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Journal of Ophthalmological Society of Assam (JOSA)

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Editorial

Dr Madhurjya Gogoi, MD (AIIMS)

The ***J***ournal of ***O***phthalmological ***S***ociety of ***A***ssam (JOSA) is the official scientific peer-reviewed, society owned publication of OSA. JOSA received its first article on 21.07.2017. The first issue was released on 8th December 2017 at the Golden Jubilee Conference at Guwahati. As it completes 3 years in November 2019, we take a look at how JOSA has performed.

Year	Received	Accepted	Under Review	Withdrawn	Rejected
2017	8	7	1	0	0
2018	7	6	0	0	1
2019	15	9	2	2	2
Total	30	22	3	2	3

The beginning has been modest at best. What lies ahead is important.

As a first step, JOSA has been able to put in place a peer-review process. Reviewers have been selected on a very minimalistic criteria of at least 1 (one) peer reviewed publication in the preceding 10 years. Encouragingly, all the reviews have come in within a month of being sent. This has greatly facilitated the subsequent correspondence with contributing authors.

Secondly, JOSA is being published once a year at this time, but the aim is to make it at least biannual, comprising at least 6 articles per issue, so as to eventually make it eligible for metrics like indexing, abstracting and impact factor.

An effort to improve the quality of articles is the IJO-JOSA session at OSA Annual Conference, a practice that was started in 2017. The 2019 session shall cater primarily to the beginner. It is strongly advisable to be familiar with the following:

1. Latest recommendations of the International Committee of Medical Journal Editors (ICMJE) -2018
2. The New Drugs and Clinical Trials Rules, 2019, especially the definitions and sections related to Academic Clinical Trials and Clinical Trials

The soft copy is being delivered by email to all life members. All members are requested to intimate/update email ID at ophsocassam1967@gmail.com. The print copy of the journal shall be available to life members at the venue of Annual Conference.

We offer our heartfelt thanks to all contributors, editorial board members, reviewers, and well wishers. Suggestions are most welcome at journal.osa@gmail.com.

Sincerely yours,

Dr.Madhurjya Gogoi, MD
Editor, Journal of Ophthalmological Society of Assam
Guwahati, Assam

About the Journal

1. **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.

2. **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.

3. **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 (*updated December 2018*). 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.

4. **The Editorial Process**

4.1. A manuscript will be reviewed for publication subject to the Author's declaration
4.2. The journal requires a corresponding author who will be responsible for all communication with the Journal related to the manuscript.
4.3. All manuscripts received are duly acknowledged and given a Manuscript Number.
4.4. 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.
4.5. 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.
4.6. The Journal follows a double-blind review process, wherein the identity of reviewers and authors are concealed from each other.
4.7. 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.
4.8. Manuscripts accepted for publication are copy edited.
4.9. 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.
4.10. *All* communication shall be by *email only* until further notice.
4.11. The journal publishes articles online as 'Ahead of Print' on acceptance

5. Clinical Trial Registry

5.1. 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

5.2. For studies conducted in India, registration is required at http://www.ctri.in/

5.3. Guidelines on registration for PG thesis conducted in India is available at http://ctri.nic.in/Clinicaltrials

5.4. 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

5.5. For clinical trials that have begun enrollment of subjects before June 2008, retrospective registration with clinical trial registry is admissible.

6. Authorship Criteria

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

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

6.2.1. Concept and design of study or acquisition of data or analysis and interpretation of data;

6.2.2. Drafting the article or revising it critically for important intellectual content; and

6.2.3. Final approval of the version to be published.

6.3. 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.

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

7. Contribution Details

7.1. 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.

7.2. 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.

7.3. At least one author should take responsibility for the whole work and he/she shall be designated as *'guarantor'*.

8. Conflicts of Interest/ Competing Interests

8.1. 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.

8.2. The conflict of interest form is separate from the copyright transfer form

9. Submission of Manuscripts

9.1. All manuscripts must be submitted by email.

9.2. Authors do not have to pay for submission, processing or publication of articles.

9.3. The manuscript should be submitted in the form of two separate files: **Covering Letter** and **Article File**

9.4. **Covering Letter/Title Page/First Page File**

9.4.1. This file should provide The type of manuscript (Original Article, Review Article, Case Report/Short Case Series, Letter the Editor/Letter in Response, Guest Editorial, Research methodology, OSA Meeting Papers) title of the manuscript, running title, names of all authors/ contributors (with their highest academic degrees, designation and affiliations) and name(s) of department(s) and/ or institution(s) to which the work should be credited. All information which can reveal your identity should be here. Use files, preferably Microsoft Word files saved as .doc or .docx. Do not zip the files.

9.4.2. The total number of pages, total number of photographs and word counts separately for abstract and for the text (excluding the references, tables and abstract), word counts for introduction + discussion in case of an original article.

9.4.3. Source(s) of support in the form of grants, equipment, drugs, or all of these.

9.4.4. Acknowledgement, if any. Acknowledgement should not be in the main article file.

9.4.5. If the manuscript was presented previously, a full statement to that effect should be included with Subject title, place and date of presentation

9.4.6. Registration number in case of a clinical trial

9.4.7. Conflicts of Interest form

9.4.8. Authors' declaration and copyright transfer form

9.4.9. The name, address, e-mail, and telephone number of the corresponding author and Guarantor

9.5. **Article File**:

9.5.1. The file must not contain identity of the author(s), institution, or acknowledgements (Blinded)

9.5.2. The main text of the article, beginning from Abstract, Key Words, Body of the Manuscript, References, Tables, Legend to Figures should be in this file in the specified sequence

9.5.3. The pages should be numbered consecutively, beginning with the first page of the article file.

9.5.4. Page headers/running title may include the title but not the authors' / institution identity.

9.5.5. Use Microsoft Word .doc or .docx files.

9.5.6. Do not zip the files.

9.5.7. Limit the file size to 1 MB.

9.6. **The Authors' Declaration and Copyright Transfer Form** has to be submitted in original with the signatures of all the contributors within two weeks of submission via email as a scanned image. It will not be possible to process the submission otherwise.

9.7. **Copies of any Permission(s) of Copyright material**

9.7.1. It is the responsibility of authors to obtain permissions for reproducing any copyrighted material. A copy of the permission obtained must accompany the manuscript. Kindly note that ***Plagiarism is unethical and liable for punitive action.***

10. **Types of Manuscripts**

10.1. Editable checklists for reporting guidelines can be found on the EQUATOR Network site (www.equator-network.org)

10.2. 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	Structured: Aim	3000 words	5	40	Randomized Controlled Trial (RCT), Prospective,	CONSORT,STROBE,RECORD,

ARTICLES	Methods, Results, Conclusion Maximum 250 words				Retrospective Observational / Interventional Study, Non-randomised Trial, Descriptive Data, Cost Analysis, Animal Studies	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 documentation required. Case series must contain 3-10 cases.	CARE
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 METHOD OLOGY	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.

11. **Ethics**

11.1. 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.

11.2. 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.

12. **References**
    - 12.1. JOSA recommends formats used by the National Library of Medicine (NLM) *in Index Medi*cus (https://www.nlm.nih.gov/bsd/uniform_requirements.html).
    - 12.2. References should b*e numbered c*onsecutively in the order in which they are first mentioned in the text (not in alphabetic order).
    - 12.3. Iden*tify references in text,* tables, and legends by Arabic numerals in superscript with square bracket after the punctuat*ion marks.*
    - 12.4. *References cited onl*y 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.
    - 12.5. Names of non-indexed journals should be mentioned in full.
    - 12.6. Avoid using abstracts,"personal communication", and "unpublished observations" as references.

13. **Tables**
    - 13.1. Tables should be self-explanatory and should not duplicate textual material.
    - 13.2. Each table should be limited to maximum of 5 columns and 20 rows.
    - 13.3. Number tables, in Arabic numerals, consecutively in the order of their first citation in the text and supply a brief title for each.
    - 13.4. Explanatory notes, credit lines to be placed in footnotes.
    - 13.5. Explain in footnotes all non-standard abbreviations that are used in each table.
    - 13.6. For footnotes, symbols and their order should be: *, †, ‡, §, ‖,¶ , **, ††, ‡‡
    - 13.7. Tables with their legends should be provided at the end of the text after the references in the manuscript file, not as separate file
    - 13.8. The table number should be cited at the relevant place in the text.

14. **Illustrations/Figures/Images**
    - 14.1. 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
    - 14.2. Submit good quality color images as JPEG files less than 2 MB in size, 1600x1200 pixels, 5-6 inches.
    - 14.3. Graphs can be submitted as images separately without incorporating them in the article file.
    - 14.4. Files should not be zipped.
    - 14.5. Figures should be numbered consecutively according to the order in which they have been first cited in the text.
    - 14.6. 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.
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    - 14.8. 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.
    - 14.9. The Journal reserves the right to edit illustrations.

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

16. **Protection of Patients' Rights to Privacy**
    16.1. Identifying information should not be included in written descriptions, photographs, scan reports, etc., and pedigrees.
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17. **Sending a Revised Manuscript**
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18. **Reprints and Proofs**
    18.1. 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.
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    24.4. Numerals at the beginning of the sentence spelt out
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    24.7. Species names should be in italics
25. For ready to use templates authors may refer to www.ijo.in
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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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Authors' Declaration and Copyright Transfer form

(One signed copy to be submitted with the manuscript)

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Manuscript Number:

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I/we declare that this manuscript under my/our authorship is being submitted to Journal of Ophthalmological Society of Assam alone at that point in time, and has not been published/already accepted for publication, nor is being considered for publication elsewhere

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Journal of Ophthalmological Society of Assam (JOSA)

Articles may be submitted to JOSA at any time
For 'Instructions for Authors', kindly visit www.osa.ind.in/journal.htm.

It is mandatory to submit the following

1.1. ICMJE Form for Disclosure of Potential Conflicts of Interest
1.2. 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

1. 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.
2. 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.
3. 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.
4. Reviewers are asked to assess submissions based on depth of original research, accuracy, appropriate documentation, readability, and suitability of content.

5. **Reviewers shall make One of Four Recommendations**:

    5.1. Acceptance
    5.2. Provisional Acceptance With Revision
    5.3. Provisional Non-Acceptance
    5.4. Rejection

6. For points 5.2, 5.3 and 5.44, reviewers are required to include comments explaining the recommendation.
7. Authors may expect to know the results of the manuscript peer review within four weeks from the date of submission.
8. Authors shall receive the reviewers' comments and be advised to revise their manuscripts in line with the reviewers' and/or editor's suggestions.
9. 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.

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Invited article
DNA Gene Therapy

Harsha Bhattacharjee

Life is a DNA driven programme and death results from its technical failure. Protein and DNA were evolved naturally from the gas present in the environment of the primitive world. It was the effect of high voltage thunderbolts and asteroid hits on the surface of the earth. Millions of years ago passage of the high voltage energy across the environmental gas created protein and DNA. Origin and evolution of DNA and DNA replication mechanism are not yet fully known, but the major event that matter, is the transition from RNA to DNA world. Recent findings from comparative genomics, traditional biochemistry and structural biology suggests transition from RNA to DNA genomes was complex and involved several enzymatic activities and in fact puzzling [1].

In human biology scientists in 1950 realized that a virus can inject their DNA in the host cells. This realization gave rise to a new thinking-can human DNA be modified by injecting a new DNA carried in the back of a known attenuated vector virus? If it is possible, can it be a treatment modality for hereditary diseases? The scientists started experiments using viral technology. The attenuated viruses are created in the laboratory. These viruses do not have any replication ability. Vector virus carries the DNA on their back and offloads it in the cells of the host and eventually die.

In medicine Friedman and Robin (1972) published an article showing for the first time, the possibility of treatment of human genetic disorders by gene therapy. They postulated that desired genetic code which is to be injected in the DNA of the host is simply to be loaded on the virus- which will act as a carrier. But in reality the implementation of the idea was not found at all simple and risk free. Since then the gene modification and gene therapy trial began[2].

Blasé et al first treated a 4 year old patient suffering from severe combined immunodeficiency disease (SCID) which is caused by adenosine deaminase deficiency. In this trial CD34 + autologous peripheral blood cells were used for treatment[3]. In 1999, one volunteer received gene therapy for ornithine transcarbamylase deficiency and died four days after therapy due to multiple organ failure[4].

These early trails were encountered with therapy related toxic effects and even life threatening complications.

The initial trials were extremely hazardous but presently Gene therapy as a treatment modality for hereditary diseases has been approved across the globe. Common variety of gene therapy is 'replacement gene therapy' or 'augmentation therapy'. In future there is possibility of gene correction and other targeted gene modification and for that technique of clustered regulatory inter spaced short palindromic repeat (CRISPR) / Cas 9 nuclease technology will be used in the field of ophthalmology. CRISPR is a simple and powerful tool for editing genomes. By this technology DNA sequences can be altered to modify gene functions. The protein Cas 9 is an enzyme-this enzyme is like a scissor that can cut strands of DNA[5].

Recently FDA USA for the first time approved Luxturna (on December19'2017) for treatment of inherited retinal disease caused by mutation in both copies of RPE65 Gene. Of course there should be enough remaining viable retinal cells. It does not cure but sustainability improves vision. Since 2018, Luxturna is commercially available and the price in USA is $425,000/- per year. Luxturna is an adeno assisted virus type 2 and through the vector the corrected copy of the RPE 65 gene is delivered with undisturbed genome. The drug is injected in the subretinal space. RPE 65 gene is responsible for regeneration of visual pigment.

After the success of Luxturna practically, a new era of Gene Therapy has emerged. The active retinal gene replacement multiple phase ½ trial has been started. In this research Stargardt's disease, retinitis pigmentosa, Usher syndrome etc. have been targeted. For these trial adeno-assisted virus (AAV) or Lente virus are used as Vector.

