

Ophthalmology and Allied Sciences

Editor-in-Chief

Kamal Jeet Singh

Professor & HOD of Ophthalmology,
Moti Lal Nehru Medical College, Allahabad

National Editorial Advisory Board

Anita Panda

Sharda University, Greater Noida

B. Nageswar Rao Subudhi

MKCG Medical College, Berhampur

Bijnya Birajita Panda

AIIMS, Bhubaneswar

Kalpana Suresh

*Sri Ramachandra Medical College, & Research
Institute, Chennai*

Poninder Kumar

Army College of Medical Sciences, New Delhi

Praveen Subudhi,

Ruby Eye Hospital, Berhampur

Roopa R Naik,

*Padmashree Dr. Vitthalrao Vikhe Patil Foundation
Medical College, Ahmednagar*

Salil Kumar,

Gandhi Medical College, Bhopal

Sandeep Saxena

King George's Medical University, Lucknow

Sanjiv Kumar Gupta

King George's Medical University, Lucknow

Shamid Ahmed

J.N. Medical College, Aligarh

Sujata Lakhtakia

Shyam Shah Medical College, Rewa

Taklikar Anupama

Navodaya Medical College, Raichur

V.P. Singh

*Institute of Medical Sciences, Banaras Hindu
University, Varanasi*

Managing Editor

A. Lal

Publication Editor

Manoj Kumar Singh

© 2016 Red Flower Publication Pvt. Ltd. All rights reserved.

The views and opinions expressed are of the authors and not of the **Ophthalmology and Allied Sciences**. Ophthalmology and Allied Sciences does not guarantee directly or indirectly the quality or efficacy of any product or service featured in the the advertisement in the journal, which are purely commercial.

Corresponding address

Red Flower Publication Pvt. Ltd.

48/41-42, DSIDC, Pocket-II, Mayur Vihar, Phase-I

Delhi - 110 091 (India)

Tel: 91-11-22754205, 45796900, Fax: 91-11-22754205

E-mail: info@rfppl.co.in

Website: www.rfppl.co.in

Ophthalmology and Allied Sciences (OAS) (pISSN: 2454-7816, eISSN: 2455-8354) is a half yearly peer-reviewed journal for ophthalmologists and visual science specialists, with a broad international scope. The journal publishes original, peer-reviewed reports of research in ophthalmology, including basic science investigations and clinical studies. Topics include new diagnostic and surgical techniques, treatment methods, instrument updates, the latest drug findings, results of clinical trials, and research findings. In addition to original research papers, the journal presents review articles, editorial comments, an international calendar of events and book reviews.

Subscription Information

Institutional (1 year): INR5500/USD550

Payment methods

Bank draft / cashier s order / check / cheque / demand draft / money order should be in the name of **Red Flower Publication Pvt. Ltd.** payable at **Delhi**.

International Bank transfer / bank wire / electronic funds transfer / money remittance / money wire / telegraphic transfer / telex

1. **Complete Bank Account No.** 604320110000467
2. **Beneficiary Name (As per Bank Pass Book):** Red Flower Publication Pvt. Ltd.
3. **Address:** 41/48, DSIDC, Pocket-II, Mayur Vihar Phase-I, Delhi – 110 091(India)
4. **Bank & Branch Name:** Bank of India; Mayur Vihar
5. **Bank Address & Phone Number:** 13/14, Sri Balaji Shop, Pocket II, Mayur Vihar Phase- I, New Delhi - 110091 (India); Tel: 22750372, 22753401. **Email:** mayurvihar.newdelhi@bankofindia.co.in
6. **MICR Code:** 110013045
7. **Branch Code:** 6043
8. **IFSC Code:** BKID0006043 (used for RTGS and NEFT transactions)
9. **Swift Code:** BKIDINBBDOS
10. **Beneficiary Contact No. & E-mail ID:** 91-11-22754205, 45796900, E-mail: info@rfppl.co.in

Online You can now renew online using our RFPPL renewal website. Visit <http://rfppl.co.in/subscribe.php?mid=7> and enter the required information and then you will be able to pay online.

Send all Orders to: **Red Flower Publication Pvt. Ltd.**, 48/41-42, DSIDC, Pocket-II, Mayur Vihar Phase-I, Delhi – 110 091 (India). Phone: 91-11-22754205, 45796900, Fax: 91-11-22754205, E-mail: customer.rfp@rfppl.co.in, customer.rfp@gmail.com, Website: www.rfppl.co.in

Ophthalmology and Allied Sciences

January - June 2016
Volume 2, Number 1

Contents

Editrial

- Advances in CSR Treatment** 5
Kamaljeet Singh

Original Articles

- Effect of Intravitreal Ranibizumab in CSCR with Ink Blot Type of Leakage and NSD More Than 3 Months Duration** 7
Abhinav Singh, Poninder Kumar, Sagarika Patyal, Gaurav Kapoor, Sonya Puri, Anuradha Makkar
- Strabismus Surgery: Difficult Situations Simplified** 13
Salil Kumar, Pawan Soni, Tejas Patel, Dolly Randive
- To Study the Effect of Topical Diclofenac Sodium 0.1% as An Alternative to Topical Steroid, Dexamethasone Phosphate 0.1% for Post-Operative Control of Inflammation after Small Incision Cataract Surgery** 17
Sharanabasamma M., Vaibhav K.
- Efficacy and Safety of Timolol 0.5% Versus Brimonidine 0.2% in Lowering IOP in Cases of Primary Open Angle Glaucoma** 21
Sharanabasamma M., Vaibhav K.

Case Reports

- An Atypical Case of Sympathetic Ophthalmia Following Zone 1 Corneal Injury** 27
Praveen Subudhi, B.N.R. Subudhi
- Cystoid Macular Oedema Due to Cancer Associate Retinopathy: A Rare Presentation and Its Response to Intravitreal Bevacizumab Injection** 31
Gupta Sanjiv K., Kumar Ajai, Sharma Arun, Katiyar Vishal, Agrawal Siddharth
- A Rare Case of Unilateral Star Shaped Cataract Following Electric Shock** 39
Meena Ashok Kr, Soni Akshar, Gupta Tarun

- Guidelines for Authors** 43

Ophthalmology and Allied Sciences

Library Recommendation Form

If you would like to recommend this journal to your library, simply complete the form below and return it to us. Please type or print the information clearly. We will forward a sample copy to your library, along with this recommendation card.

Please send a sample copy to:

Name of Librarian

Name of Library

Address of Library

Recommended by:

Your Name/ Title

Department

Address

Dear Librarian,

I would like to recommend that your library subscribe to the **Ophthalmology and Allied Sciences**. I believe the major future uses of the journal for your library would provide:

1. useful information for members of my specialty.
2. an excellent research aid.
3. an invaluable student resource.

I have a personal subscription and understand and appreciate the value an institutional subscription would mean to our staff.

Should the journal you're reading right now be a part of your University or institution's library? To have a free sample sent to your librarian, simply fill out and mail this today!

Stock Manager

Red Flower Publication Pvt. Ltd.

48/41-42, DSIDC, Pocket-II

Mayur Vihar Phase-I

Delhi - 110 091(India)

Phone: 91-11-45796900, 22754205, 22756995, Fax: 91-11-22754205

E-mail: customer.rfp@rfppl.co.in, customer.rfp@gmail.com

Advances in CSR Treatment

This is the second issue of our journal 'Ophthalmology and Allied Sciences'. In this issue we have several good articles for the readers. A special attention needs to be drawn towards the article on Central Serous Retinopathy(CSR)[1] bysuggesting the role of anti VEGF in improvement of vision, delay in inkblot pattern of leaks on FFA and improvement in mean central macular thickness on OCT. This treatment modality could be used as alternative to laser therapy in chronic CSR.

The gold standard treatment of CSR has always been masterly inactivity as the disease is self limiting [2]. Recently the treatment options for CSR have undergone a sea change. In case the resolution does not occur in three months laser photocoagulation is the treatment of choice for well defined focal and extrafocal leaks but side effects such as permanent scotoma, laser scar enlargement and laser induced CNV occur. In addition, it does not influence the visual outcome or rate of recurrence. When the leak is foveal or diffuse, infrared micropulse laser is the treatment of choice. They may be considered first line treatment methods. The risk factors should also be tackled e.g. discontinuing exogenous steroids intake in any form.

For chronic CSR, recurrent CSR and first time CSR attack of more than three months old, apart from laser photocoagulation and micropulse diode laser photocoagulation, transpupillary thermotherapy (TTT), standard photodynamic phototherapy(PDT), and PDT with reduced dose(half to one-third), PDT with reduced fluence(decreasing laser time or power) have been tried. All these studies have been undertaken in an attempt to reduce the choriocapillary ischemia. The results have been gratifying but more studies are needed to achieve the best results with the least possible complications [3].

