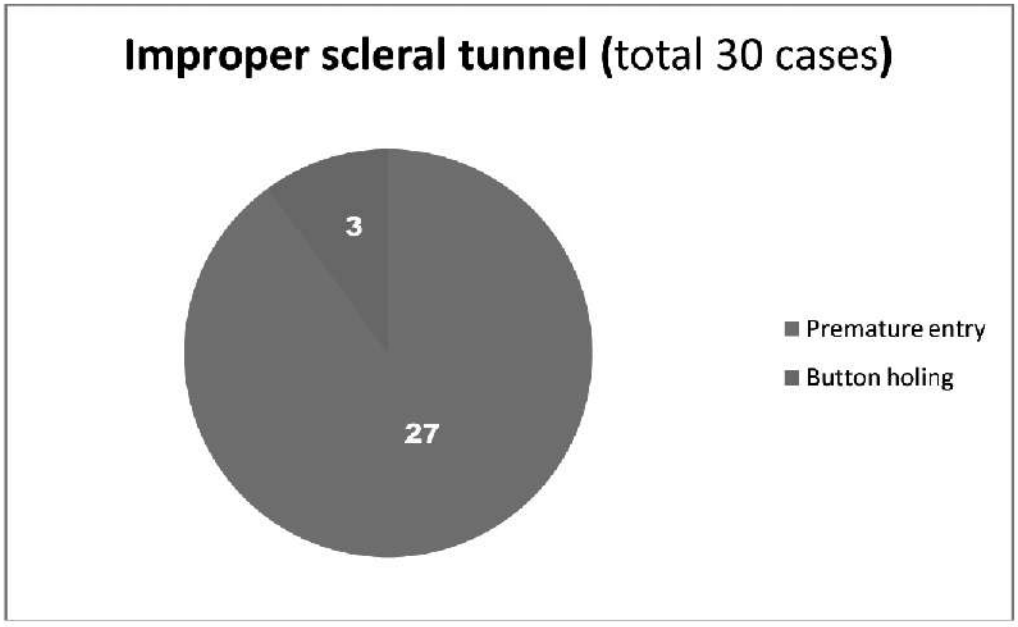


Table 2: Incidence of sclera tunnel related complications.

In our institute, the capsulorrhexis is done by Continuous Curvilinear Capsulorrhexis (CCC) with a bent 26-gauge needle via the 90◦ side-port entry. There have been 27 cases related to improper capsulorrhexis, with run-away of capsulorrhexis being the most common (20 cases; 20%). Other complications included capsulorrhexis size being too small, irregular margins and leaving behind of anterior capsule tags. In case of irregular margins or capsular tags, they were trimmed first with Vannas scissors before prolapsing the nucleus.

There has been high incidence of iritis (24 cases, 24%) and corneal striate keratopathy (58 cases, 58%). In case of increased reaction in the anterior chamber, it was managed with topical steroid antibiotic eye drop (Eye drop Gatifloxacin + Dexamethasone or Moxifloxacin + Dexamethasone, 1 drop 1 hourly), Cyclopentolate eye drop (1 drop 3 times a day) and Inj. Dexamethasone (2 cc. IM stat) if required. Corneal striate keratopathy was managed by using Sodium Chloride 5% eye drops (1 drop 4 times a day).

Another, common complication faced by the post-graduate trainees is rent of the posterior capsule (PCR) (42 cases, 42%). It was most commonly encountered while performing cortical wash. Although vitreous loss in case of PCR in MSICS is not as extensive as ECCE, it does lead to some amount of vitreous disturbance. If the rent is small, the posterior chambers intraocular lens (PCIOL) can be implanted in the bag. There have been 2 cases (02%) where anterior vitrectomy had to be performed and patients were kept aphakic.