So it is critical for offering best possible care to our patients and up to date knowledge is equally critical. Success will depend on early and accurate diagnosis, appropriate work up, clinical characterization, genetic testing

and genetic counseling. In the field of Gene Therapy multiple issues and questions are to be solved, but it is certain that a realistic hope has been generated for the patients suffering from genetic disorders.

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ROP: CASE SERIES

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Dr.Chintan Desai , Dr.Divakant Misra , Dr.Jitender Kumar

Introduction:-

Retinopathy of prematurity (ROP) was originally termed as retrolental fibroplasias by Terry in 1952 who related it with premature birth.[1] Heath was the first one to coin the term ROP in 1951.[2] ROP is a disorder of vascularisation of retina especially in premature babies. Normal retinal vascularisation reaches nasal ora by 32-34 weeks and temporal retina by 10 months of gestation (39-41 weeks). Adult vasculature is attained by 5 months of age after birth.[3] Retinopathy of prematurity (ROP) is a disease related to low birth weight, prematurity, oxygen administration, and other unidentified factors. Improved neonatal survival rate of premature babies with unmonitored oxygen supplementation has lead to increase in ROP cases. Currently, India is going through the third epidemic of ROP, also called as the 'Modern epidemic' with incidence of ROP varying from 38% to 51.9% among low birth weight babies (< 2000grams).[4] Blindness due to ROP being preventable, ,timely identifying and screening of at-risk premature infants by an experienced ophthalmologist remains the most important strategy in the management of ROP. We report 3 cases of ROP highlighting the importance of time and mode of intervention.

Case Report

Case 1: Premature female child with history of in vitro fertilisation (IVF) was born out of caesarian section at 32 weeks gestational age with birth weight of 2200 grams. Baby was brought to us at 36 weeks post menstrual age (PMA) and weight 2200 grams. History of Rh factor incompatibility was present and baby was kept on a ventilator for 72 hours & oxygen supplementation for 18 hours. Fundus examination revealed normal vascularisation extending upto middle of zone 2 and ridge in the temporal 6 clock hours with elevated dichotomous branching vessels extending into the vitreous cavity over the ridge in both eyes. Diagnosis of zone 2 Stage 3 ROP without plus disease was made [Fig 1] in both eyes and child was kept on one weekly observation for the first 3 visits and fortnightly thereafter. Vascularization reached anterior edge of zone II by 40 weeks and complete vascularisation in zone 3 was noted by 48th week PMA. Fundus evaluation was done with indirect ophthalmoscopy and photo documentation with fundus imaging camera (RETCAM). No intervention was required.

Case 2: Premature male child, gestational age 32 weeks, birth weight 1990 grams, oxygen supply of 4 days, born out of cesarian section presented at 38 weeks PMA with zone 2 stage 3+ disease in both eyes. Pupillary dilatation was adequate for laser photocoagulation to be performed in both eyes. One week later, injection ranibizumab 0.16 mg was injected in both eyes due to further progression of plus disease as noted by decreased pupillary dilatation and increased tortousity of the retinal vessels.

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Improvement in condition with disappearance of the ridge and popcorn vessels, decrease in vessel tortousity and improvement in pupillary dilatation was seen in subsequent weeks followed up to week 50 PMA.[Fig 2]

Case 3: A premature female child with 29 weeks gestational age, birth weight 1055 grams and history of oxygen supply in NICU for 10 days after birth presented at 31 weeks and 1280 grams with aggressive posterior retinopathy of prematurity with poor pupillary dilatation, iris neovascularization and severely dilated and tortuous vessels terminating within zone 1. The child was managed aggressively with intravitreal injection ranibizumab 0.16 mg in both eyes, followed by laser photocoagulation on the same day. Three more sessions of laser augmentation were performed at weeks 36, 39 and 48. Progressive decrease in tortuosity and dilatation of vessels was noted in each follow up visit. Improvement and stabilisation of disease was noted at 54 weeks PMA with vascularisation complete till the termination of zone 1 and visible laser scars over the avascular retina. [Fig 3]

	CASE 1	CASE 2	CASE 3
SEX OF BABY	FEMALE	MALE	FEMALE
PERIOD OF GESTATION	32 WKS	32 WKS	29 WKS
BIRTH WEIGHT	2200 Grams	1300 Grams	1055 Grams
WK OF PRESENTATION (PMA)	36 WKS	38 WKS	31 WKS
WEIGHT AT PRESENTATION	2600 Grams	1720 Grams	1280 Grams
ASSITED PREGNANCY	YES	NO	NO
OXYGEN SUPPLEMENTATION	YES	YES	YES
RH MISMATCH	YES	NO	NO
DIAGNOSIS	ZONE 2 STAGE 3 (OU)	ZONE 2 STAGE 3+ (OU)	APROP (OU)
TREATMENT	OBSERVATION	SECTOR PRP (OU) +1/3 rd Ranibizumab	1/3 dose Ranibizumab (twice) + PRP 4 sittings

DISCUSSION

All premature infants with birth weight <2000grams and/or gestational age less than 34 weeks should be screened by an ophthalmologist not later than 4 weeks after birth for ROP. Babies with birth weight <1200g or age <28 weeks should be screened within 3 weeks. It should also be done for gestational age > 35 weeks, if there are additional risk factors like unmonitored O2 supply > 30 days, history of respiratory distress syndrome, anaemia, poor weight gain, sepsis, multiple blood transfusions, multiple births(twins/triplets, etc.), apneic episodes and intraventricular haemorrhage.[5] Clinically ROP cannot be picked up in 1st week of life so follow up schedule after

the first visit is extremely important and depends on the state of vascularisation at initial presentation and stage of disease as per the International classification for ROP (ICROP).

The goal of treatment in ROP is to remove the stimulus for growth of new blood vessels by either ablating the peripheral avascular retina or by use of anti VEGF injections. Indications for treatment by pan retinal photocoagulation are well defined by the ETROP (Early treatment of ROP) study.[6] Treatment is indicated in Type 1 disease, while bimonthly follow up for Type 2. Follow up is usually recommended till vascularisation is complete at around 48-52 weeks. BEAT-ROP (Bevacizumab in the treatment of ROP) study established the efficacy of Intravitreal anti VEGF in the management of ROP.[7] Currently, anti VEGF injection are indicated in a) aggressive posterior ROP (APROP) especially in cases non dilating pupil obscuring peripheral fundus visualization for laser and b)persisting plus disease post laser therapy. Combined treatment with anti VEGF therapy with deferred laser in cases of APROP has also yielded good results.

ETROP TREATMENT GUIDELINES		
TYPE 1 ROP (HIGH RISK PRE-THRESHOLD DISEASE)	ZONE 1 ANY STAGE WITH PLUS	TREAT WITHIN 48-72 HOURS ADMISTER LASER
	ZONE 1 STAGE 3 WITHOUT PLUS	
	ZONE 2 STAGE 2 OR 3 WITH PLUS	
TYPE 2 ROP (LOW RISK PRE-THRESHOLD DISEASE)	ZONE 1 STAGE 1 OR 2 WITHOUT PLUS	OBSERVE 1 WEEKLY FOR PROGRESSION / REGRESSION
	ZONE 2 STAGE 3 WITHOUT PLUS	

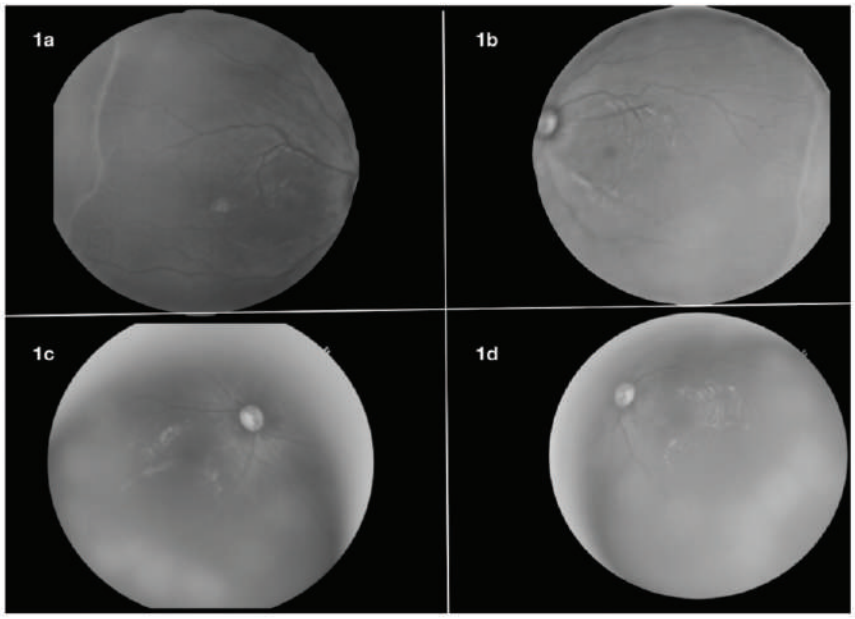

FIGURE 1 showing ROP zone 2 stage 3 at 36 weeks (1A & 1B). Retina at 48 weeks post natal showing spontaneous resolution (1C & 1D).

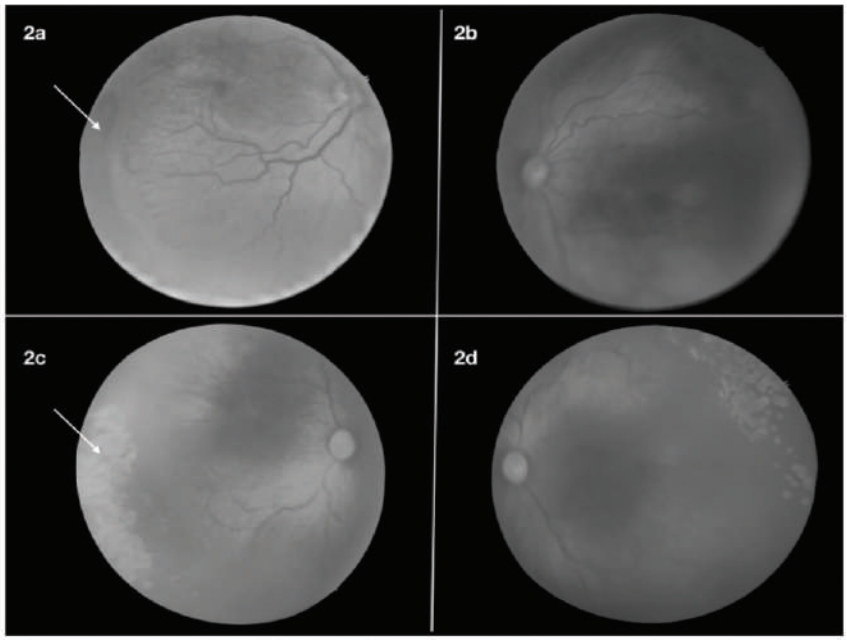

Fig 2 showing ROP zone 2 stage 3 with plus disease pre and post treatment in a premature child with gestational age 32 weeks and birth weight 1300 grams. Note the presence of ridge with elevated neo-vessels in zone 2 (2 A and 2 B). Laser scars and disappearance of ridge clearly visible post treatment in 2C and 2D

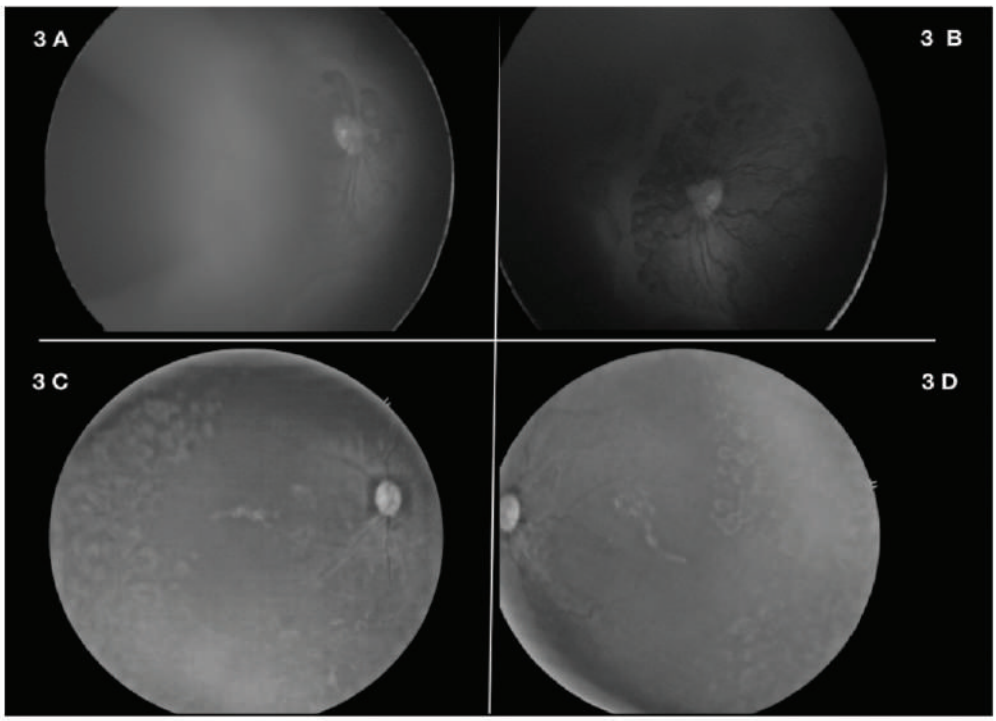

Fig. 3 APROP pre and post treatment in a premature child with gestational age 29 weeks and birth weight 1055 grams. Note the excessive tortuosity and dilatation of vessels in zone 1 (3A and 3B). Laser scars and disappearance of vessels tortuosity post treatment is clearly visible in 3C and 3D

CONCLUSION

Through this case report, we have demonstrated the effectiveness of appropriate management in treating ROP. With the current standard of treatment available in our armamentarium for ROP, including laser photocoagulation and Intravitreal anti VEGF injections, eyes with even aggressive posterior ROP could be salvaged, provided screening and intervention are applied within the appropriate time frame. Ablating the peripheral avascular retina in such cases decreases the VEGF load, preventing further progression and complication of the disease. Anti VEGF agents can be used as primary therapy in APROP or non dilating pupil or as an adjuvants to laser in other cases. Spread in awareness of the disease amongst paediatricians and ophthalmologists, and timely referral to an ROP specialist, is the keystone in the battle against ROP.