In the article 'Effect of intravitreal ranibizumab in CSR with ink blot type of leakage and neurosensory retinal detachment (NSD) >3 months duration', an attempt has been made to treat the patients on the hypothesis that choroidal hyperpermeability is

associated with increased activity of VEGF. But higher levels of VEGF have not been detected in aqueous humour[4]. Other studies have found that anti VEGF has well established role in CNVMs secondary to CSR but its primary role in CSR needs further studies[5].

Several clinical trials have been done to observe the effects of anti corticosteroids on the basis that there is increased cortisol activity in patients of CSR which may be decreased by giving anticorticosteroid treatment. Important among anticorticosteroids are ketoconazole, mifepristone(RU-486), spironolactone and eplerenone. They have been tried but are not associated with significantly better outcome[6].

Adrenergic blockers, systemic carbonic anhydrase inhibitors, aspirin, helicobacter pylori and methotrexate have also been tried but in all these studies no conclusion could be drawn and more studies are needed [6].

Finally, the gold standard of treatment of CSR remains the masterly inactivity in majority. Further intervention in chronic CSR may be undertaken on individual case basis.

References

1. Effect of intravitreal ranibizumab in CSR with ink blot type of leakage and neurosensory retinal detachment (NSD) >3 months duration. Journal Ophthalmology and Allied Sciences.
2. Ross A, Ross AH, Mohamed Q. Review and update of central serous chorioretinopathy. Curr Opin Ophthalmol. 2011; 22(3): 166-73. <http://dx.doi.org/10.1097/ICU.0b013e3283459826>.
3. Nicoló M, Eandi CM, Alovisei C, et al. Half-fluence versus half-dose photodynamic therapy in chronic central serous chorioretinopathy. Am J Ophthalmol. 2014; 157(5): 1033-7. <http://dx.doi.org/10.1016/j.ajo.2014.01.022>.
4. Lim JW, Kim MU, Shin M-C. Aqueous humor and plasma levels of vascular endothelial growth factor and interleukin-8 in patients with central serous chorioretinopathy. Retina 2010; 30(9): 1465-71.

- [http:// dx.doi.org/10.1097/IAE.0b013e3181d8e7fe](http://dx.doi.org/10.1097/IAE.0b013e3181d8e7fe).
5. Montero JA, Ruiz-Moreno JM, Fernandez-Muñoz M. Intravitreal bevacizumab to treat choroidal neovascularization following photodynamic therapy in central serous choroidopathy. *Eur J Ophthalmol*. 21(4): 503-5. doi: <http://dx.doi.org/10.5301/EJO.2011.6290>.
 6. Marwan A. Abouammoh, MD, Advances in the treatment of central serous chorioretinopathy. *Saudi Journal of Ophthalmology*. 2015; 29: 278-286. [http://www.saudiophthaljournal.com/article/S1319-4534\(15\)00023-5/pdf](http://www.saudiophthaljournal.com/article/S1319-4534(15)00023-5/pdf).

Kamaljeet Singh

Editor-in- chief

Ophthalmology and Allied Sciences

Professor and Head

Department of Ophthalmology

MLN Medical College, Allahabad.

Email id: kamaljs2@rediffmail.com

Effect of Intravitreal Ranibizumab in CSCR with Ink Blot Type of Leakage and NSD More Than 3 Months Duration

Abhinav Singh*, Poninder Kumar**, Sagarika Patyal***, Gaurav Kapoor****, Sonya Puri****, Anuradha Makkar*****

Authors Affiliation: *Resident, Ophth Base Hospital & ACMS, Delhi Cantt, **Professor & HOD, Ophth Base Hospital & ACMS, Delhi Cantt, ***Formerly Professor & HOD, Base Hospital & ACMS, Delhi Cantt, ****Associate Professor, Base Hospital & ACMS, Delhi Cantt, *****Prof & Head Microbiology Base Hospital & ACMS, Delhi Cantt.

Abstract

Objective: To evaluate the effect of intravitreal Ranibizumab in CSCR with ink blot type of leakage and NSD > 3 months duration. **Study Design:** Prospective Study. **Place and Duration of Study:** Base Hospital Delhi Cantt, New Delhi 110010 and Army College of Medical Sciences. **Methodology:** 20 eyes of 20 adult patients with CSCR were included in the prospective study of duration 08 weeks (02 months). Patients with bilateral CSCR, smoke stack and diffuse leakage of dye on FFA, choroidal neovascularization, treated cases of CSCR, history of thromboembolism, and intraocular inflammation were excluded from the study. After informed consent, all patients were given intravitreal injection of Ranibizumab. Best corrected visual acuity (BCVA) and central macular thickness (CMT) measurement with OCT and pattern of leaks were recorded on FFA at baseline and follow up at 02 and 08 weeks. The outcome measures were mainly BCVA status, CMT on OCT and changes in pattern of leaks on FFA pre and post Anti VEGF. **Results:** There were 15 (75%) males and 5 (25%) females. All cases were unilateral. Mean age was 39.09 ± 8.49 years. 11 (55%) eyes showed between 3 to 6 months involvement and 9 (45%) eyes showed more than 6 months involvement. All the cases were treated with single intravitreal dose of 0.5 mg Ranibizumab. After 08 weeks followup, It was observed that the CSCR with ink blot pattern showed moderate visual gain as well as 66.6% decrease in leak intensity on FFA ($p < 0.001$). In addition, mean CMT on OCT showed 70% ($p < 0.001$) decrease at 02 months follow up period. **Conclusion:** Intravitreal Ranibizumab injection was associated with improvement in BCVA, decrease in intensity as well as delay in onset of ink blot pattern of leaks on FFA and improvement in mean CMT as well as decreased NSD height on OCT in patients of CSCR.

Keywords: Central Serous Chorioretinopathy; Injection Ranibizumab; Optical Coherence Tomography, Intravitreal Injection.

Introduction

Central Serous Chorioretinopathy (CSCR), a condition which is characterized by an idiopathic serous neuro sensory detachment primarily affecting the macula [1]. CSCR is associated with retinal pigment epithelial (RPE) leakage and angiographic RPE and choroidal hyper-permeability [2]. CSCR is among the top ten most common diseases affecting the macula and is a common disorder in young and

middle aged patients. In most cases, recovery of vision follows acute episodes. However, there can be permanent loss of vision with repeated episodes, persistent macular detachment or diffuse disease [3].

Reprint Request: Poninder Kumar,
Prof & HOD Ophth Base Hospital & Army College of
Medical Sciences (ACMS), Near Base Hospital, Bar
Square, New Delhi, Delhi 110010.
E-mail: poninder@hotmail.com

CSCR frequently manifests symptomatically in one eye, while 18% of cases may be bilateral. Research indicates that the disease process in CSCR is more diffuse and shows bilateral retinochoroidal dysfunction, even when the disease is manifesting clinically only in one eye [5].

CSCR is commonly associated with type-A personalities, organ transplantation, systemic lupus erythematosus and Cushing disease [6]. Patients with CSCR show impaired autonomic response with significantly decreased parasympathetic activity and significantly increased sympathetic activity [6]. Glucocorticoids and possibly adrenergic hormones play a role in the pathophysiology of CSCR and exert their effects on the retinal pigment epithelium, choroid or both [8].

CSCR has been associated with the abnormalities of choroidal circulation [8,9]. It is associated with development of choroidal ischaemia that possibly leads to hyperpermeability of the choroidal vessels. Leakage in the choroid might affect the overlying retinal pigment epithelium and lead to serous RPE detachment and neurosensory detachments [3].

Photodynamic therapy, laser photocoagulation and pharmacological agents (acetazolamide, propranolol, mifepristone and ketoconazole) have been used to treat CSCR. However, these treatment options serve only to shorten the duration of symptoms and have no effect on the recurrence rate and the final visual acuity [10]. In cases with chronic diffuse or persistent focal leakage, retinal pigment epithelium may decompensate leading to gradual visual loss with a less favourable visual prognosis [11]. The pathophysiology of CSCR remains unclear. Recent studies relying on indocyanine green angiography (ICG) have shown that the aetiology may begin with the changes in choroidal permeability [12]. It seems reasonable to target the choroidal vascular changes with new strategies to treat CSCR.

Laser photocoagulation is applied to the site of fluorescein leakage. Although this has been proved to reduce the duration of the serous detachment, it has no effect on the final visual prognosis.

More recently, photodynamic therapy has been reported to be a more effective treatment with a lower complication rate for patients with subfoveal or juxtafoveal leaks.