Discussion:

Manual small incision cataract surgery (MSICS) has become popular in India in the last decade. Cataract is the leading cause of avoidable blindness in India [8], and cataract surgery forms the major workload of most ophthalmic units in the country. An estimated 4 million people become blind because of cataract every year, which is added to a backlog of 10 million operable cataracts

in India, whereas only 5 million cataract surgeries are performed annually in the country [9,10]. Thus, a technique of cataract surgery that is not only safe and effective but also economical and easy for the majority of ophthalmologists to master is the need of the hour.

Two randomized, controlled trials in Pune, India, had found MSICS to be more effective and economical than ECCE and almost as effective as and more economical than phacoemulsification [11,12,13,14]. MSICS is a much simpler technique, requires less sophisticated instruments and shorter surgical time. UCVA at near was statistically significantly better with MSICS due to astigmatism and safer during the learning phase ($P = 0.003$) [3]. Thus, MSICS is an ideal technique for beginners to master and then switch over to phaco.

Because MSICS is also a type of ECCE surgery, the complications are similar, although there are certain unique ones. MSICS involves more maneuvers in the anterior chamber, first the capsulotomy, then dislodging the nucleus from the posterior to the anterior chamber, and finally removing the nucleus from the scleral tunnel. The surgeon has to enter again for cortical aspiration and intraocular lens implantation. As such the techniques are more demanding in terms of manual dexterity and skill [4].

The various complications of MSICS and their appropriate management have been discussed in various studies. A thorough knowledge of the surgical anatomy of the anterior segment, instruments and the management of various complications is a pre-requisite that every beginner must have before undertaking the surgery.

The first step where complications can take place is while administering the peribulbar block. They range from minor complications like – periocular bruising, corneal abrasion while doing massage etc. to major complications like – retrobulbar block with raised IOP, globe puncture, choroidal hemorrhage etc. Life-threatening complications include myocardial ischemia, cardiovascular depression, respiratory arrest, and convulsions. Convulsions may have several causes such as hypoglycemia, medication errors, stroke, and severe hypoxia caused by deep sedation or following a cardiac arrest complicating cardiac ocular reflex and CNS intoxication by the spread of local anesthetic agent.

The most important step in MCISC is the proper construction of the self sealing sclera-corneal tunnel. In manual SICS, the entire crystalline lens is removed through a self-sealing scleral tunnel incision (5–7 mm) and a rigid polymethyl methacrylate (PMMA) intraocular lens implanted. The expected size and density of the nucleus should determine the size of the tunnel [4,15]. The extraction of immature cataracts requires a small tunnel, just large enough for the intraocular lens (IOL) optic to pass through. Very big, brown nuclei require a larger tunnel size, up to 8 mm in diameter. However, a large tunnel need not be a problem: even larger tunnels are self-sealing and don't need suturing if prepared correctly. The size of the tunnel should be predetermined judging from the grading of the cataract, a too small tunnel can lead to difficulty in nucleus delivery and nuclear contact with the corneal endothelium. The tunnel should be trapezoidal in shape with sufficient pockets on either side which facilitates the delivery of the nucleus. Improper construction of the scleral tunnel can lead to either button holing, if the tunnel is too shallow and premature entry, if the tunnel is too deep. A poorly constructed tunnel with premature entry causes trauma to the iris base and may result in iridodialysis and subsequent

hemorrhage in the anterior chamber [4]. The dialysis can be further extended during nucleus delivery. The premature entry into the anterior chamber makes the tunnel less self-sealing. The continual iris prolapse during the surgery may predispose to superior iris injury and chaffing, and even iridodialysis in extreme cases. A buttonhole can be corrected by making a deeper 'frown' incision and dissecting the tunnel in a deeper plane, starting at the opposite side of the buttonhole [15]. A premature entry is managed by starting a more shallow dissection at the other end of the tunnel [15]. Suturing of the wound is required at the end of surgery. An unfortunate superior iridodialysis can be managed by suturing it into the posterior lip of the incision at the end of surgery. Any doubt in the integrity of the tunnel should be managed by giving one or two sutures. If correctly tied, these will, at the same time, reduce any induced astigmatism [15].