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Demographics Of A Large Cohort And Long Term follow-up of Idiopathic Macular Telangiectasia - Type 2

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BL.Sunitha, Kavitha S Rao, Sagarmoy Purkayastha

Abstract

Purpose: To describe the demographics and disease characteristics of a large cohort of idiopathic macular telangiectasia- type II.

Design : Retrospective cohort of 508 eyes for 13 years.

Methods : Included were patients of type II idiopathic macular telangiectasia between 1999 - 2012. Staging was done. Visual outcome was seen. Prevalence of CNVM was recorded. Associated diabetes mellitus and associated vascular diseases of the retina were also noted. Treatment modality was recorded over a maximum of 13 years.

Results: Mean age of the patient was 58 years. The overall presenting logMAR BCVA was < 1.00 in 45(8.85%) eyes, 1.00 – 0.6 in 194(38.18%) eyes, 0.48 – 0.30 in 156(30.70%) eyes and 0.18 – 0.00 in 113(22.24%) eyes. 256 (50.39%) were in stage IV.

44(8.66%) eyes were followed up for a period of > 4 years. In this group, the mean logMAR BCVA declined from 0.52 at presentation to 0.57 at the final visit. (p=0.29, > 0.05) by the last visit. 34(77.27%) eyes had no CNVM. 10(22.72%) had CNVM - 8(18.18%) at the time of diagnosis and 2(4.25%) on follow up. 6(13.63%) maintained vision after treatment, 2(4.54%) improved more than 1 line with treatment, 1(2.27%) dropped vision of 1 line, 1(2.27%) dropped vision >2 lines due to scarring.

The prevalence of CNVM in this series is found to be 8.66%. Diabetes was present in 135(26.57%) patients.

Conclusion: This is a large cohort over a long follow up of 13 years showing type II IMT presenting with mild dimness of vision. There is minimal change of visual acuity during the follow-up period. At least one eye maintains workable vision till the last. The main reason for dimness of vision is for development of choroidal neovuscular membrane.

Keywords: IMT (Idiopathic Macular Telangiectasia), Choroidal Neovascular Membrane (CNVM) , Parafoveal Telangiectasia (PFT), FA (Fluorescein Angiography), Mac Tel

Introduction:

Reese proposed the term 'retinal telangiectasia' to describe ectasia and irregular dilation of retinal capillaries which can occur as a developmental anomaly[1]. Manifest or occult telangiectasia which can occur near the macular region without a known cause were originally studied and classified by Gass and Oyakawa[2] in 1982, described as Idiopathic Juxtafoveolar Telangiectasis. Gass and Blodi [3] revised the classification further in 140 patients. In 2006, Yannuzi et al[4], have coined a new term of Idiopathic Macular Telangiectasia (IMT) to replace the older term. Recently, D. Shukla et al [5] also reported long term visual prognosis and incidence of CNVM in 203 eyes of 104 patients over a period of 8 years from southern India. While type I IMT has a more or less definite natural history and consensus on treatment, the most reported subtype type II IMT remains a poorly understood disease [4,6,7]. Though widely recognised to be more common, the scope of therapeutic intervention remains obscure. This is mainly because of less number of cases in study for a less period of time of follow-up and the exact pathogenesis of the disease has not been elucidated so far. It is now known to be not a primary retinovascular disorder but a primary

neurodegenerative disease with a secondary vascular involvement and that the Müller cells is the main suspect.

We retrospectively analysed the patients of Type II IMT in a referral institute, diagnosed by an experienced retinologist and the patients were on a long term follow up of even up to 13 years, to see the demographics, visual prognosis, associated retinal changes and prevalence of CNVM and the treatment modalities over a period of time.

Materials and Methods:

Clinical records, FFA and OCT features of Retina Institute of Karnataka, Bangalore were reviewed to find IMT TypeII between January1,1999, and December31, 2012.

The inclusion criteria were the unilateral or bilateral presence of abnormal juxtafoveal vessels that leaked fluorescein into the surrounding and were associated with central vision loss or distortion. Any radiation exposure and drug therapy which are maculotoxic was ruled out.

At the first visit, each patient underwent complete ophthalmologic examination, including dilated fundus examination. All had color photographs and FA (HRA 2 Heidelberg Engineering) and OCT(spectral OCT SLO combination imaging system, Optos). Patient recruitment started in 1999 and OCT came in 2006. And as such there is a pre OCT era of IMT where patients were diagnosed based on fundus photographs and FA.

Review of the clinical and FA and OCT of all patients and determination of the type of IMT was done according to the Yannuzi4 classification by an experienced retinologist.

Vision was assessed by using the ETDRS logMAR chart on the first and follow-up visits. Comparative visual change between visits was reported as lines of loss. For example, a decline from logMAR 0.00 to 0.30 was reported as 3-line loss; from logMAR 0.00 to 1.00 was 10-line loss. Vision below logMAR 1.00 was considered one additional line of lost vision.

Type II IMT (Perifoveal telangiectasia or Mac Tel) is the most common type of telangiectasia encountered. This type has subtle biomicroscopic changes best delineated on FA. Typical fundus features are bilateral symmetrical areas of retinal thickening usually one disc diameter or less involving the temporal half of the fovea. These changes may involve the nasal para fovea3. This form is associated with no serous detachment and no lipid exudation. Instead, tiny yellowish crystalline deposits Fig.1 (i) in the area of the thickening and characteristic pigment spots are the hallmarks of this entity.

Biomicroscopic staging was done.[8]

Stage 1: It is usually found in the asymptomatic fellow eye, diagnosis mainly by FA in which late phases show mild staining in outer retina of the temporal perifovea. Fig.1(ii)

Stage 2: Slight greying and loss of transparency of the parafovea Fig. 1(iii). FA shows mild capillary telangiectasia affecting primarily outer capillary network.

Stage 3: Blunted and dilated retinal venules extending at right angles into the depth of the perifovea on the temporal side Fig.1(iv) are present with mild vision loss. FA shows well defined telangiectatic network and well delineated right angled venule.

Stage 4: Stellate black hyperplastic RPE plaques along the right angle venules and OCT shows corresponding RPE elevation Fig.2(i) . FA shows blocked fluorescence corresponding to the pigments.

Stage 5: CNVM, hallmark of which is subretinal hemorrhage. This stage of IMT requires treatment.

OCT and Ultra High Resolution OCT (UHR-OCT) have given very useful details which can help us understand the pathology better.[9,10] It can reveal pathologic alterations indicating degeneration or atrophy of neurosensory retina.[11,12]

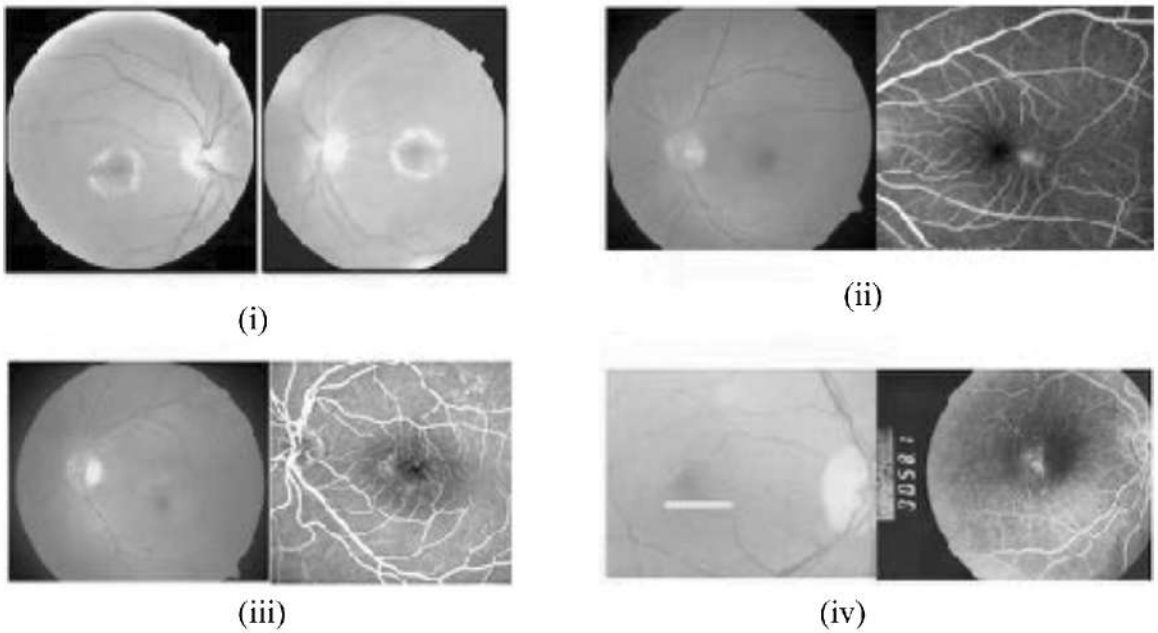


Fig. 1.. Biomicroscopic and FA changes in Type II Mac Tel (i) Parafoveal yellowish crystalline deposits(ii)Asymptomatic fellow eye with late phase FA perifoveal stain.(iii) Greying of parafoveal retina with capillary telangiectasia (iv) Right angled venules showing telangiectatic network with right angled venules.

OCT findings were as follows: [9,10,13]

1 Foveal cyst: these may be small and located in the inner layer of the foveola temporally Fig.2(ii) or may be prominent involving the entire foveolar thickness. These are usually not associated with foveal thickening.

Disruption of the inner segment/ outer segment photoreceptor junction. Fig. 2(iii)

Hyperreflective dots or elevation corresponding to the pigment hyperplasia seen clinically.

Foveal detachment Fig. 2(iv) and thinning Fig. 2(iv).

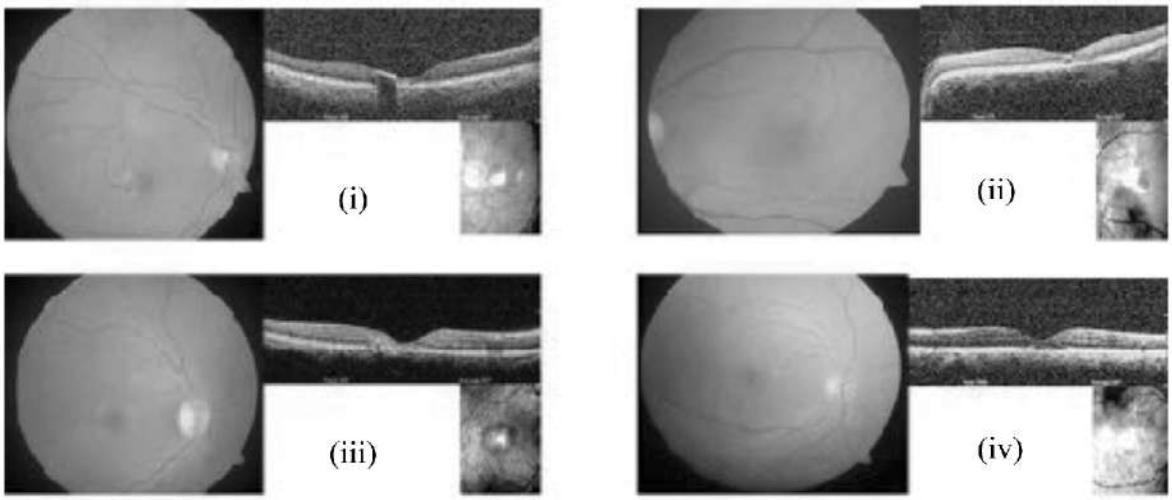


Fig. 2. Biomicroscopic and corresponding OCT changes in Type II Mac Tel (i) Stellate black RPE plaques with corresponding RPE elevation (ii) Slight greying with corresponding foveal cyst (iii) Slight greying with corresponding IS/OS disruption (iv) Slight greying with corresponding foveal detachment with foveal thinning

Associations with other systemic diseases were found out.
Statistical analysis was done using STATISTICA software using t-test for dependant variables. $P < 0.05$ was considered statistically significant.

Results

Total number of patients included was 254. Included were patients who had type 2 bilateral IMT. So, total numbers of 508 eyes were included. Duration of the study was 13 years (1999-2012).

Demographic Profile

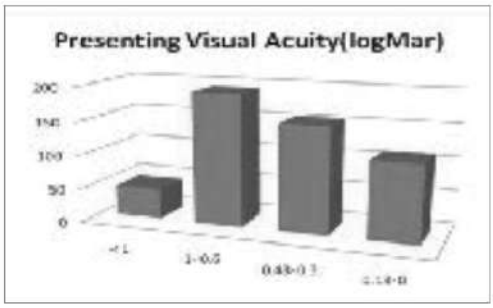
Males were 87 and females 167. Mean age was 58yrs (40-77yrs). Number of patients in the age group < 40yrs were 8(1.57%), 41-50yrs 57(11.22%), 51-60yrs 110(21.65%) and > 60 yrs were 79(15.55%) as is shown in the bar diagram Fig.3 (i).

Visual Prognosis

The overall presenting mean logMAR BCVA was < 1.00 LogMAR in 45 eyes(8.85%), 1.00 – 0.6 logMAR in 194 (38.18%), 0.48 – 0.30 logMAR in 156 (30.70%) and 0.18 – 0.00 logMAR in 113 eyes(22.24%) Fig.3 (ii).