Ranibizumab, being an antibody to vascular endothelial growth factor A (VEGF-A), as well as having anti-permeability properties therefore, may theoretically reverse the changes seen in CSCR.

This study was performed to evaluate the effect of intravitreal Ranibizumab in CSCR with ink blot type of leakage and NSD in more than 3 months duration

Methodology

The study was conducted after the approval of research/ethical committee of the hospital. This prospective study included 20 eyes of 20 patients with CSCR. Both genders between 22 and 54 years were included. Patients having acute or chronic CSCR were studied. Acute CSCR was defined as resolution of disease before 3 months, while chronic CSCR persisted longer than 3 months.

Inclusion criteria were subfoveal fluid documented by OCT and active leak ink blot type documented by fundus fluorescein angiography. Exclusion criteria were bilateral cases of CSCR, case with smoke stack and diffuse leaks on FFA, choroidal neovascular membrane, prior treatment with laser photocoagulation, transpupillary thermotherapy or photodynamic therapy, history of thromboembolic events including stroke, transient ischaemic attacks, intraocular inflammation and history of previous treatment with intra vitreal anti-VEGF.

Patients fulfilling the inclusion criteria were selected from Retina Clinic of Base Hospital Delhi cantt. Informed consent was taken from all patients. Socio demographic profile like name, age, gender and history of current disease with respect to symptoms, severity and duration was taken. At baseline and follow-up visits, examination included detailed anterior segment examination with slit lamp, visual acuity with Snellen's chart (converted into decimal), intraocular pressure measurement with Goldman's applanation tonometer and dilated fundus examination. Fundus fluorescence angiography (FFA) and optical coherence tomography (OCT) to document leak and retinal thickness respectively was performed at baseline examination and at each follow-up visit which was 4 weeks apart after intervention with Anti VEGF agent. Outcome measures were changes in pattern of leaks of ink blot variety on FFA and resolution of neurosensory detachment measured as CMT on OCT

All patients were instructed to instill E/D moxifloxacin 6 times one day prior to the intervention. In all patients, the intravitreal injection of Ranibizumab was given in the operation theater under complete aseptic conditions. Proparacaine 0.5% topical eye drops were instilled followed by scrubbing of eyelids by 10% povidone-iodine and conjunctiva instilled with 5% povidone-iodine

several minutes before the procedure. A sterile eyelid speculum was inserted. Topical proparacaine was instilled and the preferred site of injection was the supero-nasal quadrant. Inj Ranibizumab was injected through the pars plana 3.5 – 4.0 mm posterior to the surgical limbus using a 30 gauge needle at a dose of 0.5 mg in 0.05 ml. Post-injection, a sterile cotton swab is placed at the site of injection to prevent reflux of vitreous or the drug. Topical antibiotic drop was instilled and a sterile pad placed few hours. Patients were instructed to apply topical antibiotic drops 4 times a day for 5 days. Post injection follow-up included repeated clinical examination. Patients were assessed for adverse events including elevated intraocular pressure, cataract progression, retinal detachment, post-injection inflammation and endophthalmitis. Follow-up visits were scheduled to next day, at appx 1 week, at 04 weeks and at 08 weeks. Repeated OCT was performed after every 4 weeks. The FFA was repeated at 08 weeks duration to observe the changes in pattern of leaks and at times at 04 weeks too.

The data was entered into Statistical Package for Social Sciences (SPSS) version 17 and analyzed accordingly. The variables analyzed were demographics (age, gender) and examination. The quantitative data (age) was presented with simple descriptive statistics like mean and standard deviation. Mean was calculated for BCVA and CMT. The qualitative data (gender) presented as frequency and percentage. P-value equal to or less than 0.05 was considered statistically significant.

Results

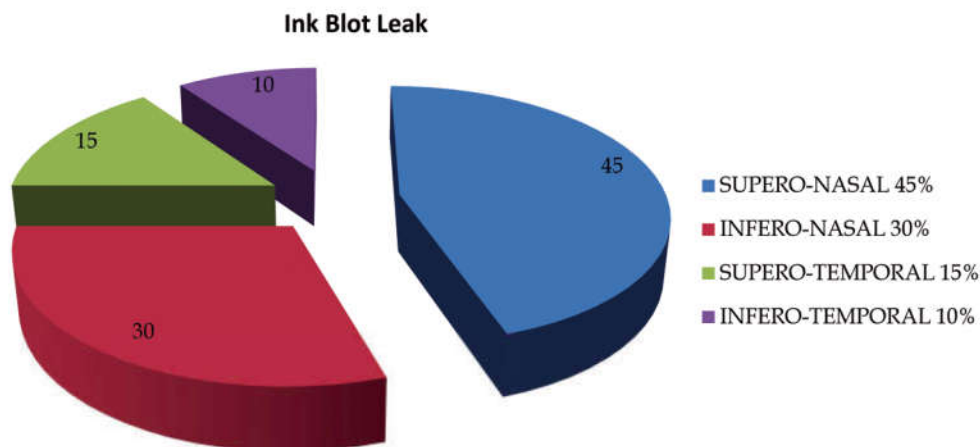


Fig. 1: Showing pattern of leak of ink blot type on Fluorescein Angiography (FA) in 20 eyes

This study included 20 eyes of 20 patients with CSCR. There were 15 (75%) males and 5 (25%) females. Mean age was 39.09 ± 8.49 years. 11(55%) eyes showed less than 6 months involvement and 9(45%) eyes showed more than 6 months involvement however all were over 03 months duration. All patients presented with complaint of decreased vision. 7(23.3%) patients presented with positive scotoma and 8(26.7%) patients presented with metamorphopsia as their main presenting complaint.

On FFA, only patients with ink blot pattern leaks on FFA were included in study. Total number of Ink Blot leaks were 20. 9(45%) patients showed ink blot leaks on FFA in supero-nasal quadrant of posterior pole, 6(30%) in infero-nasal quadrant of posterior pole, 3(15%) in supero-temporal quadrant of posterior pole and 2(10%) in infero-temporal quadrant of posterior pole. 8 (40%) patients had one leak and 12(60%) had two or more than two leaks.

Mean CMT (central macular thickness) on OCT was $375 \mu m$ with a sub foveal neuro-sensory detachment. CMT decreased in 16 patients while in 04 it increased or remained the same. As far as BCVA is concerned it improved by 2 lines or more in 13 patients and in the remaining the either there was deterioration or the vision remained static. FFA picture of Ink Blot leaks showed significant decrease in intensity as well as size in 13 patients out of 20.

A gain of two lines was considered significant. Table 3 shows the various parameters of BCVA (snellen's converted to decimal), NSD on OCT and FFA picture base line and post inj Ranibizumab. Figure 4 shows the baseline as well as post inj Ranibizumab BCVA. Mean CMT on OCT was 375

Fig. 1: above shows different patterns of leaks of ink blot type encountered during FFA at time of presentation. Figure 2 shows the the typical FFA pattern while Figure 3 shows the resolution of NSD on OCT.

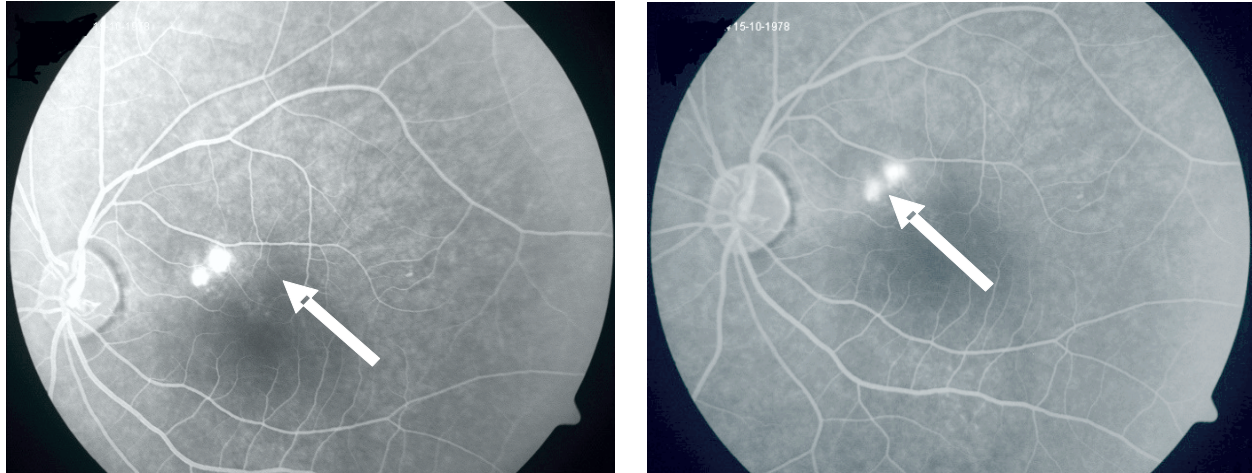


Fig. 2: Typical INK BLOT LEAK (marked by arrow head) in superonasal quadrant at 04:00 mins in a young Male patient on FFA& and shows improvement in area as well as decrease of leak in same patient at 5:05 mins, at 08 weeks follow up post Lucentis