Capsulotomies are easy to perform, but may lead to uncontrolled capsular tear extension, posterior capsule rupture, vitreous loss, and IOL decentration. The best capsular opening is a continuous curvilinear capsulorhexis (CCC): it will guarantee a long-term, 'in the bag' IOL centration. It can be done with a bent cystitome or a bent 26-gauge needle. Failing to complete the anterior capsulotomy, making a too-small CCC, and pulling residual anterior capsular tags can cause the posterior capsule to rupture during cortical wash. These irregular margins and capsular tags should be smoothened out with Vannas scissors [15].

As prolapsing the nucleus into the anterior chamber is the key step in almost all the MSICS techniques, pupillary dilation during surgery is a key facilitator. Small pupils make the nucleus delivery difficult and increase the chances of manipulation of the iris and resultant iritis. Difficulties with nucleus delivery are mostly due to the inner tunnel opening being too small. This should be checked before nucleus removal. While delivering the nucleus through the tunnel, accidental contact between the nucleus and the corneal endothelium must be avoided, else it results in post-operative corneal striate keratopathy or even corneal decompensation. Sufficient use of OVDs with minimal instrumentation is advocated. Also, gently pulling the bridle suture makes nucleus delivery through the tunnel easier [15].

A posterior capsular rent in MSICS most commonly occurs during cortical wash. Wrinkles indicate that the posterior capsule is caught in the aspiration port of the Simcoe cannula[15]. This requires immediate backflushing to avoid posterior capsular rupture. In case of PCR, aspiration of the epinucleus or sheets of cortex becomes difficult. In the event of capsular rent, dry aspiration can be done by a Simcoe canula if the rent is small or by an automated vitrectomy cutter if it is larger. If the rent is small, the posterior chambers intraocular lens (PCIOL) can be implanted in the bag or on the anterior capsular flap for a large rent. If stripping of Descemet's membrane occurs while cleaning the cortex, great care should be taken not to tear it off. If this happens, air should be injected into the chamber at the end of the operation to push Descemet's membrane against the cornea.

Striate keratopathy is common during MSICS if enough care is not taken to place the viscoelastic between the nucleus and the cornea [4]. The side-port is an excellent route to ensure this. Delivery is facilitated through a trapezoidal tunnel. Delivery of the nucleus through a small tunnel or rectangular tunnel can cause damage to the corneal endothelium and long-standing corneal edema, which is recalcitrant to treatment. Also a through aspiration of OVDs is required before closure of the surgery to prevent post-op rise in IOP.

Conclusion:

MSICS is a safe surgery [11,12,13,16,17]. The surgeon has to be extra diligent in tunnel construction as the tunnel size is larger. An excellent self-sealing incision is vital for wound architecture on which the safety and lowered astigmatism potential rests. The incidence of posterior capsular rent and iridodialysis is low, and in case of such an eventuality, it is easier to manage the vitreous loss. The prolapse of nucleus into the anterior chamber and its delivery through the tunnel involve manipulations very close to the iris and the cornea. The surgeon has to be extra careful with these structures, as postoperative inflammation and corneal edema can be all too common. More attention needs to be paid to cortical wash and capsular polishing, as PCO may be the only factor for suboptimal visual acuity in the future. Post-graduate trainees who are at the beginner level should advance in a step-wise manner, with proceeding to next step only after full confidence has been achieved in the previous steps. Post-graduate trainees are encouraged to have through theoretical knowledge of each step beforehand. Practice of each step in a wet lab with animal models, hands-on training and live/recorded video tutorials are also highly encouraged. Proper case selection with avoiding cases with zonular weakness, pseudoexfoliation, subluxated lens and sufficient use of viscoelastics is recommended for the beginners.