There were 44 eyes (8.66%) followed up for a period of more than 4yrs. In this group, the mean logMAR BCVA decline from 0.52 at presentation to 0.57 at the final visit. ($p=0.29$, > 0.05). The change of visual acuity was recorded over a mean period of 6.14yrs.

In this group, 34 eyes had no CNVM. 10 (22.72%) had CNVM [8(80%) at the time of diagnosis and 2(20%) while on follow-up. Of these 6 (60%) maintained vision after treatment, 2(20%) improved more than 1 line with treatment, 1(10%) dropped vision of 1 line, 1(10%) dropped vision >2 lines due to scarring.19 patients(38 eyes 86.36%) had more than 5yrs follow-up.



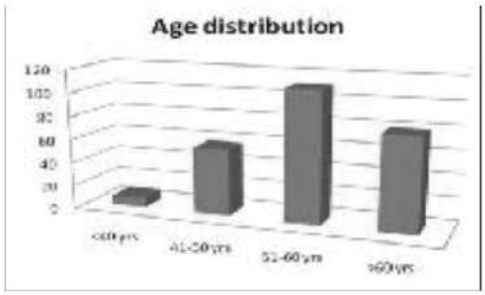


Fig.3 (i) Number of patients with <40 yrs,41-50yrs,51-60yrs and >60yrs (ii)Number of patients with presenting VA logMAR <1, 1-0.6, 0.48-0.3 and 0.18-0.

Case No.96 in stage IV IMT has the longest follow up of 13 years. Vision being maintained same that was present at the time of diagnosis in 1999 at 0.78 logMAR in right eye and 0.18 logMAR in left eye through 2012.

Clinical features and associated retinal changes

The number of eyes in stage I were 20 (3.93%),stage II 25(4.92%), stage III 163(32.08%), stage IV 256 (50.39%) and stage V 44 (8.66%).

OCT findings observed were: foveal cysts in 168 (33.07%), disruption of inner segment/ outer segment photoreceptor junction 102 (20.07%), hyperreflective dots 54 (10.62%), foveal detachments 5 (0.98%),foveal thinning in 20 (3.93%). The study started in 1999. So, OCT was done in the cases after its availability. **349 (68.70%) eyes underwent OCT.**

44 eyes (8.66%) had CNVM of which 38 (86.36%) had CNVM on presentation and 6 (13.63%) developed CNVM while on follow up. 5 (11.36%) had bilateral CNVM. Associated Diabetes mellitus : 135 patients (53.14%). Diabetic retinopathy was seen in 54 patients (21.25%) of which 10 (18.51%) had PDR. Other associated fundus findings 1(0.19%) each in BRVO, CRVO, AION, LMH and bilateral ERM in one patient (0.39%). Patients who had no CNVM were 464 (91.33%) eyes out of which 341eyes (73.49%) maintained vision throughout .

6 (1.29%) dropped 1 line vision all due to increasing pigmentation involving fovea. 3 (0.64%) dropped >2 lines - 2 had CSME and 1(0.21%) had foveal atrophy.

The prevalence of CNVM in this series is found to be 8.66%. Fig. 4 shows CNVM in IMT with corresponding FFA.

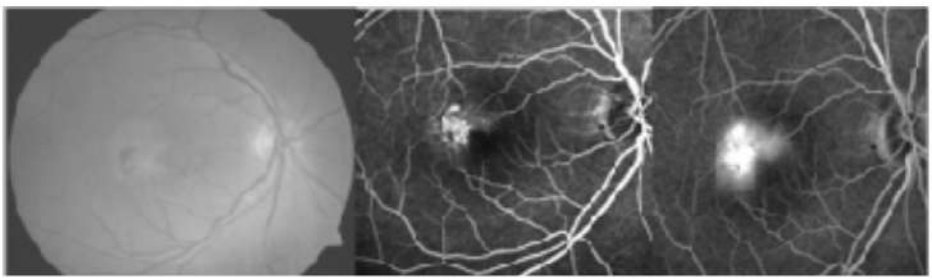


Fig.4. (i) Fundus photograph of IMT with CNVM
(ii) Corresponding early & late phase FFA of a case of IMT with CNVM

Table.I shows that transpupillary thermotherapy(TTT) was done in 09 eyes. Mostly in the initial period of the study (1999 upto 2006). Photodynamic therapy (PDT) or TTT was adviced in 6 eyes but all cases were lost to follow-up after the advise. This was also during the initial period of the study. Case no.7 in right eye had CNVM in 2006 which was treated with (PDT) with one injection of intravitreal triamcinolone acetonide(IVTA) and one injection of intravitreal bevacizumab. After which the eye showed scarring. The left eye of the same patient who had CNVM in 2009 during follow-up was given 7 injections of bevacizumab. PDT with bevacizumab was given in one more case. PDT alone was received by one case. Focal laser was given to two cases. 3 cases had scarred CNVM, so did not receive any treatment.5 cases were lost to follow-up before the advice for treatment.14 cases were given intravitreal bevacizumab injections.4 cases received multiple injections of bevacizumab and one case received ranibizumab injection. After 2006 most cases received injection bevacizumab.

Sl.No.	Case No.	Treatment Given	
1	Case No. 2	TTT	
2	Case No. 6	PDT/TTT	LOST TO FOLLOW UP
3	Case No. 4. RE	PDT/ INJ AVASTIN	
4	LE	INJ AVASTIN	
5	Case No. 25	PDT/TTT	LOST TO FOLLOW UP
6	Case No. 26	TTT	
7	Case No. 34	PDT/TTT	LOST TO FOLLOW UP
8	Case No. 17	PDT/TTT	LOST TO FOLLOW UP
9	Case No. 57	PDT/TTT	LOST TO FOLLOW UP
10	Case No. 33	PDT/TTT	LOST TO FOLLOW UP
11	Case No. 47	FOCAL LASER	
12	Case No. 68	TTT	
13	Case No. 75	TTT	
14	Case No. 108	TTT	
15	Case No. 112	TTT	
16	Case No. 113	LOST TO FOLLOW UP	
17	Case No.121	NO TREATMENT	
18	Case No.123	LOST TO FOLLOW UP	
19	Case No. 124	TTT	
20	Case No. 125	TTT	
21	Case No.126	TTT	
22	Case No. 127	FOCAL LASER	
23	Case No. 144	PDT	
24	Case No. 163	PDT+ INJ AVASTIN	
25	Case No. 164	INJ AVASTIN	
26	Case No.166 RE	RE INJ AVASTIN	
27	LE	LE INJ AVASTIN	
28	Case No. 171	LOST TO FOLLOW UP	
29	Case No. 180	INJ LUCENTIS	
30	Case No. 181	LOST TO FOLLOW UP	
31	Case No. 183	NO TREATMENT- SCARRED	
32	Case No. 197 RE	INJ AVASTIN	
33	LE	INJ AVASTIN	
34	Case No. 199	INJ AVASTIN	
35	Case No. 214	NO TREATMENT- SCARRED	
36	Case No. 216	INJ AVASTIN	
37	Case No. 217	INJ AVASTIN	
38	Case No. 218	LOST TO FOLLOW UP	

39	Case No. 219	INJ AVASTIN	
40	Case No. 225	INJ AVASTIN	
41	Case No. 244	INJ AVASTIN	
42	Case No. 250	INJ AVASTIN	
43	Case No. 251	INJ AVASTIN	
44	Case No. 253	INJ AVASTIN	

TABLE 1. Modalities of treatment received by the CNVM cases.

Discussion

IMT is less commonly discussed entity in this part of the globe. There are no reports of the prevalence of CNVM and the treatment modalities over a period of time. T Clemons et al [14] in 2010 had studied the baseline characteristics of a large international cohort of patients with 310 participants of MacTel Type 2. Our personal interest and experience over 13 long years could gather some of the information in term of the demographics, visual prognosis and associated retinal changes along with finding out the prevalence of CNVM in IMT and the change of treatment modalities over a decade in this reported cohort in southern India, comprising of 508 eyes.

IMT occurs mainly in the 5th and 6th decade and affects both sexes equally. The mean age of onset is 55yrs3. 96.85 % of our patients were above 40yrs of age and females were affected more. In the series by Yanuzzi, the mean age was 59yrs 4. Mean age was 58yrs (40-77yrs) in our series.

Bilateral involvement is the rule in this group, although the clinical features may be more obvious in one eye. However FA is able to demonstrate changes not visible clinically in the fellow eye. Good vision is usually maintained in stages I and II and patients may be symptomatic in stages III and IV 5. In this study, the number of eyes in stage I were 20, stage II 25, stage III 163, stage IV 256 and stage V 44.

There were 44 eyes followed up for a period of more than 4yrs. In this group, the mean logMAR BCVA decline from 0.52 at presentation to 0.57 at the final visit. ($p=0.29$, > 0.05) by the last visit. The change of visual acuity was recorded over a mean period of 6.14 yrs. In this group, 34 eyes had no CNVM. 10 had CNVM (8 at the time of diagnosis and 2 while on follow up). of these 6 maintained vision after treatment, 2 improved more than 1 line with treatment, 1 dropped vision of 1 line,1 dropped vision >2 lines due to scarring. Overall, 44 eyes had CNVM of which 38 eyes had CNVM on presentation and 6 eyes developed CNVM while on follow up. 5 eyes had bilateral CNVM.

Central vision loss occurs gradually and is associated with foveal atrophy that develops in the absence of cystoid macular edema. Patients who develop CNVM may have rapid loss of vision, sub-retinal hemorrhage,exudates, disciform scarring and retino-choroidal anastamosis.

UHR-OCT[9] had shown a unique feature called 'ILM drape'. This represents loss of outer plexiform layer due to central cystoid spaces. This is seen as thin ILM layer that crosses above the cystoid space in the focal area of the retina. UHR OCT could pickup large intraretinal blood vessels, probably enlarged deep vessels or vessels due to neo vascularisation. The authors have concluded that it may be possible to diagnose macular

telangiectasia on OCT and UHR OCT without FA. Recently the morphologic characteristics of IMT have been studied using spectral domain (SD-OCT) and polarisation sensitive optical coherence tomography (PD-SD-OCT) by Schutze et al[15]. A classification based on the OCT findings was suggested. Group 1 included discrete alterations in the inner retinal layers, Group 2 showed irregularities of the junction between the inner and outer photoreceptor segments with outer retinal atrophy but an intact RPE, Group 3 revealed RPE irregularities and loss in addition to intraretinal alterations and photoreceptor abnormalities. On SD-OCT, hyperreflective spots were detected in all stages of the disease in the foveolar and parafoveolar region. This phenomenon may represent an early sign of a neurodegenerative process[16]. In this series, OCT findings observed were: foveal cysts in 168 eyes, disruption of inner segment/outer segment photoreceptor junction in 102, hyperreflective dots in 54, foveal detachments in 5, foveal thinning in 20. The study started in 1999, so, OCT was done in the cases after its availability.

IMT may be associated with diabetes mellitus[17], hypertension, coronary artery disease and renal failure in 15 % of the patients, but no significant correlation with any disease exists2. This study found associated diabetes mellitus in 135 pts (53.14%).

Diabetic retinopathy was seen in 54 patients of which 10 had PDR .

Other associated fundus findings were 1 eye each with BRVO ,CRVO, AION, lamellar macular hole and bilateral epiretinal membrane.

Most patients are either asymptomatic or present with gradual loss of vision in the non-proliferative stage. Rapid loss of vision may herald the onset of proliferative stage. Role of laser photocoagulation has been studied by Park et al[18]. Grid laser photocoagulation was done. Neither the treated nor the untreated eyes had visual improvement of two or more lines. After treatment, 50 % eyes had increased retinal vascular distortion, 30% had new draining retinal venules, 50 % had intraretinal fibrovascular tissue, and 40% had retinal and preretinal hemorrhages . It was concluded that laser photocoagulation did not improve or stabilize long term visual acuity. We have not recommended laser photocoagulation to any of our patients except those with CNVM. Lahitte et al [19] reported that photodynamic therapy did not improve the vision.

Martinez and Jose [20] reported the use of IVTA in group 2 patients in 2003. Long term effect IVTA in the non proliferative stage has been reported by Wu et al [21].They reported 53 % incidence of cataract and 37% incidence of raised IOP which could be managed medically. Intravitreal anti VEGF has also been tried for non- proliferative IMT and a report published in 2008 shows functional improvement and transient decrease in leakage and retinal thickness, but a rebound effect was observed after 3 to 4 months with increase in retinal thickness [22,23]. Roller et al [24] reported in 2011 that no improvement in retinal thickness or visual function occurred in the non-proliferative stage with anti-VEGF and only the proliferative stage showed involution of the neovascular membrane. In 2010, Matt G et al [25] reported after bevacizumab in non-proliferative type 2 IMT despite an overall moderate effect, individual patients experienced a marked functional and morphological long term benefit. Ranibizumab have also been tried for non-neov ascular IMT type 2 and the results of the phase II clinical trial was released [26]. They observed that despite significant anatomical response to treatment, functional improvement in visual acuity was not detected.

At present treatment is indicated only for the proliferative stage of the disease. Since most neovascular membranes in the proliferative stage are subfoveal, laser treatment is not an option in these patients [27].