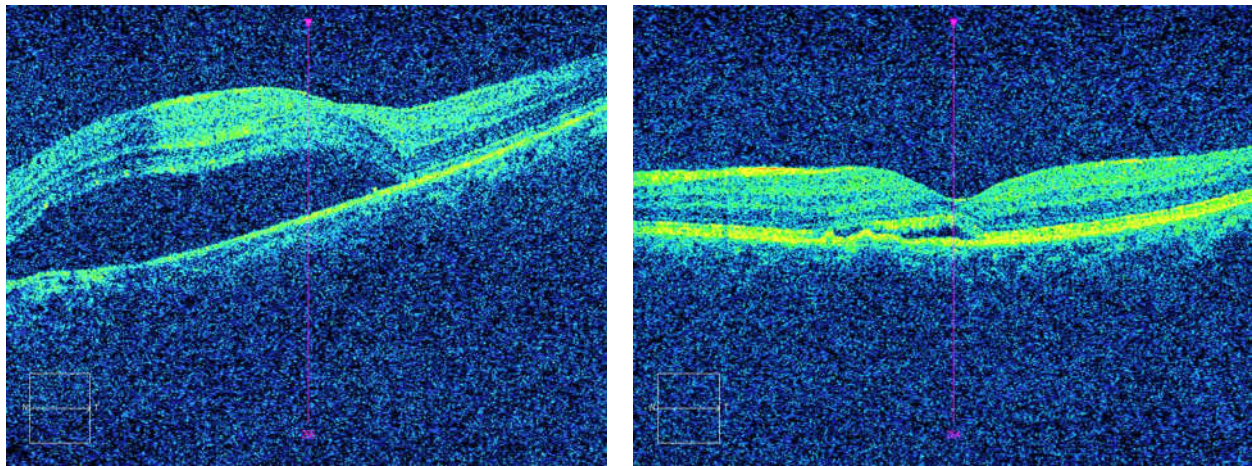


Fig. 3: CMT (central macular thickness) of 380 μm and post injection shows CMT of 250 μm of same patient at 08 weeks follow up post Inj Ranibizumab

Table 1: Baseline and Post Ranibizumab BCVA, CMT & Leaks on FFA

	Baseline	Post INJ Ranibizumab
BCVA (decimal)	0.8	0.5
MEAN CMT (μm)	375	259
LEAKS on FFA	20 patients showed leaks	13 showed improvement in size and intensity

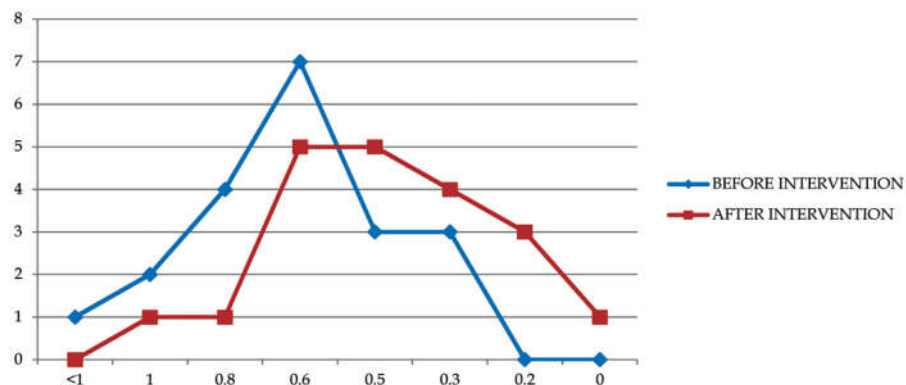


Fig. 4: Showing improvement in BCVA over a period of 02 months

um at baseline and decreased to 259 μ m. There was significant improvement in CMT of 70% ($p < 0.001$).

Half fluence PDT was kept as a rescue option for all patients of chronic CSCR who did not respond to Anti VEGF therapy.

Discussion

In this prospective study, the effect of intravitreal injection of Ranibizumab on ink blot leaks on FFA and CMT on OCT in 20 cases CSCR was evaluated. The precise pathophysiology of CSCR remains unclear. There is no standard treatment for it. Various medical treatments have been attempted to treat it, including acetazolamide, beta-blockers, vitamins and non-steroidal anti-inflammatory medicines.

There was reduction in intensity and time of onset of leak in all patterns observed during study after 02 months of single injection of Ranibizumab. The leaks which appeared in early phases on FFA before intervention were delayed and reduced in intensity as well as area. There was improvement in BCVA and CMT on OCT noted at all follow-up visits.

Median CMT at baseline was 375 μ m and at 2 month was 259 μ m. Difference between baseline and 2 month CMT was statistically significant, $p < 0.001$. The BCVA showed improvement from 0.8 to 0.5 on decibel scale. These results show anatomic and functional improvement following intravitreal Ranibizumab injections, which suggest that VEGF may be involved in fluid leakage in patients with CSCR.

Ranibizumab is a recombinant humanized full-length monoclonal antibody that binds VEGF- A isoform. The Ranibizumab molecule can penetrate the retina and is transported into the RPE, the choroid and photoreceptors outer segments after intravitreal injection [19]. Intravitreal Ranibizumab has been utilized to treat ocular disorders, which are associated with neovascularization or vascular leakage as a result of an underlying disease. In this study, it was demonstrated that intravitreal Ranibizumab injection in patients with CSCR could bring resolution of subretinal fluid, which was accompanied by improvement in BCVA. The mechanism by which the intravitreal Ranibizumab therapy brings relief is unknown but it may be related to its ability to affect vascular permeability [1]. Recent studies relaying on ICG have shown that the aetiology of CSCR rests on choriocapillaris, in which a focal increase in the permeability of the choriocapillaris overwhelms the RPE and causes leakage of fluid into the subretinal space and

subsequent RPE detachment. The hyperpermeability of choriocapillaris may be caused by capillary and venous congestion, possibly because of choroidal ischemia. Localized choroidal ischaemia has been observed in normal fellow eyes of some patients of CSCR [12]. Choroidal ischemia in CSCR may induce an increase in the concentration of VEGF, which has profound effects on vascular permeability [1,20]. Therefore, theoretically reduced levels of VEGF may improve choroidal ischemia.

Laser photocoagulation may accelerate the resolution but it can result in permanent scotoma, which may enlarge with time, and laser can induce choroidal neovascularization (CNV) [15]. Indocyanine green (ICG) guided photodynamic therapy (PDT) has been used for the treatment of CSCR [16]. But PDT is expensive and cases of CNV and severe choroidal ischaemia have been reported with use of PDT [17,18].

The results suggest a possible role for anti-VEGF agents in the treatment of CSCR. However, limitations of this study include a short follow-up and small number of patients. Further evaluation of intravitreal Ranibizumab for CSCR patients in controlled randomized large number of patients with longer follow-up period are necessary to confirm the efficacy and safety of Ranibizumab and to determine the ideal protocol for this new promising treatment.

It would be helpful if a larger cohort be included in the study with longer follow up duration to assess the role of Intravitreal Ranibizumab, FFA changes & CMT on OCT in CSCR.

Conclusion

Intravitreal Ranibizumab injection was associated with improvement in ink blot leaks on FFA and CMT (central macular thickness) on OCT in chronic CSCR, in the majority of patients. All patients who had reduction in CMT did not necessarily manifest with visual improvement. It was observed that 65% improvement in BCVA, ink blot leaks reduction in 65% and decrease in CMT on OCT in 80% patients. These short-term results suggest that intravitreal Ranibizumab injection may constitute a promising therapeutic option in central serous chorioretinopathy.