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Non-Strabismic Binocular Vision Anomalies (NSBVAs) based on accommodative function in young adults

Mousumi Saikia PhD, Prof (Dr) Jaydeep Datta

Abstract

Purpose: To investigate the accommodative function to rule out Non Strabismic binocular vision anomalies in young adults

Methods: 175 subjects; 90 female and 85 male; mean age 21.45±3.46 years evaluated with facility and status of accommodative test with their best corrected visual acuity and further the quantitative findings were correlated with full orthoptic work up values to know whether these two tests alone can lead to a diagnosis or not.

Results: 46.26% of the subjects were found with Non Strabismic binocular vision anomalies which include 14.84% Convergence insufficiency, 17.14% with accommodative insufficiency and 10.28% with convergence insufficiency secondary to accommodative sufficiency, 4% with accommodative infacility. A positive correlation between binocular accommodative facility and status of accommodation with a p value <0.00001(r=0.56)

Conclusion: Status of accommodation and facility test can help to differentiate the accommodative and vergence problems solely. Both these two procedures should be a part of general eye examination in young adult age group.

Introduction:

Non-strabismic accommodative and vergence binocular anomalies affect clarity and binocularity, and impair comfort and efficiency of visual performance when near tasks such as reading, writing and computer-based work is performed 1,2,3,4,5 Accommodative anomalies and non-strabismic binocular dysfunctions are vision disorders which affect the binocularity and visual performance of subjects, particularly when close vision is needed. Studies reported that children who have binocular anomalies experience anxiety, emotional and social problems.[6]

Children with uncorrected NSBVAs may be misdiagnosed as being dyslexic.[7,8,9] Hence it is important to assess the Non Strabismic binocular vision anomalies in young adult group population. In recent years, many authors have reported the clinical significance of testing accommodative response and facility as well as amplitude 10, 11. An important concept is that an individual may experience asthenopic symptoms and have an accommodative disorder even when the accommodative amplitude is normal 12. Several studies have investigated the relationship between accommodative facility and the presence of symptoms. Both Hennessey et al.[12] and Levine et al.[11] reported that symptomatic subjects perform significantly poorer than asymptomatic subjects on both monocular accommodative facility (MAF) and Binocular accommodative facility (BAF) test.

Any test performed under binocular conditions is affected by both accommodative and binocular function. Thus, although MEM is considered a test of accommodative function, binocular vision is

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also being assessed.

Objectives:
Objective of our study was –
1) To measure Monocular and Binocular accommodative facility in young adult 2) To measure the status of accommodation in young adult 3) To find out the correlation between accommodative facility and status of accommodation 4) To find out the Non Strabismic binocular vision anomalies in young adult group

Methodology
Inclusion criteria included Age group between 17 years to 30 year, people with best corrected distance visual acuity of at least 6/9 monocularly (0.20 Log unit) and near visual acuity of N6 at 33cm. All selected subjects having refractive error, were spectacle user only. Phorias were included. We excluded those with any eye disease or had undergone any eye surgery and manifest deviation

We conducted a retrospective study of 175 subjects where 90 female and 85 male; mean age (21.45±3.46); at Optsight Eye Care, Sivasagar, Assam. Accommodative facility (AF) with flipper of +2/ -2D was measured both monocularly and binocularly in all subjects at 40cm testing distance. Before assessing the accommodative facility, visual acuity both distance and near along with objective and subjective refraction for best corrected visual acuity was done. General slit lamp examination was done for anterior and posterior segment assessment. In continuation, accommodative status was measured objectively with MEM (monocular estimation method) technique and noted against AF. Later subjects with abnormal facility and status of accommodation were further evaluated with detail orthoptic workup to confirm the diagnosis.