Photodynamic therapy (PDT) has been reported as a treatment modality. The first report was by Bernadette Snyers et al [28] in 2003. However, Shanmugam et al have described RPE atrophy following PDT [29]. Potter and Szabo et al [30] in 2001 reported that though PDT causes resolution of leakage from the membrane, there was no effect on the edema associated with the telangiectasia. With the introduction of antiVEGF, the role of PDT has changed, but will maintain an important role in combination therapy due to its unique properties of selective vascular targeting.[31]

Bevacizumab did not improve acuity or reduce retinal thickness in non-proliferative macular telangiectasia Type 2 at final follow-up. In proliferative IMT Type 2, bevacizumab caused involution of neovascularization and improved visual acuity [32]. In another study [33] non- proliferative IMT, bevacizumab decreases FA leakage in telangiectasia but has no short-term effect on visual acuity or OCT appearance. In proliferative IMT , bevacizumab arrests the leakage and growth of SRNV with visual acuity improvement. In another study [34], individual patients experience a marked functional and morphological long-term benefit. In a 56 year old woman, combination therapy with PDT and ranibizumab appears to be efficacious in the treatment of SRNVM associated with proliferative type 2 IMT [35]. In a clinical trial enrolling 5 participants [36,] despite significant anatomical responses to treatment, functional improvement in visual acuity was not detected. In this series, after 2006 most cases received bevacizumab.

The present study is potentially valuable since it confirms a number of previous observations, including the high prevalence of diabetes, the slow rate of visual loss and the relatively low risk of CNVM that are seen in IMT. The development of PDR in people with IMT is surprising. The series was amassed over more than a decade.

Limitations: Today there are many more imaging modalities such as fundus autofluorescence and microperimetry done in IMT.[37] Patients with Mac Tel 2 are being enrolled in a trial with Neurotech's implant of CNTF. This sudy was done, however, to find the demographic profiles of IMT in India with the resources available at that point of time.

Conclusion

This is a largest cohort from India being reported in type II idiopathic macular telangiectesia over a long follow up of 13 years showing that IMT with good vision to start with, maintains vision. Mostly were of stage IV, for patients had presented in advance stage in at least one eye. Only 8.66% develops CNVM needing treatment. CNVM may cause severe to moderate visual loss in idiopathic macular telangiectesia. At least one eye maintains workable vision.

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Importance of HIV, HBsAg and HCV testing before ocular surgery

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Blood-borne viral infections (BBVI) like Human immunodeficiency virus (HIV), Hepatitis B virus (HBV) and Hepatitis C virus (HCV) pose significant risk to the health care workers (HCWs) through needle-stick/sharps injury (NS/SI)and to other patients through cross-transmission. *Universal screening* or serological tests before surgery is not routinely practiced by ophthalmologists across India. It is reported that 5.9% of patients undergoing cataract surgery are seropositive (HIV: 0.09%; HBV: 1.8%; HCV: 4%).[1] A recent survey found that 18% of ophthalmologists do not do universal screeningand 20% order tests only in patients that they feel may be high-risk.[2]More alarmingly nearly one-fifth of the surveyed ophthalmologists were not aware about *universal precautions*, while 42% of the ophthalmologists did not know what precautions to be take when operating a seropositive patient. This ambivalent scenario does not auger well as prevalence of HBV, HCV and HIV is significantly high in India, with highest prevalence in the southern and north-eastern region.[3]

In our Institute we perform universal screening before all surgeries. The data from January 2015 to September 2019 is given in Table 1. HIV was positive in 0.3%, HBV in 1.0% and HCV in 0.6%. HBV infection was more common in our setting, than the previous study.[1]No seropositive patients are refused surgery in our Institute. All seropositive patients are referred for confirmatory tests and management.

Is performing universal screening foolproof? It is not. If tested within the window period before antibodies are detectable in the serum, an infected individual will test negative. Each test type has variable sensitivity and specificity. Testing also adds to the cost of eye care. As per the National Aids Control Organization, India, guidelines and National Viral Hepatitis Control Program 2018, India, no tests should be conducted without informed consent and pre- and post-test counselling of patients. Further because of lack of conclusive evidence proving utility of universal screening it is not recommended by National Program for Control of Blindness in India, or by other authoritative bodies in other countries like the American Academy of Ophthalmology or the National Institute of Clinical Excellence, UK. Yet, universal screening has been described as a risk-reduction strategy for HCWs, allows for immediate post-exposure prophylaxis in the event of NS/SI and also as a tool for detecting hitherto unknown cases of BBVI.[4]

Equally important is universal precaution,[5]which can be practiced along with routine infection control practices in both resource rich and resource poor settings (Table 2), to reduce risk of transmission of BBVI. Standard precautions must be used in all, while extra personal protective equipment should be utilized in those seropositive or high risk for BBVI.[4]

The dilemma of universal screening exists chiefly because it is resource dependent and its utility is not firmly established. One may follow the approachsuggested by Ahmed and Bhattacharya which is based on available resources. This approach is [i] rich-resource setting: routine HIV, HBV and HCV for all; [ii] limited-resource setting: test in high-risk cases based on history and clinical examination; [iii] poor-resource settings: test only in exceptional circumstances.[4]Although we work in a limited-resource setting, it is still possible to perform universal screening at our Institute for all patients as the test kits are quite cost effective, do not require expensive equipment, and are easy to perform. The total cost price of all the 3 rapid test kits in India is less than Rs.125.

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While one may debate on universal screening, universal precautions need to be routinely applied in our day to day practice. Referring seropositive patients to another center, a practice done by some,[2] is unethical. Ophthalmologists need to educate themselves about universal screening and universal precautions as '*no man is an island*'.

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Table 1. Prevalence of seropositive patients undergoing ocular surgery to BBVI

Year	HIV		HBV		HCV	
	No of patients tested	No of positive patients	No of patients tested	No of positive patients	No of patients tested	No of positive patients
2015	4345	15 (0.3)	4303	61(1.4)	4304	41(1.0)
2016	5220	15(0.3)	5203	60(1.2)	5203	49(0.9)
2017	5710	11(0.2)	5700	41(0.7)	5705	28(0.5)
2018	7100	25(0.4)	7152	78(1.1)	7162	36(0.5)
2019*	5270	10 (0.2)	5256	37(0.7)	5265	19(0.4)
Total	27645	76(0.3)	27614	277(1.0)	27639	173(0.6)

* Data till September 2019. Numbers within parentheses indicate percentages.

Table 2: Universal precautions and prevention strategies in preventing BBVI transmission in eye operating room

Type of precaution	Principle	Processes and methods
Standard precautions	Mitigates transfer of infection from patient to patient, patient to HCW, or HCW to patient	• Hand washing • Personal protective equipment • Patient placement • Environmental practices • Handling and disposal of sharp instruments • Work practices • Specimen handling and transport • Care of equipment

Assessing BVI status of all HCW. Pre-exposure vaccination	Facilitates vaccination of HCWs against HBV Reduces risk of HCW from developing active infection	Hepatitis B vaccination to all HCWs & staff who come in contact with sharps and body fluids – employer must make these available
Protective barriers	Personal protective equipment (PPE) reduces risk of exposure of HCW's skin and mucus membranes to potentially infective materials	• Gloves : single gloves, double gloves, special puncture resistant gloves • Masks • Caps • Sturdy covered footwear • Protective eye wear • Face shields • Plastic aprons, waterproof gowns
Work practice & engineering controls	Modifications in work practices mitigate parenteral exposures	• No re-capping, bending or breaking used needles syringes before disposal. If recapping is required then single-handed scoop technique be followed • Use of puncture-resistant needle and sharp object disposal containers. This should be as close as possible to the work area • Avoid hand to hand passage of needles & sharps • Use of ports and other needleless vascular access • Needleless or protected needle infusion system • Avoid unnecessary phlebotomies/ intravenous catheterization • Avoid re-using sharps • Hand washing • General purpose utility gloves for housekeeping chores • Do not do operation room duty in case of cuts or breaks in the skin • Biomedical waste disposal as per Biomedical Waste Management Rules 2016 • Continuous safety training programs
Cleaning spills of blood and body substances	Reduces exposure to HCW and patients	Use PPE prior to cleaning spills (prepare a Hazardous material – Hazmat bag) Use freshly prepared 0.1% sodium hypochlorite (household bleach)
Surgery in seropositive individuals	All bodily fluids of all patients should be regarded as potentially hazardous substances	• Scheduling seropositive individuals as the last case in operation schedule • Double gloving/ puncture resistant gloves

		then immediately refer to physician for PEP as per National AIDS Control Organization guidelines. These drugs should be initiated as early as possible within hours(2 – 72 hours) after exposure. • For HBV, PEP by physician. No PEP for HCV

Sources of information:

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2. National AIDS Control Organization, India [http://naco.gov.in/about-us/policies-guidelines];
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ROLE OF SECONDARY IMPLANT IN COSMETIC ENHANCEMENT OF SURGICAL ANOPHTHALMOS

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Anophthalmic socket is a major factor of facial disfigurement and psychosocial setback to the individual as well as their family. In the aesthetic world, facial appearance is given much importance, eyes contributing most to it. . Feeling of shame, insecurity, fear, inferiority and anger are very much common among one-eyed patients. They find it difficult to take part in social activities, to continue their education and to work.

Surgical Anophthalmos, following Evisceration and Enucleation; in the field of Ophthalmic surgery is not new to this era having evidences of it since the 18th century. In present day, Patients undergoing an evisceration or enucleation generally receive an orbital implant at the same time for volume replacement. This is referred to as a primary implant .It facilitates optimal cosmesis, decreasing disfigurement, reduced deepened superior sulcus. It also decreases upper and lower eyelid malposition. But sometimes, there are still patients who do/did not receive primary implant at the time of removal of the eye. This might be due to lack of facilities in the form of manpower and infrastructure. In such kind of patients the prosthesis cannot be fitted properly later. So, a subsequent procedure is considered which is called Secondary orbital implant surgery. It is a more complicated surgical procedure as the planes are already disturbed post enucleation. Putting the implant in extraconal space is challenging. Post evisceration secondary implant is difficult as the sclera gets shrunken and it becomes tough to put a large implant. Different techniques and types of implant and wrapping materials are used nowadays.

If a patient doesn't receive a primary implant at the time of removal of the eye, the most challenging situation arises after couple of years of using Readymade Stock Eye in socket. The challenges are-

a) The socket might be very roomy without the volume replacement by the implant, hence the prosthesis to be fitted will turn out to be very large and heavy. This will put a lot of stress on the inferior fornix and lower eyelid and can result in lower lid laxity and ectropion in due course of time.

b) The prosthesis without having an orbital implant to properly sit on will definitely show lower motility.

c) The eye would appear shrunken, deep seated with very low in terms of cosmesis value.

d) Long term leaving the eye anophthalmic without implant will result in surface loss and volume loss within the socket and become contracted. This makes it tough to fit a prosthesis.

Children undergoing enucleation for Retinoblastoma must be tried to insert an implant at the time of surgery. Any implant, PMMA or Porous can be put. Porous implants are more expensive, chances of migration are less. But they have more chances of exposure and extrusion. In PMMA implants, chances of migration are more. Absence of an implant will adversely impact the proper growth of the soft and bony tissues leading to bony hypoplasia and contracture of the socket. In later years, Facial Asymmetry can be seen in such patients. Readymade stock eyes fail not only in providing proper cosmetic appearance but also is not able to maintain a socket in good shape.

Contraction of the socket can be classified, according to Gopal Krishna Classification:-

Grade 0: Socket is lined with healthy conjunctiva and has deep and well formed fornices.

Grade 1: Shallow lower fornix or shelving of the lower fornix.

Grade 2: Loss of the upper and lower fornices.

Grade 3: Loss of the upper, lower, medial and lateral fornices.

Grade 4: Loss of all fornices and reduction of the palpebral aperture in horizontal and vertical dimensions.

Grade 5: In some cases, there is recurrence of contraction of the socket after repeated trials of Reconstruction.

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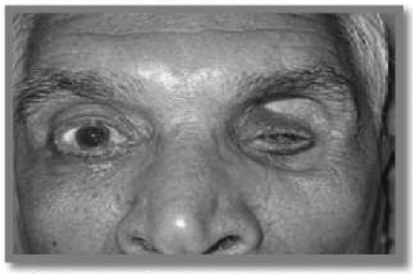
Another classification (WHO) is as follows-

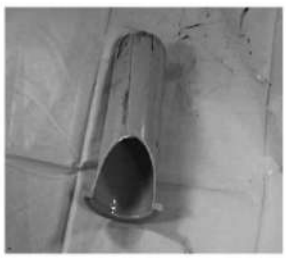
Mild grade: where only one fornix is involved and shortening of the posterior lamella of the lids.
Moderate: where both superior and inferior fornices are involved.
Severe: all fornices are involved along with phimosis of palpebral aperture.

Extensive loss of conjunctival surface area, deep cicatrix formation, atrophy of the orbital fat, fornix contraction and volume redistribution provides very poor cosmetic outcome with ocular prosthesis.

The best treatment is always prevention and this can be done by placing an implant in the surgical anophthalmic socket. But if the socket had already started contracture, only placing a secondary implant will not do, requiring additional steps of socket reconstruction. This includes deepening the inferior fornix by **fornix formation sutures**, **mucous membrane grafts**.

The introduction of the implant exerts a pressure posteriorly in the socket, pulling the orbital tissue surface and helps in creating a pocket like space (fornix) between the lid margin and the orbital tissue.

Orbital implant ***Implant Introducer*** ***Implant Placement*** ***Implant In-situ***

Different kinds of Orbital Implants are available nowadays for volume replacement. Mainly classified into two major groups- Non integrated orbital implants (primarily PMMA) and Integrated Implants (bone, plastic).The most important factor of successful outcome is surgical expertise, using whichever implant system the surgeon prefers. Rough implants, like hydroxyapatite is wrapped which is important to prevent erosion of the delicate tissues that are closed over the implant. Plain unwrapped silicone spheres have very low extrusion rates. Using a peg system to couple prostheses to ocular implants is costly and associated with a higher rate of complications. Evisceration may provide better motility and less superior sulcus deformity, but requires special care to ensure minimization of the risk of undiagnosed intraocular malignancy and prevention of sympathetic ophthalmia. Optimal outcomes require good working relationships with experienced ocularists.