References

1. J.Gaudreault, D. Fei, J.C.Beyer, A.Ryan, L. Rangell, V.Shui, L.A.Damico, "Pharmacokinetics and

- Retinal Distribution of Ranibizumab, a Humanized Antibody Fragment Directed against VEGF-A, Following Intravitreal Administration in Rabbits," *Retina*. 2007; 27(9): 1260-1266.
2. Haimovici R, Koh S, Gangnon DR, Lehrfeld T, Wellik S. Risk factors for central serous chorioretinopathy. *Ophthalmology* 2004.
 3. Jampo ILM, Weinreb R, Yannuzzi L. involvement of corticosteroids and catecholamines in the pathogenesis of central serous chorioretinopathy: a rationale of new treatment strategies. *Ophthalmology*. 2002; 109: 1765-6.
 4. Gass JD, editor. Stereoscopic atlas of macular diseases: diagnosis and treatment. 4th ed. St. Louis: Mosby; 1997.
 5. P. J. Rosenfeld, J. S. Heier, G. Hantsbarger and N. Shams, "Tolerability and Efficacy of Multiple Escalating Doses of Ranibizumab (Lucentis) for Neovascular Age-Related Macular Degeneration," *Ophthalmology*. 2006; 113(4): 623-632.
 6. Stephen L, Perkins, Judy E, Kim, John S. Pollack, Pauline T. Clinical characteristics of central serous chorioretinopathy in women. *Ophthalmology*. 2002; 109: 262-6.
 7. Tewari HK, Gadia R, Kumar D, Venkatesh P. Sympathetic & parasympathetic activity and reactivity in central serous chorioretinopathy. *Invest Ophthalmol Vis Sci*. 2006; 47: 3474-8.
 8. Guyer DR, Yannuzzi LA, Slakter JS. Digital indocyanine green video angiography of central serous chorioretinopathy. *Arch Ophthalmol*. 1994; 112: 1057-62.
 9. Prunte C, Flammer J. Choroidal capillary and venous congestion in central serous chorioretinopathy. *Am J Ophthalmol*. 1996; 121: 26-34.
 10. Schaal K B, Hoeh AE, Scheuerle A, Schuett F, Dithmar S. intravitreal Bevacizumab for treatment of chronic central serous chorioretinopathy. *Eur J Ophthalmol*. 2009; 19: 613-7.
 11. Jalk AE, Jbbour N, Avila MP. Pigment epithelium decompensation, clinical features and natural course. *Ophthalmology*. 1984; 91: 1544-8.
 12. Taban M, Boyer DS, Thomas EL, Taban M. Chronic central serous chorioretinopathy: photodynamic therapy. *Am J Ophthalmol*. 2004; 137: 1073-80.
 13. Pikkil J, Beiran L, Ophir A, Miller B. Acetazolamide for central serous retinopathy. *Ophthalmology*. 2002; 109: 1723-5.
 14. Bujarborua D, Chatterjee S, Choudhury A, Bori G, Sarma AK. Fluorescence angiographic features of asymptomatic eyes in central serous chorioretinopathy. *Retina*. 2005; 25: 422-9.
 15. Burumcek E, Mudun A, Karacorlu S, Arslan MO. Laser photocoagulation for persistent central serous retinopathy: results of long-term follow-up. *Ophthalmology*. 1997; 104: 616-22.
 16. Battaglia Parodi M, Da Pozzo S, Ravalico G. Photodynamic therapy in chronic central serous chorioretinopathy. *Retina*. 2003; 23: 235-7.
 17. Colucciello M. Choroidal neovascularization complicating photodynamic therapy for central serous retinopathy. *Retina*. 2006; 26: 239-42.
 18. Lee PY, Kim KS, Lee WK. Severe choroidal ischemia following photodynamic therapy for pigment epithelial detachment and chronic central serous chorioretinopathy. *Jpn J Ophthalmol*. 2009; 53: 52-6.
 19. Heiduschka P, Fietz H, Hofmeister S, Schultheiss S, Mack AF, Peters S. Penetration of Bevacizumab through the retina after intravitreal injection in the monkey. *Invest Ophthalmol Vis Sci*. 2007; 48: 2814-3.
 20. Mazhar PS, Hanfi AN, Khan A. Angiogenesis is and role of anti VEGF therapy. *Pak J Ophthalmol*. 2009; 25: 169-73.

Strabismus Surgery: Difficult Situations Simplified

Salil Kumar*, Pawan Soni*, Tejas Patel*, Dolly Randive*

Authors Affiliation: Regional Institute of Ophthalmology, Gandhi Medical College, Bhopal.

Abstract

Purpose: In the present study we assessed the vertical effect of horizontal muscle transposition when performing a resection-recession procedure on a patients with moderate vertical deviation along with horizontal deviation with some additional displacement of horizontal recti i.e. 3/4th instead of 1/2 width displacement. *Material and Method:* Present study was carried out in a series of 20 cases in the age group 3-40 years of both sexes with complicated squint having both horizontal and vertical deviation of varying amount with special emphasis on sensory status. Cases were broadly divided into two groups- Group 1(12 cases): Patients with Exotropia (40-50PD) and Hypertropia of <12 PD. Group 2(8 cases): Patients with exotropia(40-50PD) and Hypertropia of 12-20 PD. *Results:* Postoperative vertical deviation corrections are graded as- Very Good :- < 2-4PD. Moderate: - 4-8PD, Fair: - 10-16 PD. In group 1-8 cases (66%) out of 12 results were very good; Rest 4 cases (34%) results were moderate. In group 2- 6 cases (75%) out of 8 results were very good; Rest 2 cases (25%) results were moderate. *Conclusion:* Vertical muscle displacement is a very good option for correction of moderate vertical deviation especially for ophthalmic surgeons not experienced in tackling oblique muscles. Present study suggests that 3/4th width muscle displacement was more effective than 1/2th width muscle displacement.

Keywords: Squint surgery; Strabismus Surgery; Hypertropia; Exotropia

Introduction

Aim of squint surgery is not only to correct deviation but also to improve field of vision, binocularity and stereopsis when performed at earlier age (up to 12 years) but when performed at later age(above 12 years) above mentioned benefits is less evident. In late age group more important benefits are correction of deviation, cosmetic improvement and gain in confidence

Vertical deviation along with horizontal deviation is not an uncommon condition, It is seen due to oblique over action, A or V phenomenon or can be present in primary position without an apparent oblique over action. Small amount of vertical deviation is common with large angle horizontal deviation.

Materials and Method

Present study was carried out in a series of 20 cases in the age group 3-40 years of both sexes with complicated squint having both horizontal and vertical deviation of varying amount.

Detailed ophthalmic examination including Visual acuity, cycloplegic refraction and fundus examination was done.

Squint work up including - Hirschberg test , PBCT (prism bar cover test), both for near and distant in all gazes , sensory status of BSV (binocular single vision) by using worth four dot test, type of fixation whether

Reprint Request: Salil Kumar,
E/3-73 Arera Colony, Bhopal- 462016 (M.P).
E-mail: dr_salilkumar@gmail.com

central or eccentric were assessed. Preoperative treatment for refractive error or Amblyopia was given before squint surgery.

Cases were Broadly Divided into Two Groups

Group 1(12 cases)

Patients with Exotropia(40-50PD) and Hypertropia of <12 PD.

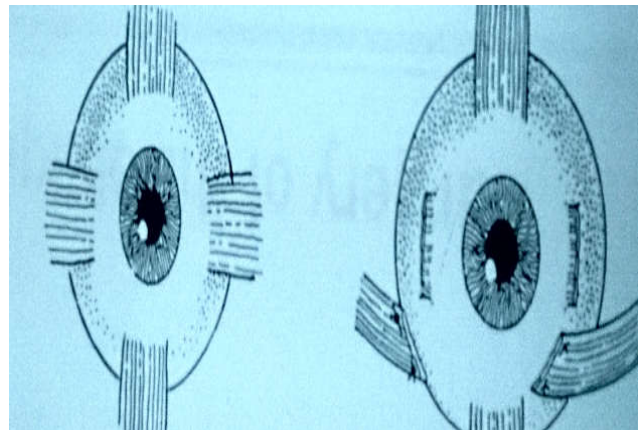
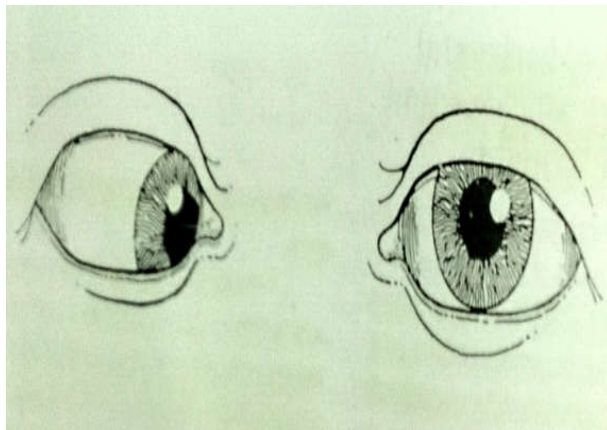
Group 2 (8 cases)

Patients with exotropia(40-50PD) and Hypertropia of 12-20 PD.

All these patients underwent surgery by a single Surgeon; plan executed as per squint work up for horizontal squint with addition of vertical correction.