Results
On accommodative facility testing, out of 175 subjects 91 were found normal. 23.33 % (n=41) and 24.57% (n=43) were having problem with monocular and binocular accommodative facility respectively. 21.1% lead and 25.1% lag of accommodation were noticed among the subjects where 37 subjects showed lead and 44 showed lag of accommodation. All those 81 subjects showed abnormal status of accommodation, further evaluated with detail orthoptic tests and surprisingly 46.26% of the subjects were found with Non Strabismic binocular vision anomalies includes 14.84% Convergence insufficiency, 17.14% with accommodative insufficiency and 10.28% with convergence insufficiency secondary to accommodative sufficiency , 4% with accommodative infacility.
We found out a positive correlation between binocular accommodative facility and status of accommodation with a p value <0.00001($r=0.56$).

Discussion:
Garci et al.[13] reported in their study that monocular accommodative facility results showed more information about the dysfunction of the patient compared with the results of the binocular accommodative facility results. A relation between reduced accommodative facility and a general binocular dysfunction (accommodative or binocular) were found in 48 subjects, ages 10–30 (those were pre-diagnosed), which demonstrated the importance of the accommodative facility test in diagnosing an accommodative or binocular anomaly.

In our study we have observed reduced monocular accommodative facility correlated with accommodative related issues and binocular accommodative facility mainly correlated with vergence issues. Along with that we found a positive relation between facility and status of accommodation. Our study showed 14.84% Convergence insufficiency, 17.14% with accommodative insufficiency and 10.28% with convergence insufficiency secondary to accommodative insufficiency, 4% with accommodative infacility among the young adult age group. Whereas in another study Darko-Takyil et al (2016) found 21.9% and 12.4% non-strabismic accommodative and vergence dysfunctions respectively in ages ranging from 19 to 27 years.

In the literature, the search for accommodative problems associated with myopia has resulted in some inconsistencies. For example, amplitude of accommodation has been found variously to be reduced in myopes,14 increased in myopes,15 and unaffected by the refractive error16 Again, although a reduced accommodative response is found in myopes,17 there is some controversy about whether responses worsen as myopia progresses18 or whether there is no relationship between progression and accommodative. Response19 Accommodative dynamics, assessed by facility of accommodation measurements are reduced for distance viewing in myopes, 20 but not for near work21. Our research could not give any specific result in near monocular and But the binocular accommodative facility of myopic subjects compared to emmetropia was found p=0.0007. No any significant difference was seen in accommodative facility between hypermetropia and emmetropia (p=0.18); both monocularly and binocularly. Study with a larger sample size could help in finding the difference of monocular and binocular accommodative problems more detail in ametropia.

Conclusion

It has been observed that accommodative facility testing always play a vital role in diagnosing Non Strabismic binocular vision anomalies. Our study has also shown status of accommodation and facility testing can lead us to differentiate the accommodative and vergence problem alone. So we suggest that these two procedures be included in the general eye examination in young adult age group.

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Letters to Editor….

Management of DMO - NHS approach

Management of Diabetic macular edema, DMO in England and Wales is slightly different from the rest of the world. NHS follows National Institute for Health and Clinical Excellence (NICE) guidelines to reduce variation in availability and quality of NHS treatments and care, taking into consideration cost effective use of NHS resources.

Nice guidelines states:
Visual impairment due to DMO is treated with intravitreal anti-VEGF agents only if

(1) The eye has a central retinal thickness (CRT) of 400 micrometer or more at the start of treatment.
(2) The company provides anti VEGF agent with the discount in patient access scheme.

In eyes with CRT less than 400 micrometer, laser photocoagulation can be considered or a 'wait and watch' approach is considered till the thickness reaches 400 micrometer.

As there is evidence of gain in visual acuity associated with anti-VEGF agent with thicker retina and severe impairment of vision hence anti-VEGF is not considered cost effective in such cases.

Aflibercept is given as a dose of 2mg monthly injection for 5 months as an initial loading dose and then 2 monthly till 12 months without any monitoring between treatments. After the 1st year treatment interval maybe extended based on visual and anatomic outcome. It has to be discontinued and switched to other agent if not beneficial. It is estimated that the patient gets around 8 injections in the 1st year, 4 in the 2nd and 3 in the 3rd year approximately.