As already mentioned, non porous implants has a higher rate of implant migration than porous implants. However, it is largely dependent on the surgical technique. Implants are wrapped in different kinds of materials, including autologous sclera, donor mesh, polyester fiber (Mersilene) mesh, and many others. The reasons for wrapping implants are generally to allow the surgeon to attach the muscles to nonporous implants. This also provides a layer of material between the implant and the conjunctiva which decreases the risk of exposure. One drawback of all wrapping materials is increased procedure time.

Volume Augmentation is done for better cosmetic result in the surgical anophthalmic socket which usually turned into contracted socket and making it difficult for even fitting of a prosthesis.

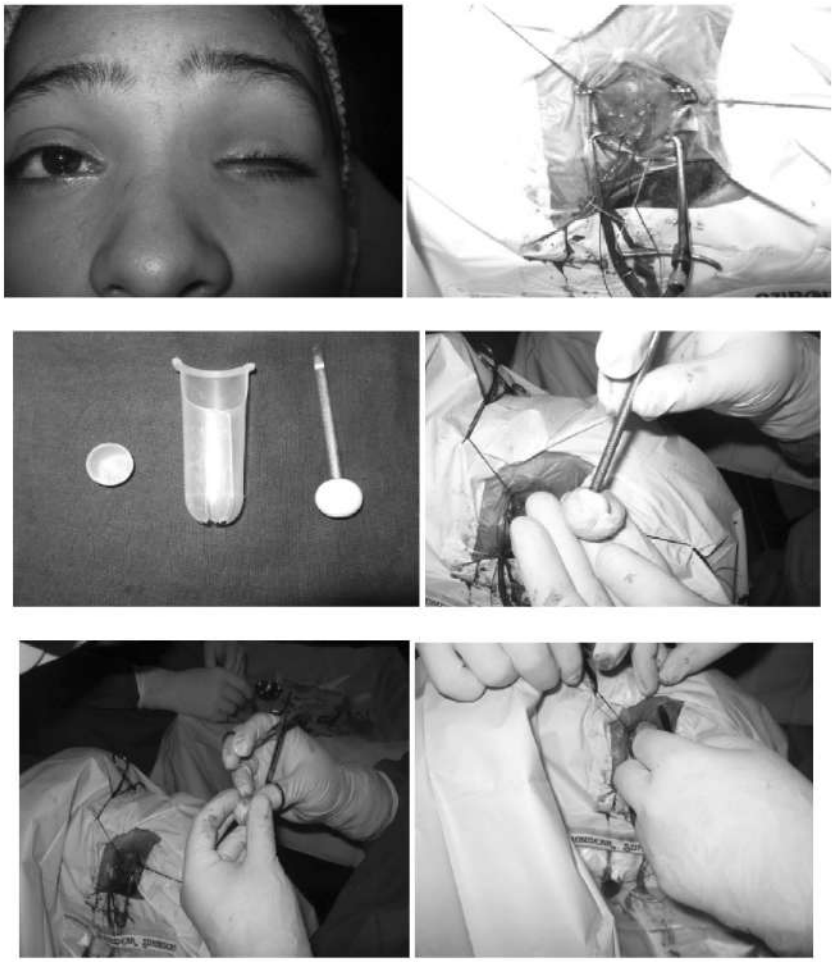
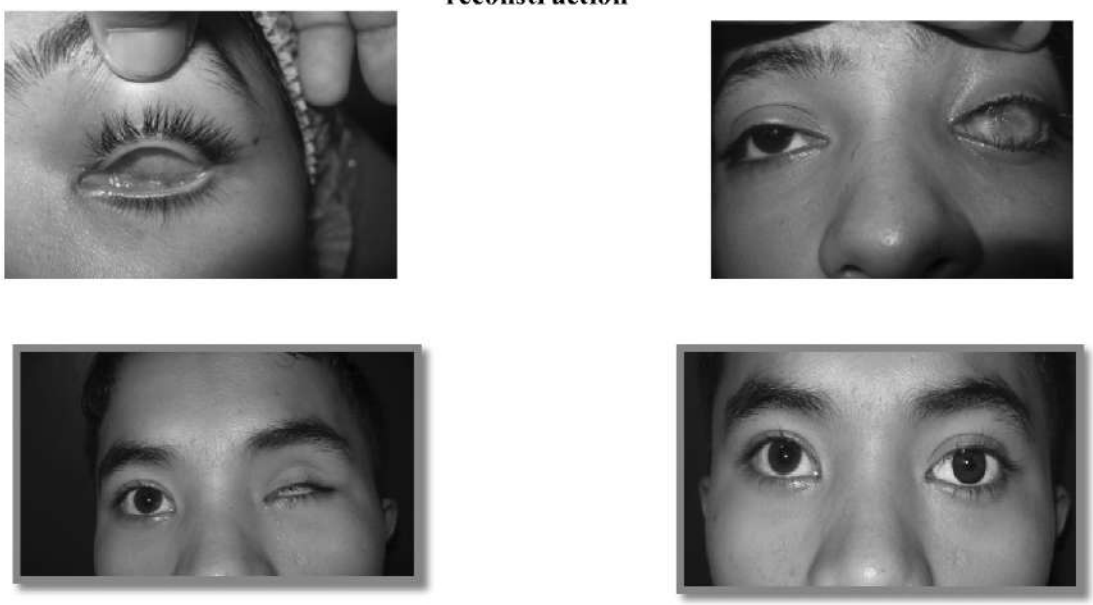


Figure: Volume Augmentation seen in a Contracted Socket by Secondary Implant and socket reconstruction



BEFORE **AFTER**

Recently, Injectable calcium hydroxyapatite (Radiesse), a volumizing filler, has been injected in the extraconal space for orbital volume augmentation in a series of anophthalmic patients. It is a costly procedure and moreover the potential risks include orbital inflammation and implant migration.
Taking into consideration all the factors involved and the consequences, volume augmentation by introducing a secondary implant in the surgical anophthalmic socket can immensely improve the quality of life of such patients. This makes them more acceptable in society, thus motivating them to continue their normal day to day activities.

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Postictal blindness

Hiranmoyee Das

Abstract

Aim - To study the etiology, clinical features and effectiveness of treatment in postictal blindness.

Methods- A 4-year retrospective review of patients presented with postictal blindness but normal ocular & optic nerve finding was conducted.

Results- 16 cases of postictal blindness studied (10 generalized seizures, 4 focal motor discharges while 2 children had the constellation of migraine headaches). 11 were children. The causes of seizure were idiopathic, febrile convulsion, ecclampsia & occipital lobe lesion like (trauma & tumour). The duration of postictal blindness was mostly 48- 72hours (6 hours and11 months). EEG revealed Occipital discharges were more common in the younger age group.

Conclusion- An EEG should be considered in any patient presenting with sudden onset of unexplained visual loss. Postictal blindness is associated with a favorable outcome when promptly diagnosed and treated appropriately.

Key words: Seizure, blindness, postictal

Introduction

Cortical or cerebral blindness (CB) refers to loss of vision produced by lesions affecting geniculocalcarine visual pathways [1]. Cortical blindness as a consequence of seizure is referred as postictal blindness. It is a rare presentation of epileptic seizures & associated with electroencephalographic (EEG) epileptic discharges. The clinical features of postictal blindness were summarized by Ashby and Stephenson in 1903 in 11 children and infants [2].

We report a series of 16 cases with documented epileptic blindness, describing the accompanying fits and thereafter the response to therapy to resolve the blindness and control associated seizures.

Methods & materials

A 4-years retrospective review of patients presented with postictal blindness was done in a 420 bedded multispecialty mission hospital, a tertiary health center, from Jan 2014 to December 2017. All patients had history of seizure & treated by Pediatricians or Neurologist. Ophthalmic consultation was sought, as upon regaining consciousness blindness was complained either by patients or by attendant. Blindness was confirmed by failure to respond to any visual stimuli. Optokinetic nystagmus was also could not elicited in these patients. Detailed ocular examination including dilated fundoscopic examination with 20D lens was carried out in every patient. Pupillary reactions and on fundoscopic examination were normal in these patients. Electroencephalogram (EEG) was done in all patients. CT scan & MRI were done whenever indicated. Patients were followed up subsequently. Permission to conduct this research was obtained from institutional review board. This study adhered to the tenets of the Declaration of Helsinki.

Results

Table1: Age & sex incidence

16 cases of postictal blindness studied. 11 were children. Cases were divided into 3 age groups.

Age group	Male	Female
< 5 years	4	3
5 -18 years	2	2
>18 years	3	2

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10 cases had generalized seizure and 4 had focal motor discharge. 2 children had the constellation of migraine headaches, focal motor seizures and complete blindness along with occipital EEG discharges

The causes of seizure were idiopathic, febrile convulsion, eclampia, occipital lobe lesion like trauma & tumour .There does not seem to be a clinical difference between patients with and those without occipital lesions. Some had encephalitis or cerebral malaria.

Table 2: Causes of seizure

Causes of seizure	No. of patients
Idiopathic	2
Febrile convulsion	4
Brain infection	3
Neurocysticercosis	2
Eclampsia	2
Surgical lesion like trauma or tumour	3

Blindness was bilateral and continued for days, weeks or months and only exceptionally for hours. In our study the minimum duration of blindness was 6 hours & the maximum was11 months. In most of the patients the blindness was lasted for 48 -72 hours. Sometimes blindness was accompanied by other neurological deficits such as weakness, deafness, aphasia or dementia.

Table 3: Duration of blindness

Duration of blindness	< 1days	1– 7 days	7 – 30 days	> 30 days
No. of patients	1	10	3	2

Ten patients with accompanying generalized seizures were treated with valproic acid 10-(15mg/kg/day). They regained full vision and eight became seizure free. 4 cases had accompanying focal motor seizures and two additional cases with isolated blindness and focal discharges were treated with carbamazepine (10-30mg/keg/day), regained full vision and all became asymptomatic within a period of 1-4 years.
Awake/ sleep EEG record taken using 30-20 international system of electrode placement under guidance of neurologist & neuro psychiatrist. EEG revealed Occipital discharges were more common in the younger age group.

Discussion

Amaurosis (Greek meaning darkening, dark, or obscure) is vision loss or weakness that occurs without an apparent lesion affecting the eye. According to Shahar & Barak postictal blindness has been primarily described in children following generalized and focal occipital seizures [3]. In the fewer reported adult cases, postictal blindness has been reported in association with focal occipital seizures, but more commonly generalized seizures with patients often in denial of their objective blindness (Anton's syndrome) [4]. Without any prior history of seizure-related visual disturbances complete postictal blindness can occur after focal extra-occipital seizures in adult patients with epilepsy. M.Sadeh et al stated 5 cases (all female) who had suffered Grand Mal Seizure. The ages were 33year, 70years, 25 years, 23years and of 20 years of age. All presented with grand mal seizures. In one case they found no pathology. Viral encephalitis, SSPE, astrocytoma, and emboli with left hemianopia were other diagnosis found [5].

Walsh and Hoy compared the blindness to postictal (Todd's) paralysis, the cause of which was thought to be exhaustion of neurons by hyperactivity [6]. The hypothesis that postictal paralysis is caused

by an active inhibitory mechanism was first suggested by Gowers [7], who objected to the "exhaustion theory" that had been proposed by Todd [8].

Kosnik et al described that a hyperpolarisation mechanism developing during the epileptic activity was responsible for ictal or postictal inhibition. In their cases, vision loss for several hours was associated with focal seizures and epileptic discharges involving the occipital lobe [9].

Brain anoxia could well account for the bilateralism of the blindness and its relatively long duration since the occipital lobes are especially sensitive to anoxia, being located in the cerebral blood supply border zone. In the last two decades with the wide use of cardio-pulmonary resuscitation, many severe hypoxaemic episodes and hypotensive events have occurred. Not uncommonly they have been complicated with cortical blindness [10]. Thus, anoxia has been recently considered to be one of the most frequent immediate causes of bilateral cortical blindness [11].

Our data for patients with epileptic blindness was similar to previous reports regarding the reported duration of blindness and associated seizures, as well as the overall response to therapy and outcome. Previous studies on postictal blindness were either in paediatrics or in adults. But both the groups were included in our study. It was seen occipital discharges were more common in the younger age group whereas in adults occipitoparietal discharges were mostly found. Younger age group was associated with better prognosis. Permanent postictal was described by Ashby & Stephenson in one of their cases [2] as well as by Sadeh et al in a case of status epilepticus lasting for several hours [4]. In our study complete resolution of blindness is found in all.

Conclusion

Analysis of the literature, in addition to our overall experience, indicates that postictal blindness is associated with a favorable outcome when promptly diagnosed and treated appropriately. Though complete resolution of post ictal blindness is found in all cases in our study, it does not indicate post ictal blindness is always reversible. An EEG should be considered in any patient presenting with sudden onset of unexplained visual loss, even in the absence of other epileptic phenomena.

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Dr Haimanti Choudhury, Dr H.K.Choudhury

Abstract

Headache is a common symptom of convergence insufficiency (CI).
Purpose: Effectiveness of vision therapy (VT) in patients with symptomatic CI.
Method: Subjects received 10 sessions of office based VT with home reinforcement. Primary outcome measure was symptom score. Secondary outcome measures were near point of convergence (NPC) & positive fusional vergence (PFV).
Results: 80 patients in age group 08-25years evaluated. Symptoms were significantly reduced, score being 34 to 10. Subjects demonstrated statistically and clinically significant changes in NPC (from 16.3 cm to 5.2 cm; P<.05) & PFV (from 11.8ΔD to 28.4ΔD; P<.05).
Conclusion: VT is effective in reducing symptoms such as headache.