Surgical Procedure

Group 1 (12 cases) underwent unilateral lateral rectus (LR) recession and medial rectus (MR)



Preoperative



Postoperative

resection for unilateral squint and bilateral LR recession for alternate squint with addition of downward displacement of half width of horizontal muscle for group 1.

In group 2 management for horizontal squint was same as group 1 with addition of downwards displacement of three fourth width of horizontal muscle for vertical squint correction.

Results

Postoperative vertical deviation corrections are graded as-

Very Good :- < 2-4PD

Moderate :- 4-8PD,

Fair :- 10-16 PD.

In group 1-8 cases (66%) out of 12 results were very good; Rest 4 cases (34%) results were moderate.

In group 2- 6 cases (75%)out of 8 results were very good; Rest 2 cases (25%) results were moderate.

Discussion

In normal eyes with good fusion vertical deviation up to 4PD is controlled; if more than 6PD it becomes manifest as AHP(abnormal head posture) or diplopia in vertical gaze .

A small angle of hyper deviation (<6PD) is common with large angle of horizontal deviation (>40PD)and it doesn't require any additional treatment .

Results for vertical squint were moderate(4-8PD) to fair (6-8PD)with half width displacement of the horizontal recti.

Our study shows that when some additional displacement of horizontal recti i.e. 3/4th instead of 1/2 width the results were satisfactorily very good.

In our study additional extra displacement (three fourth width displacement as compared to half width) the results were very good to moderate .

In the present study we assessed the vertical effect of horizontal muscle transposition when performing a resection-recession procedure on a patient who also has moderate vertical deviation along with horizontal deviation.

The muscles are moved upward one-half muscle width or more if the eye is hypodeviated and downward one-half muscle width or more if the eye is hyperdeviated

Both rectus muscles shifted vertically in the same direction. This approach can treat the vertical deviation without altering the effect of the procedure for the esodeviation or exodeviation.

Rationale for vertical transposition is based on the observation that strength of the horizontal rectus muscle is increased when the eye is vertically rotated in the direction opposite to the direction of its

transposed insertion.

For example, lowering the insertion of a horizontal rectus muscle improves the effect of this muscle when the eye is in elevation.

Conversely, an elevated horizontal rectus muscle produces more effect in depression.

Combined horizontal with vertical deviation should be differentiated from pattern deviation (A OR V) or oblique overaction because each condition has different management.

Vertical transpositions of horizontal muscles do not appreciably alter the horizontal alignment in primary position.

Conclusion

Horizontal muscle surgery is much easier than oblique surgery .

This is an alternate approach and effective means for management of vertical deviation.

Many Ophthalmologists are not experienced for oblique muscles handling.

Half muscle displacement is found to be good for mild to moderate cases and 3/4th displacement should be tried for larger deviation.

This is true only if there is no significant oblique overaction; if present appropriate oblique muscle surgery must be performed

References

1. Kushner BJ: Arch Ophthalmol. 1988; 106: 18.
2. Esswein MB, von Noorden GK: Am J Ophthalmol. 1993; 116: 424.
3. Hamilton SM, Elsas FJ, Dawson TL: J PediatrOphthalmol Strabismus. 1993; 30: 288.
4. Freeman RS: Am Orthopt J. 1994; 44: 2.
5. Munoz M: Am J Ophthalmol. 1994; 118: 664.
6. Hunter DG, Lam GC, Guyton DL. 1995; 102: 501.

Red Flower Publication Pvt. Ltd.

Presents its Book Publications for sale

- | | |
|--|---------------------|
| 1. Breast Cancer: Biology, Prevention and Treatment | Rs.395/\$100 |
| 2. Child Intelligence | Rs.150/\$50 |
| 3. Pediatric Companion | Rs.250/\$50 |

Order from

Red Flower Publication Pvt. Ltd.

48/41-42, DSIDC, Pocket-II

Mayur Vihar Phase-I

Delhi - 110 091(India)

Phone: 91-11-45796900, 22754205, 22756995, Fax: 91-11-22754205

E-mail: customer.rfp@rfppl.co.in, customer.rfp@gmail.com, Website: www.rfppl.co.in

To Study the Effect of Topical Diclofenac Sodium 0.1% as An Alternative to Topical Steroid, Dexamethasone Phosphate 0.1% for Post-Operative Control of Inflammation after Small Incision Cataract Surgery

Sharanabasamma M.*, Vaibhav K.**

Authors Affiliation: *Assistant Professor **Post Graduate, Dept of Ophthalmology, Navodaya Medical College, Raichur Karnataka, India.

Abstract

Aims: To study the effect of topical Diclofenac sodium 0.1% as an alternative to topical steroid, Dexamethasone phosphate 0.1% for post-operative control of inflammation after small incision cataract surgery. **Settings and Design:** Double blinded study done in a tertiary care hospital. **Methods and Material:** 100 patients with uncomplicated senile cataract who underwent uneventful small incision cataract surgery with posterior chamber intra ocular lens implantation were selected and divided into 2 groups of 50 each. One group was given dexamethasone phosphate 1%, other group diclofenac sodium 0.1% topically. They were examined on 7, 15, 30th day for congestion, corneal edema, anterior chamber flare, cells, intraocular pressure and grading was done as per severity and total score was assessed. The results were compared between the two groups. **Statistical Analysis used:** Chi square test with p value < 0.05 as significant. **Results:** Day 7 and 15 the response was good for dexamethasone group for all the parameters. By day 30 there was no significant difference between the two groups in all the parameters. No significant difference in IOP at baseline and at 6 weeks post operatively between the two groups. **Conclusions:** The resolution of inflammation was quicker in the dexamethasone group than in the diclofenac group. But by the end of 30 days the effect on inflammation in both the groups are similar. So Diclofenac sodium 0.1% can be used as an alternative to Dexamethasone phosphate 0.1% in post operative patients following cataract surgery.

Keywords: Postoperative Inflammation; Dexamethasone Phosphate 0.1%; Diclofenac sodium 0.1%.

Introduction

In India 3.8 million become blind yearly due to cataract [1]. Mild post-operative inflammation is a normal accompaniment occurs due to surgical trauma leading to disruption of blood aqueous barrier, leakage of protein and cells into anterior chamber triggering the inflammatory cascade[2]. Anti-inflammatory therapy is to hasten the resolution and avoid complications of prolonged inflammation, like pain, photophobia, foreign body sensation, reduced visual acuity, posterior synechiae, raised intraocular pressure [3].

Topical corticosteroids are effective in suppressing

postoperative inflammation. However, they have many side effects, like impaired wound healing, elevation of intraocular pressure and increased tendency of infections, tear-film instability[4]. Recent studies suggest that topical non-steroidal anti-inflammatory drugs (NSAIDs) are as effective as corticosteroids in re-establishing the blood aqueous barrier following cataract surgery.

This study aims at studying the effect of topical Diclofenac sodium 0.1% as an alternative to topical

Reprint Request: Sharanabasamma M.,
Sri Nursing & Maternity Home, Near Ek Minar,
Beharoon quilla, Raichur Ho, Raichur - 584101, karnataka
E-mail: dr.msharanabasamma@gmail.com

steroid, Dexamethasone Phosphate 0.1% for post-operative control of inflammation after cataract surgery.

Key Message

Topical Diclofenac is an alternative for topical Dexamethasone after SICS.

Subjects and Methods

This study was conducted in a tertiary care hospital from 2013 to 2014. Written informed Consent was obtained from the patients. It is a randomized double blind study. Included 100 patients.

All the patients who were having an uncomplicated senile cataract who underwent uneventful SICS+PCIOL implantation were included in the study. Any operative or postoperative

complication, those taking topical/oral steroids or NSAIDs, patients requiring postoperative additional medication, ocular or systemic diseases, and patients having history of ocular trauma were excluded. Pre-operative assessment was done. Patients underwent SICS with PCIOL under LA. Post operatively either Diclofenac (group A) or Dexamethasone (group B) was administered to the 2 groups which included 50 patients in each group. Patients were examined post-operatively on days 7, 15, 30. Patients were examined for congestion, corneal edema and anterior chamber flare and cells, intraocular pressure. All parameters were graded (0, 1, 2, 3) according to their severity. Total score was assessed. Grade None (0) Mild (1-3) Moderate (4-7) and Severe (8 & above).

Results

Mean age of the patients was 59.98 ± 9.11 years with age range of 40 – 75 years.