Ranibizumab is given as a monthly dose of 0.5mg, till maximum VA, minimal OCT thickness and stable VA for 3 months is achieved. In practice, 6 doses are given at monthly interval, initially as a loading dose and then monitored monthly or in 6 to 8 weeks depending on stability. Treatment interval is increased to 2 months after the initial loading dose.

Dexamethasone implant is recommended as an option for treatment only if
(1) Eye is pseudophakic
(2) DMO doesn't respond to non corticosteroid treatment or such treatment is unsuitable.

Flucinolone acetonide is recommended for chronic DMO which is insufficiently responsive to available therapies.

Though it is not a NICE criteria, treatment is usually not started with VA at or better than 6/9.5 unless the treating physician has a rational to do so. At every visit the importance of good control of HbA1C, B.P and cholesterol is emphasized and liaised with GP or Diabetologist if necessary.

Thank you.
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An ideal abstract

The Scientific Committee, Ophthalmological Society of Assam, invites your kind attention to an example of an ideal abstract, which was received for presentation at 51st OSACON, 2018, Silchar, Assam

Title (not exceeding 30 words): Face trabeculectomy challenge with courage

Abstract (not exceeding 150 words)

1. **Course objectives**: To encourage more and more ophthalmologists perform trabeculectomy

2. **Course description**: As far as treatment of glaucoma is concerned, intraocular pressure is the only modifiable risk factor. It is now well documented that one important cause of failure of glaucoma treatment is under treatment, especially if the target IOP is not achieved with medical treatment. This is due to the fact that many ophthalmologists are reluctant to proceed to the next level of care that is surgical treatment with trabeculectomy. It is primarily because of the apprehensions of encountering the associated risks of complications, which may be occasionally vision threatening. We intend to demonstrate through video-assisted lectures that management of these complications is not difficult, so that more and more people embrace this simple but most effective surgical option.

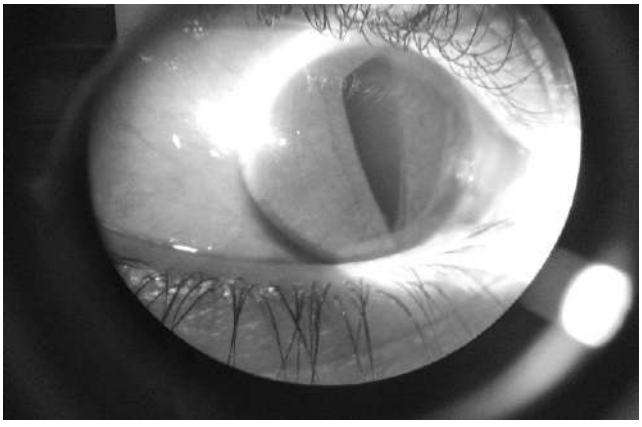
3. **Course duration, breakup of each speaker's topic and allotted time**:

a) Tips to avoid complications	- 15 mins - Dr. Prafulla Sarma
b) Management of shallow chamber and bleb leak	- 15 mins - Dr. Chengsira Sangma
c) Rescue of failing bleb	- 15 mins - Dr. Prafulla Sarma
d) Management of choroidal effusion and beyond	- 15 mins - Dr. Sahunur Tayeb

Authors: Dr. Prafulla Sarma, Dr Shahinur Tayab, Dr Chengchira A Sangma

Institution: Sri Sankaradeva Nethralaya, Guwahati

Spot Diagnosis



A 25yrs old female presented with allergic conjunctivitis, and on examination it was detected that the patient is having congenital abnormal pupil in her right eye. Other eye was normal. The lady had slit like pupil , which was slightly wider superiorly and narrow inferiorly and pupillary light reaction was normal. The lady had 6/6 vision in both the eyes. The size of the eyes were normal average size and the fundus was normal .The patient was not aware of her this abnormality.

Contributed by Dr. Jaya Nath
Lions Eye Hospital, Silchar

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