Introduction

Headaches are one of the most common medical complaints; most people experience them at some point in their life. They can affect anyone regardless of age, race, and gender. The World Health Organization (WHO) reports that almost half of all adults worldwide will experience a headache in any given year[1]. A headache can be a sign of stress or emotional distress, or it can result from a medical disorder, such as migraine or high blood pressure, anxiety, or depression. The International Headache Society (IHS) categorize headaches as primary, when they are not caused by another condition, or secondary, when there is a further underlying cause.

Convergence insufficiency (CI) is a common binocular vision disorder in which the eyes have a strong tendency to drift outward (exophoria) when doing near work[2]. Patients present with eye strain, headaches, double vision, print moving on the page, inability to concentrate and short attention span. CI is characterized by exophoria greater at near than at distance, a receded near point of convergence (NPC), and reduced positive fusional vergence (PFV) at near. Headaches caused by convergence or accommodative insufficiency usually occur at school age, most often after third or fourth grade when the reading print becomes smaller and it takes a longer time to finish assignments[13,14].

Vision therapy (VT) is eye training to improve or enhance a patient's visual skills and performance. The systematic use of lenses, prisms, filters, occlusion and other appropriate materials, modalities, equipment and procedures is integral to vision therapy[3,4]. Non-surgical treatments used for treating convergence insufficiency include base-in prism reading glasses, home-based convergence exercises (pencil push-ups as), home-based vision therapy, and office-based vision therapy[2].

Purpose: Effectiveness of vision therapy (VT) in patients with symptomatic convergence insufficiency.

Methods: It is a prospective study design. Written informed consent was obtained from all participants, and for children assent was obtained from parents. A total of 80 participants were included in the study. The inclusion criteria followed for CI were adopted from CITT study[5]. Symptomatic subjects reporting to the clinic with minimum two of the three primary

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signs (Near exophoria≥4Δ than far, reduced near PFV <15 Δ D break and receded NPC>6cm) were included for the study. Any subject who reported near visual symptoms such as eyestrain, headache, blurred vision, diplopia or asthenopia were considered as symptomatic. Best corrected visual acuity of 6/6, N6 was also in the inclusion criteria. Exclusion criteria were amblyopia, nystagmus, constant strabismus or any previous ocular surgery.

Participants underwent a comprehensive ocular examination including fundus evaluation. The test for binocular vision included: visual acuity, ocular movements & cover test, near point of convergence, fusional vergence ranges and near point of accommodation. Selected participants were given a detailed description of the treatment and test procedures that they were expected to undergo. All the measurements were taken with the participants' best refractive correction. The CISS (Convergence insufficiency symptom survey) was administered to all participants before any other testing. Each response was scored as 0 to 4 points, with 4 representing the highest frequency of symptom occurrence (i.e., always). The 15 items were summed to obtain the total CISS score. The lowest possible score (least symptoms) was 0 and the highest was 60 (most symptomatic). A symptom score ≥ 16 has been found to differentiate children with symptomatic CI from those with normal binocular vision[6].

The near point of convergence and near point of accommodation was measured three times with RAF rule[7]. In the RAF rule, the dot on the line is the standard target. The cheek rest is placed on the inferior orbital margin. The RAF Near Point Rule is kept in the depressed position of 45 degrees. The subject is asked to focus on the black dot. The target is moved towards the patient slowly. When the patient reports diplopia, the movement is stopped. Recovery is noted when the patient reports one target when the slide is slowly moved back. All readings were measured to nearest 0.5 cm.

Positive fusional vergence was measured three times with a horizontal prism bar (prismatic levels from 1Δ to 45Δ) while the patient fixated a hand-held fixation target with a single column of letters of 20/30 equivalent at a distance of 40 cm.. The patient was asked to report when the letters became blurred or double as prism was introduced at approx 2Δ/sec, pausing at each prism to confirm that the target was single and clear. Primary outcome measure was symptom score (CISS). Secondary outcome measures were near point of convergence (NPC) & positive fusional vergence (PFV).

Treatment regime: All participants underwent office based vision therapy for 60 minutes per day for 10 sessions (5 days a week). In each session, there were various exercises to train the gross convergence, vergence amplitudes and facility, and accommodative amplitude and facility. The therapy procedures were administered as per the guidelines in CITT manual of procedures. The exercises involved Synoptophore, Brock string training with and without flippers and prism bar training. The patients performed the procedures under the supervision of therapist[5]. Home reinforcement was demonstrated by an optometrist on an individual basis. They were conversed with procedures to perform at home. Home exercise (Pencil push up procedures) was advised for 15 minutes per day for 5 days a week. The participants were evaluated at the end of 10 sessions. They were asked to follow up at 1 month, 3 months and 6 months. Those who were symptomatic at the course of follow up were advised repeat sessions of office based vision therapy.

Results: 80 patients in age group 08-25years evaluated. There were 44 female and 36 males. Mean age of the patient was 17.6±4.3 years. Symptoms were significantly reduced, score being 34 to 10. Subjects demonstrated statistically and clinically significant changes in NPC (from 16.3 cm to 5.2 cm; P<.05) & PFV (from 11.8ΔD to 28.4ΔD; P<.05). Symptom score

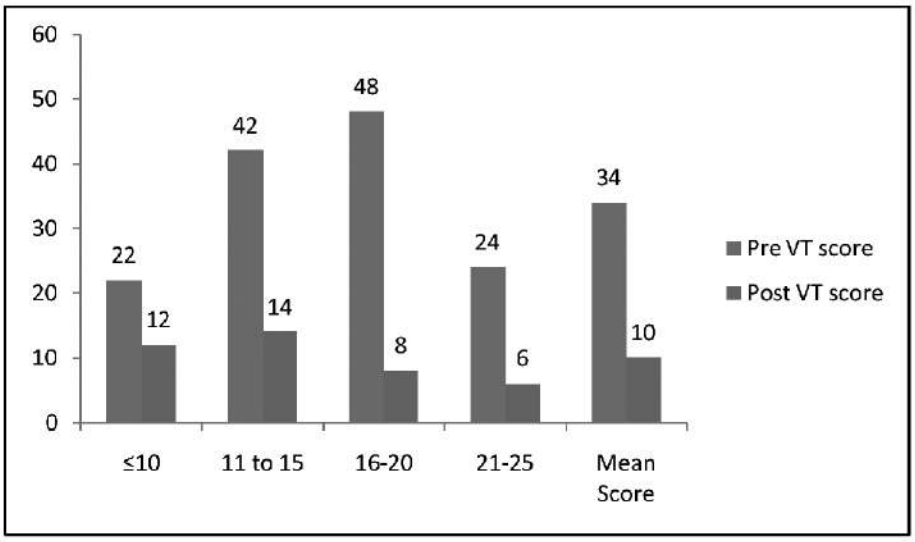
and objective measurements were performed prior to and after completion of vision therapy. Paired t test was used to get the "p value" in Microsoft Excel.

Table 1: Age distribution

Age group	No. of patients
≤10	10
11-15	19
16-20	28
21-25	23

Table 2: CISS Score

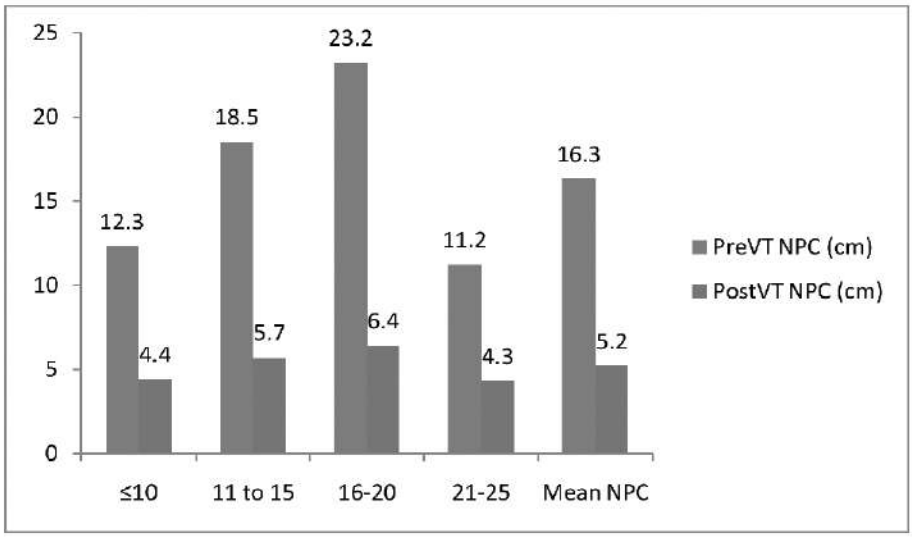
Age group	Pre VT score	Post VT score
≤10	22	12
11-15	42	14
16-20	48	8
21-25	24	6
Mean Score	**34**	**10**



Graph 2: CISS Score

Table 3: Pre and post VT Near point of convergence

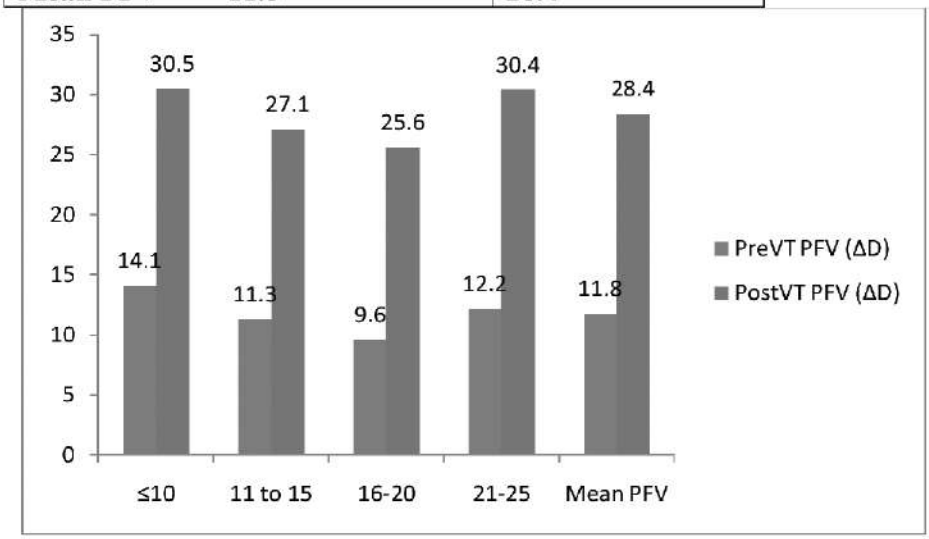
Age group	Pre VT NPC (cm)	Post VT NPC (cm)
≤10	12.3	4.4
11-15	18.5	5.7
16-20	23.2	6.4
21-25	11.2	4.3
Mean NPC	**16.3**	**5.2**



Graph 3: Pre and post VT NPC

Table 4: Pre and post VT Positive fusional vergence

Age group	Pre VT PFV (ΔD)	Post VT PFV (ΔD)
≤10	14.1	30.5
11-15	11.3	27.1
16-20	9.6	25.6
21-25	12.2	30.4
Mean PFV	**11.8**	**28.4**



Graph 4: Pre and post VT PFV

Discussion: The results of the CITT determine if any of the forms of active therapy are effective for symptomatic CI in children. Fifty-four children were randomly assigned to the home-based pencil push-up group, 53 to the home-based computer vergence/accommodative therapy and pencil push-ups, 60 to the office-based vergence/accommodative therapy with home reinforcement group, and 54 to the office-based placebo therapy group. Using analysis of variance to compare the groups, none of the differences in CISS, near point of

of variance to compare the groups, none of the differences in CISS, near point of convergence, or positive fusional vergence(near) were clinically or statistically significant[5,12].

Neeraj Kr Singh et al reported stimulus and response AC/A ratio increased following VT, accompanied by clinically significant changes in vergence and accommodation parameters in subjects with convergence insufficiency. Ten sessions of in-office VT, each one of 60min (total 600min), produced significant changes in the outcome measures of response and stimulus AC/A crosslink ratios, and accommodation and vergence parameters[8]. Scheiman M et al reported vision therapy/orthoptics was the only treatment that produced clinically significant improvements in the near point of convergence and positive fusional vergence. However, over half of the patients in this group (58%) were still symptomatic at the end of treatment, although their symptoms were significantly reduced. All three groups demonstrated statistically significant changes in symptoms with 42% in office-based vision therapy/orthoptics, 31% in office-based placebo vision therapy/orthoptics, and 20% in home-based pencil push ups[9,10,11].

Scheiman M et al reported a systematic review of 6 randomized controlled trial on the effectiveness of non-surgical interventions for convergence insufficiency with a total of 475 participants. They concluded that Office-based vision therapy/orthoptics is more effective than either home based pencil push-ups or home-based computer vergence/accommodative therapy in children and young adults[2].

Our study showed that office based vision therapy with home reinforcement in subjects with symptomatic convergence insufficiency had statistically and clinically significant changes in NPC and PFV, similar to studies done by NK Singh et al and Scheiman M et al. Limitations of our study are short follow up and possibility of observer bias while taking the pre and post VT measurements. Computer software based office procedures was not used. Recommendations are study with a longer follow up period. Inclusion of one or more control group undergoing home therapy/placebo therapy/computer based software program will further enlighten on the effects of VT. VT is effective in reducing symptoms such as headache and improving signs of convergence insufficiency.

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Clinical profile and risk factors for Chronic Central Serous Chorioretinopathy at a tertiary eye care institute in North East India

Hemalata Deka, Saurabh Deshmukh, Manabjyoti Barman
Ronel Soibam, Awaneesh Upadhyay, Harsha Bhattacharjee

Abstract

Purpose: To form a profile and to investigate the risk factors resulting in chronic central serous chorioretinopathy (CSC) at a tertiary eye care institute in North East India

Materials and Methods: This was a retrospective chart review study done on all patients of chronic CSC who have undergone one sitting of focal laser photocoagulation from August 2017 to August 2019. All the patients with risk factors for CSC were documented. The risk factors documented were gender, age, presence of hypertension, depression, tobacco use, alcohol use, stress at work, sleep disturbance and steroid usage.