Table 1: Superficial conjunctival congestion

Total score Grades	Day 7		Day 15		Day 30	
	Group A	Group B	Group A	Group B	Group A	Group B
None	29(58%)	4(8%)	49(98%)	33(67%)	50(100%)	50(100%)
Mild	21(42%)	45(90%)	1(2%)	17(32.6%)	0(0%)	0(0%)
Moderate	0(0%)	0(0%)	0(0%)	0(0%)	0(0%)	0(0%)
Severe	0(0%)	1(2%)	0(0%)	0(0%)	0(0%)	0(0%)

p value for day 7 is 0.00001 statistically significant

p value for day 15 is not statistically significant

Table 2: Ciliary congestion

Total score Grades	Day 7		Day 15		Day 30	
	Group A	Group B	Group A	Group B	Group A	Group B
None	49(48%)	28(56%)	50(100%)	47(95.9%)	50(100%)	50(100%)
Mild	1(2%)	22(44%)	0(0%)	3(4%)	0(0%)	0(0%)
Moderate	0(0%)	0(0%)	0(0%)	0(0%)	0(0%)	0(0%)
Severe	0(0%)	0(0%)	0(0%)	0(0%)	0(0%)	0(0%)

For day 7 p value is statistically significant

For day 15 p value is 0.14. not significant

Table 3: Corneal edema

Total score Grades	Day 7		Day 15		Day 30	
	Group A	Group B	Group A	Group B	Group A	Group B
None	46(92%)	44(88%)	47(94%)	48(96%)	50(100%)	50(100%)
Mild	4(8%)	6(12%)	3(6%)	2(4%)	0(0%)	0(0%)
Moderate	0(0%)	0(0%)	0(0%)	0(0%)	0(0%)	0(0%)
Severe	0(0%)	0(0%)	0(0%)	0(0%)	0(0%)	0(0%)

p value for day 7 is 0.5 not statistically significant

p value for day 15 is 0.64 not statistically significant

Table 4: Anterior chamber flare

Total score Grades	Day 7		Day 15		Day 30	
	Group A	Group B	Group A	Group B	Group A	Group B
Absent	45(90%)	11(22%)	49(98%)	41(83.6%)	50(100%)	50(100%)
Mild	3(6%)	36(72%)	1(2%)	8(14.2%)	0(0%)	0(0%)
Moderate	2(4%)	3(6%)	0(0%)	1(2%)	0(0%)	0(0%)
Severe	0(0%)	0(0%)	0(0%)	0(0%)	0(0%)	0(0%)

P value for day 7 is <0.0001 statistically significant

p value for day 15 is 0.007 statistically significant

Table 5: Total score of postoperative inflammatory response

Total score Grades	Day 7		Day 15		Day 30	
	Group A	Group B	Group A	Group B	Group A	Group B
Absent	25(50%)	0(0%)	43(86%)	22(45%)	50(100%)	50(100%)
Mild	24(48%)	30(60%)	7(14%)	27(53%)	0(0%)	0(0%)
Moderate	1(2%)	20(40%)	0(0%)	1(2%)	0(0%)	0(0%)
Severe	0(0%)	0(0%)	0(0%)	0(0%)	0(0%)	0(0%)

p value for day 7 and day 15 are > 0.05 so statistically not significant

Table 6: Intraocular pressure at end of 6 weeks

IOP	Group A	Group B
14 mm of Hg	4(8%)	4(8.3%)
16 mm of Hg	18(36%)	27(56.2%)
18 mm of Hg	21(42%)	17(35.4%)
20 mm of Hg	7(14%)	0(0%)
Total	50(100%)	50(100%)

In This Study

Days 7 & 15: Significant difference was noted with respect to conjunctival and ciliary congestion, with faster response occurring in patients on Dexamethasone

Anterior chamber flare and cells responded much quicker, within 15 days to topical Dexamethasone 0.1%, when compared to Diclofenac 0.1%.

At the end of 30 days, according to total scoring of inflammation both drugs showed equal efficacy.

No significant difference in IOP at baseline and at 6 weeks post operatively between the two groups.

Discussion

It is a routine practice to use steroids after cataract surgery. Steroids interfere with the inflammatory response in a variety of ways, but the steroid treatment can have many side effects such as increased IOP, delayed wound healing and increased chance of post operative ocular infection[5].

Diclofenac indirectly modulates also the lipoxigenase pathway in the arachidonic acid cascade. The potential advantages of using NSAIDs after cataract surgery are the lack of IOP rise and decreased impairment of wound healing. Thus NSAIDs can be used after cataract surgery to control inflammation as shown in study[6].

In a study conducted by Muhammed Wasim and Humayun the anterior chamber cell count was not comparable postoperatively between the two groups on the first day and first week where dexamethasone showed a better anti inflammatory response than the diclofenac group. The anterior chamber cell distribution did not vary significantly between the two groups at 3 weeks or 5 weeks postoperatively [7]

comparable to our study.

In a study between diclofenac sodium and dexamethasone, Reddy *et al.* found that the treatment effects for any of the variables including aqueous cells, flare, ciliary congestion, descemet's folds and intraocular pressure did not show statistical difference three weeks postoperatively[8].

Conclusion

The resolution of inflammation was quicker in the dexamethasone group than in the diclofenac group. But by the end of 30 days the effect on inflammation in both the groups are similar. So Diclofenac sodium 0.1% can be used as an alternative to Dexamethasone phosphate 0.1% in post operative patients following cataract surgery.

References

1. Venkatesh R, Manoranjan D, Prashanth S, Murali Krishnan R. Manual small incision cataract surgery in eyes with white cataracts. *Indian J Ophthalmol.* 2005; 53: 173-176.
2. Saari KM, Nelimarkka L, Ahola V. Comparison of topical 0.7% dexamethasone-cyclodextrin with 0.1% dexamethasone sodium phosphate for postcataract inflammation. *Graefe's Arch Clin Exp Ophthalmol.* 2006; 244: 620-26.
3. Nardi M, Lobo C, Bereczki A. Analgesic and anti-inflammatory effectiveness of nepafenac 0.1% for cataract surgery. *Clin Ophthalmol.* 2007; 1: 527-33.
4. Havener H. Ocular therapeutics. In: Pernan GA, Sandle DR, Goldberg MF, editors. *Principles and practice of ophthalmology.* 19th ed. Philadelphia: W.B. Saunders; 1980; p.757- 68.
5. Khan HA and Amitava AK. Topical diclofenac

- 20 Sharanabasamma M. & Vaibhav K. / To Study the Effect of Topical Diclofenac Sodium 0.1% as An Alternative to Topical Steroid, Dexamethasone Phosphate 0.1% For Post-Operative Control of Inflammation after Small Incision Cataract Surgery versus dexamethasone after strabismus surgery : A double-blind randomized clinical trial of anti-inflammatory effect and ocular hypertensive response. *Indian J Ophthalmology*. 2007; 55: 271-75.
6. Donnenfeld ED, Holland EJ, Stewart RH. Bromfenac ophthalmic solution 0.09% (Xibrom) for postoperative ocular pain and inflammation. *Ophthalmology*. 2007; 114: 1653-6 .
7. Muhammad Waseem , Sadia Humayun. Comparison of anti-inflammatory activity of dexamethasone and diclofenac sodium eye drops in phacoemulsification. *Journal of the College of Physicians and Surgeons Pakistan*. 2009; 19(9): 570-574.
8. Reddy MS, Suneetha N, Thomas RK, Battu RR. Topical diclofenac sodium for treatment of postoperative inflammation in cataract surgery. *Indian J Ophthalmol*. 2000; 48: 223-6.
-

Red Flower Publication Pvt. Ltd.

CAPTURE YOUR MARKET

For advertising in this journal

Please contact:

International print and online display advertising sales

Advertisement Manager

Phone: 91-11-22756995, 22754205, 45796900, Fax: 91-11-22754205
info@rfppl.co.in, redflowerppl@gmail.com

Recruitment and Classified Advertising

Advertisement Manager

Phone: 91-11-22756995, 22754205, 45796900, Fax: 91-11-22754205
info@rfppl.co.in, redflowerppl@gmail.com

Efficacy and Safety of Timolol 0.5% Versus Brimonidine 0.2% in Lowering IOP in Cases of Primary Open Angle Glaucoma

Sharanabasamma M.*, Vaibhav K.**

Authors Affiliation: *Assistant Professor **Post Graduate, Dept of Ophthalmology, Navodaya Medical College, Raichur Karnataka, India.