Results: In total, 136 patients were enrolled in the study: 28 (20.5%) with acute CSC and 108 (79.5%) with chronic CSC. The mean age of presentation was 43 years. 102 (75%) cases showed one eye involvement and 34 (25%) cases had the involvement of both eyes. The most common presenting symptom was the diminution of vision, followed by seeing wavy lines and also complaint of seeing scotoma.

Conclusions: Old age males (to be changed) with stress and sleep disorders are more likely to progress to chronic CSC and may benefit from early laser photocoagulation. Treatment for systemic diseases, sleep disorders and de addiction are strongly recommended. All chronic CSC patients require meticulous and timely follow-up to avert the vision loss.

Keywords: Central serous chorioretinopathy, fundus fluorescein angiography, indocyanine green angiography, optical coherence tomography

Introduction

Central serous chorioretinopathy (CSCR) is a commonly encountered disease of the macula and patients frequently present with well-circumscribed serous retinal detachment on ophthalmoscopic examination, with associated leakage points on fundus fluorescein angiography (FA).[1]CSCR was first described by Von Graefe. He coined the term 'central recurrent retinitis' in 1866 for recurrent serous macular detachment. Later Gass, in the year 1967, explained the etiopathogenesis and clinical features of CSCR and named it central serous choroidopathy.[2,3]CSCR can be acute or chronic. Acute CSCR is usually a self-limiting process resolves with minimal visual sequelae, whereas the chronic CSCR may develop RPE atrophy and neurosensory retinal changes that ultimately lead to permanent vision loss. Focal laser photocoagulation has proven to be effective in promoting the resolution of the subretinal fluid and in reducing recurrence.[2-4]Therefore, it has become a major treatment for CSCR, especially for the persistent and recurrent CSCR. [4]Multiple previous studies have detected several risk factors for the development of CSCR, such as male gender, type A personality, hypertension, depression, antipsychotic medications, allergic disease, stress, tobacco use and smoking, alcohol consumption. It has also been seen that middle-aged males with stress and sleep disorder patients are more likely to progress to chronic CSCR.[5-11] However, there is no such study on risk factors for chronic CSCR from North East India.

The purpose of this case-control study analyses the demographic profile and risk factors for progression to chronic state in patients with CSCR, to form a profile and also evaluate the risk factors for chronic CSCR in a North-East Indian population and also assess the applicability of focal laser photocoagulation in these cases.

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Materials and Methods

This was a retrospective chart review conducted at a tertiary eye care center in North-East India. The written and informed consent was obtained from all patients. The cases with serous retinal detachments persisting even 6 months after the onset of symptoms were characterized as chronic CSCR. All the patients of chronic CSCR who had undergone one sitting of focal laser photocoagulation from the database were included in the study between August 2017 to August 2019.The patients with any other coexisting ocular pathologies, such as age-related macular degeneration, hypertensive retinopathy, diabetic retinopathy, uveitis, hereditary maculopathy or choroidal infiltrate were excluded from the study. All the patients were asked detailed questions regarding the past history of diseases like hypertension, stress addictions, medications, sleep disorders.

The clinical diagnosis of CSCR was made on the basis of symptoms, like diminution of visual acuity with or without metamorphopsia or micropsia; the presence of serous retinal detachment on indirect ophthalmoscopy and assessment on the optical coherence tomography(Spectralis HRA + OCT;Heidelberg Engineering, Heidelberg, Germany)examinations. The choroidal thickness evaluated on the enhanced depth imaging (EDI) optical coherence tomography. The retinal vasculature and active leakages were detected on the FA (TRC-50IX; Topcon Corp., Tokyo, Japan); and/or choroidal vasculature dilatation was detected on the indocyanine green angiography (Spectralis HRA + OCT;Heidelberg Engineering, Heidelberg, Germany). The fundus photographs were captured with the tabletop fundus camera (TRC-NW8 Topcon Corp., Tokyo, Japan). The data were analyzed with SPSS for Windows version 21.0 (SPSS, Chicago, IL, USA).

Results

A total of 136 cases of chronic CSCR post laser therapy were included in the study. Of the 136 cases, 28 (20.5%) were the ones who presented to us as soon as there was an onset of symptoms (Acute CSCR) and then followed up regularly with the persistence of subretinal detachment for six months or more (Chronic CSCR). These cases later received laser photocoagulation for the leakages, whereas 108 (79.5%) were those who at first presentation had features of chronic CSCR. The mean duration between the first diagnosis of CSCR to first focal laser photocoagulation was 11.2±8.4 (range: 6-24) months.

Demographic profile

The gender-based study showed that 128 (94.2%) patients were males and 8 (5.8%) patients were females. The average age of presentation was 43±5 (range: 27-65) years, suggestive of occurrence in middle-aged males. On the basis of the eyes involved, 102 (75%) cases showed one eye involvement and 34 (25%) cases had the involvement of both eyes. In patients with one eye involvement, 49 (36.1%) had right and 53 (38.9%) had left eye involvement.

Risk factor profile

The risk factors profile of the patient is depicted in table 1.

Table 1

Risk factors	**Present in cases (n=136)**	**Percentage**
Male gender	128	94.2%
Hypertension	54	39.7%
Depression	23	16.9%
Tobacco use	96	70.5%
Alcohol use	82	60.3%
Stress at work	42	30.9%
Sleep disturbance	45	33%
Steroid usage	26	19.1%

Discussion

Our study showed that the male gender is predominately affected. The age of the patient also plays an important role in the occurrence of the disease. The use of steroids and sleep disturbance and stress are other independent factors contributing to the occurrence of CSCR. We found that the male to female ratio was16:1. In previous studies, it was found to range from 2.6:1 to 7:1.[7,12] This difference could be because of more chronic cases in our study. In a study by Chatziralliet al, they reported that male sex is an important risk factor for the CSCR.[7] Similarly, our study also confirmed that male sex is an independent and important risk factor for chronic CSCR. In a study by Hanumunthadu et al, they observed that women with CSCR have better visual outcomes as compared to men.[13] Various studies have suggested the role of testosterone in the development of CSCR as it causes vasodilatation. [14]It is hypothesized that the testosterone causes choroidal due to vasodilatation and leads to an increase in the hyperpermeability of the choroid, resulting in neurosensory retinal detachment.[15] In a study by Nudleman et al, they reported nine CSCR cases following the initiation of the exogenous testosterone treatment. [16] It has also been reported in the literature that the anti-androgen treatment can cause the resolution of CSCR. [17,18]

We also found that chronic CSCR is commonly seen in middle (45-65years) to old age individuals. This finding is consistent with other previous studies. [11,19] It has also been reported that in the old individuals there is an increase in the size of RPE cells changes in the shape of mitochondria which is suggestive of increased oxidative stress. [15,20] These findings signify that the repair capacity of RPE is reduced with age and lead to chronic CSCR. In our study we also found that stress and sleep disturbance were present in number of cases. The literature review shows that both these conditions increase the levels of stress hormones like cortisol and catecholamines. These hormones have been proven to be behind the pathophysiology of the CSCR. [21,22]

In our study, we found that a significant number of patients had an alcohol addiction. It has found in the study by Haimovici et al, that alcohol use is an independent risk factor for the CSCR because ethanol gets converted acetaldehyde in the liver which itself is a vasodilator. [5] It has also been reported in the literature that after the consumption of alcohol the choroid thickness increases. [23]However, the significance of alcohol in the causation of CSCR could be established.

Laser photocoagulation has proven to be an effective treatment for the CSCR. It acts by direct thermal sealing the RPE defects which lead to leakages. [24] In our study, the acute cases had persistent SRF even 6 months after the onset of symptoms, thus later belonging to chronic CSCR class. Hence, they were treated with focal laser photocoagulation. The literature review showed that a large number of studies have compared the CSCR eye with the eyes of the controls as well as the other normal eye of the patient to identify the risk factors for the CSCR development. This study is first of its kind from North East India to find the characteristics of the patients who would progress to develop chronic CSCR and require early treatment. From the results of our study we suggest that the middle-aged male patients with stress and sleep disturbance be treated at the earliest with the laser photocoagulation as they are more likely to progress to chronic CSCR, whereas the young female patients with no stress and good sleep pattern be observed as they rarely tend to progress to chronic CSCR. The high-risk patients with the presence of the risk factors be treated in consultation with the physician for the systemic diseases.

The major limitation of our study is its retrospective nature. The limitations are the small sample size. Another limitation of our study is that the usage of ICG was less and the new imaging modality of OCT angiography has not been used. Thus, to conclude, further studies with multimodal imaging with larger sample size are the need of the hour.

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Foldable Intraocular Lens in Manual Small Incision Cataract Surgery

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Cataract is one of the leading causes of blindness globally and cataract surgery is one of the most common procedures done worldwide.[1] Although phacoemulsification is considered superior than manual small incision cataract surgery (MSICS) in terms of early post-operative recovery and better uncorrected visual acuity (UCVA),[2] the cost and the maintenance of the machine is quite high. So, in developing countries like India where there is huge backlog of cataract surgeries, MSICS is preferred due to its low cost[3] and safety in challenging scenarios like hard cataract,[4] phacolytic[5] and phacomorphic glaucoma.[6]

Though in MSICS, a rigid intraocular lens (IOL) is implanted at an affordable cost, it has high rates of posterior capsular opacification (PCO)[7,8] which can lead to decreased vision increasing the burden on the patient both visually and financially. Moreover, Nd:YAG laser capsulotomy for treating PCO has its own complications like raised intraocular pressure, vitreous prolapse, retinal detachment among others.[9] To avoid these difficulties for the people coming from humble background some of whom even avoid the follow up visits due to lack of awareness or travel difficulties, we can provide the advantages of a foldable IOL with its sharp optic edge which reduces the migration of lens epithelial cellsthereby reducing the incidence of PCO. Singh et al suggests that in MSICS where the incision size is less than 5mm, foldable IOLs can be implanted.[10]

The ideal cases for in-the-bag implantation of a single piece foldable IOL are immature cataract with nuclear sclerosis less than grade 4 and soft mature cataract as in these cases we can keep the sclerocorneal incision size within 6mm and the nucleus can be removed easily through a 5-5.5 mm capsulorrhexis which will provide a 360 degree optic margin overlap which is a prerequisite for preventing PCO. In hard cataracts or in intumescent mature cataracts where we sometimes need large external incision and capsulorrhexis, in-the-bag IOL implantation and optic margin overlap is not always guaranteed. Chang et al confirmed that there is occurrence of late onset secondary pigmentary glaucoma on implantation of foldable IOL in the sulcus.[11] Hence, implantation of single piece foldable IOL in sulcus should be avoided.

If the integrity of the capsulorrhexis is compromised, an attempt to place a single piece foldable IOL in the bag may cause the anterior capsular tear to extend into the posterior capsule. In such cases, it is advisable to place a 3-piece IOL either in the bag or the sulcus. The C loop haptics of the 3-piece acrylic foldable IOL improve stabilization at the sulcus and apply even tension to the adjacent tissues.[12]The higher refractive index of the acrylic materials makes the optic thinner.[13] So, there is greater distance between the IOL and the posterior surface of the iris which reduces the chances of iris chaffing and pigment dispersion. A modification would be to implant the IOL with the haptics in the sulcus while the optic is captured into the bag. This simulates in-the-bag IOL implantation.
In conclusion, foldable IOL can be implanted in MSICS in selected cases and the rates of decreased PCO withfoldable IOL can provide patients with prolonged good visual outcome and better satisfaction.

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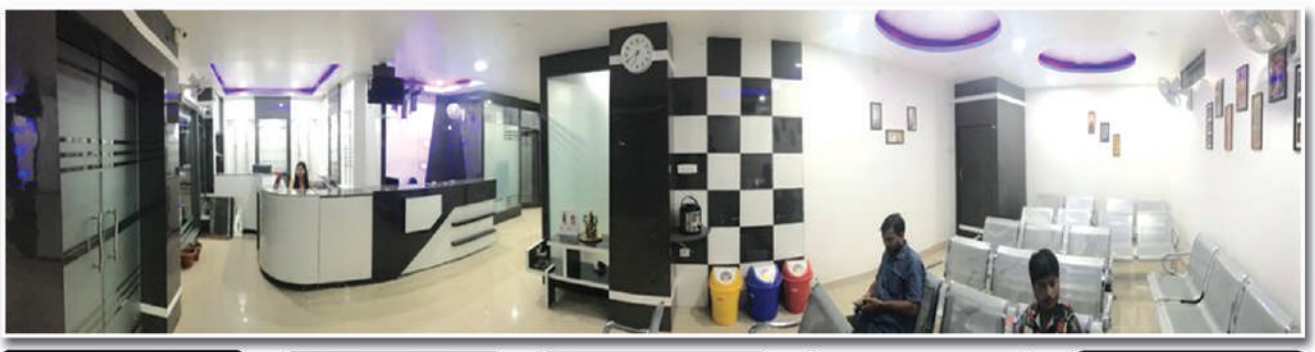




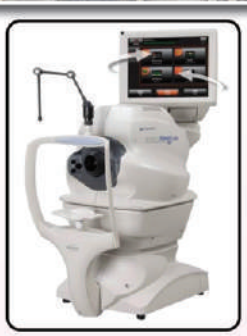
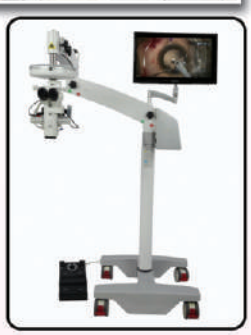

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