Abstract

Aims: To compare the efficacy and safety of Timolol maleate 0.5% and Brimonidine 0.2% in lowering IOP in cases of Primary Open Angle Glaucoma. **Settings and Design:** A single center randomized clinical trial was conducted in which the clinical outcome (efficacy) and safety profile of twice daily brimonidine tartarate 0.2% were compared with those of Timolol maleate 0.5% in patients with POAG for one year between November 2013 to October 2014. **Materials and Method:** Fifty patients were enrolled, twenty five in the Brimonidine group and twenty five in the Timolol group. Patients used drugs twice daily for five weeks, and were followed up at baseline visit and at weeks three and five. Clinical success meant reduction of intraocular pressure (IOP), Data about safety and adverse events were analyzed. **Statistical Analysis Used:** Student test. **Results:** Both drugs showed sustained ocular hypotensive efficacy in the study period of one year. At baseline the mean IOP was 24.34 ± 2.82 mm Hg in the timolol group and 24.16 ± 2.76 mm Hg in the brimonidine group. The IOP readings after treatment at 3rd and 5th week were significantly lower in both groups ($P < 0.001$) with no significant statistical difference between the two groups. 20% of the patients in Timolol group and 8% of patients in Brimonidine group, reported mild adverse events. **Conclusions:** Both the drugs have same efficacy and safety profile.

Keywords: Brimonidine; Glaucoma; Timolol.

Introduction

Glaucoma is second only to cataract as a cause of blindness worldwide^[1]. It affects about 50 million and blinds 8 million people worldwide. These dismal figures are despite the fact that in the case of open angle glaucoma, early treatment can prevent progression of the disease. Primary open angle glaucoma is a symptom complex characterized by raised Intraocular pressure (IOP), increased cupping and visual field defects. It is called "creeping thief of the sight" because the disease remains symptomless and majority of the patients are being diagnosed only on routine examination and most of the time very late. Elevated IOP is a major risk factor that contributes to the optic nerve damage directly due to pressure effect and indirectly by reducing the blood

supply to the optic nerve head (ischemia of the optic nerve head) and subsequent visual field loss in patients with primary open angle glaucoma. The disease progression can be halted by adequately lowering the IOP. The three modalities of treatment are medical, laser and surgical. Medical line of treatment to reduce intraocular pressure appears to be the first choice of treatment. Timolol, a topical non selective β -blocker which reduces the IOP by decreasing the aqueous humor secretion, Brimonidine a topical alpha 2 agonist is also used. In this study the efficacy and safety of these drugs are evaluated.

Reprint Request: Sharanabasamma M.,
Sri Nursing & Maternity Home, Near Ek Minar,
Beharoon quilla, Raichur Ho, Raichur - 584101, karnataka.
E-mail: dr.msharanabasamma@gmail.com

Subjects and Methods

The present randomized double blind controlled study was carried out to compare the efficacy between Brimonidine 0.2% twice daily and Timolol 0.5% twice daily in the reduction of Intraocular pressure, in patients suffering from open angle glaucoma.

Study Period: 1 year

Sample Size: 50 patients of POAG.

Ethical Clearance: Obtained from the institutional ethical committee board.

Inclusion Criteria

Patients with open angle glaucoma were subjected to this study protocol.

Exclusion Criteria

1. Patients with angle closure glaucoma.
2. Patients with congenital glaucoma.
3. Patients with secondary glaucoma

Evaluation of all the patients included detailed history collection followed by systemic and ocular examination.

- Determination of visual acuity was done by Snellen's chart and near vision chart.
- External ocular examination was done.
- Detailed torch light examination was done including pupillary reflex and anterior chamber depth.

Detailed Slit Lamp Examination for Assessing

1. Depth of peripheral anterior chamber by comparing it with peripheral corneal thickness.
 2. Pupillary reaction in both the eyes.
 3. Presence of posterior synechiae.
- Gonioscopy was performed by using Goldman three mirror lens.

Grading of angle width was done according to Shaffer's grading.

- Intraocular pressure measurement was done with Schiotz tonometer and Perkins applanation tonometer at morning 9 a.m, afternoon 1 p.m and evening 5 p.m.
- The mean diurnal IOP was defined as the mean of the measurements at 9 a.m., 1 p.m. and 5 p.m.
- Visual field evaluation was done by using

Humphrey field analyser.

- The pupils were then dilated with a combination of 10% phenylephrine and tropicamide 0.8% drops were instilled every 5 min over a 15 min interval.

This was followed by detailed examination by fundoscopy and 90 D lens examination on slit lamp.

- Measurement of blood pressure was done.
- Other investigations included, Urine examination for detection of sugar and albumin.

Follow-up

Patients were followed up at 3rd week, 5th week and following assessment was done.

- Visual acuity.
- IOP at 9 am, 1 pm and 5 pm.
- Any side effects of drugs.
- The levels of significance (p value) was calculated by student's 't' test.

Outcome was Defined as Follows

Complete success

I.O.P. \leq 15 mm Hg with any group.

Partial success

I.O.P. \leq 21 mm Hg with any group.

Complete success

I.O.P. \geq 21 mm Hg with any group.

Hypotony was defined as I.O.P. $<$ 6 mm Hg.

Because all patients were treated bilaterally, the mean IOP from both the eyes were used as an experimental unit in the analysis. The change from the baseline was calculated separately for each eye and then the changes from both the eyes were averaged. A p value less than or equal to 0.05 was considered statistically significant for the treatment effects.

Results

Out of 50 patients 27 patients (54%) belonged to the 41-60 year age group. 14 patients (56%) of these belonged to group I and 13 patients (52%) to group II. 19 patients were above 60 years (38%). 10 patients

(40%) belonged to group I and 9 patients (36%) to group II. 4 patients were between 20-40 years (8%), out of which 1 patient (4%) belonged to group I and 3 patients (12%) to group II.

30 patients (60%) were male and 20 (40%) were female. In group I, 16 (64%) were male and 9 (26%) were female. In group II, 14 (56%) were male and 11 (44%) were female.

52% patients had sluggishly reacting pupils, 6% patients had non reacting pupils.

The maximum number of 19 patients (38%) had best spectacle corrected visual acuity of $\leq 6/60$, 12 patients (48%) of these belonged to group I and 7 (28%) to group II. 16 patients (44%) had best spectacle corrected visual acuity between 6/6 - 6/12, 5 (20%)

of these belonged to group I and 11 (44%) to group II. BCVA between 6/18 - 6/36 with 15 patients (30%), out of which 8 (32%) belonged to group I and 7 (28%) to group II. [Table 1]

Mean diurnal baseline IOP of 34 patients (68%) was between 21 - 25 mm Hg, of 15 patients (30%) was between 26 - 30 mm Hg and 1 patient (2%) had mean diurnal baseline IOP between 31 - 35 mm Hg. In group I, mean diurnal baseline IOP of 16 patients (64%) was between 21 - 25 mm Hg, of 8 patients (32%) was between 26-30 mm Hg, and of 1 patient (4%) was between 31 - 35 mm Hg. In group II, mean diurnal baseline IOP of 18 patients (72%) was between 21 - 25 mm Hg and 7 patients (28%) was between 26 - 30 mm Hg. (Table 2)

Table 1: Best corrected visual acuity

Visual Acuity	Group I		Group II		Total	
	No.	%	No.	%	No.	%
$\leq 6/60$	12	48%	7	28%	19	38%
6/36 - 6/18	08	32%	7	28%	15	30%
6/12 - 6/6	05	20%	11	44%	16	32%
Total	25	100%	25	100%	50	100%

Table 2: Baseline IOP

Baseline IOP (mm Hg)	Group I		Group II		Total	
	No.	%	No.	%	No.	%
21 - 25	16	64%	18	72%	34	68%
26 - 30	08	32%	07	28%	15	30%
31 - 35	01	04%	00	00%	01	02%
Total	25	100%	25	100%	50	100%

Table 3: Cup disc Ratio

C : D ratio	Group I		Group II		Total	
	No.	%	No.	%	No.	%
0.3 - 0.5	05	20%	08	32%	13	26%
0.6 - 0.8	18	72%	14	56%	32	64%
0.9	02	08%	03	12%	05	10%
Total	25	100%	25	100%	50	100%

Table 4: Field defects

Field constriction	Group I		Group II		Total	
	No.	%	No.	%	No.	%
Normal	01	04%	05	20%	06	12%
Early field defects	02	08%	04	16%	06	12%
Arcuate scotoma	12	48%	08	32%	20	40%
Biaruate scotoma and residual fields	10	40%	08	32%	18	36%
Total	25	100%	25	100%	50	100%

32 patients (64%) had Cup-Disc ratio between 0.6 to 0.8. 18 patients (72%) of these belonged to group I and 14 (56%) to group II. 13 patients (26%) had Cup-Disc ratio between 0.3 - 0.5. 5 patients (20%) of these belonged to group I and 8 (32%) to group II. Five patients (10%) had Cup-Disc ratio of 0.9. Two patients (8%) of these belonged to group I and 3 patients (12%)

to group II. (Table 3)

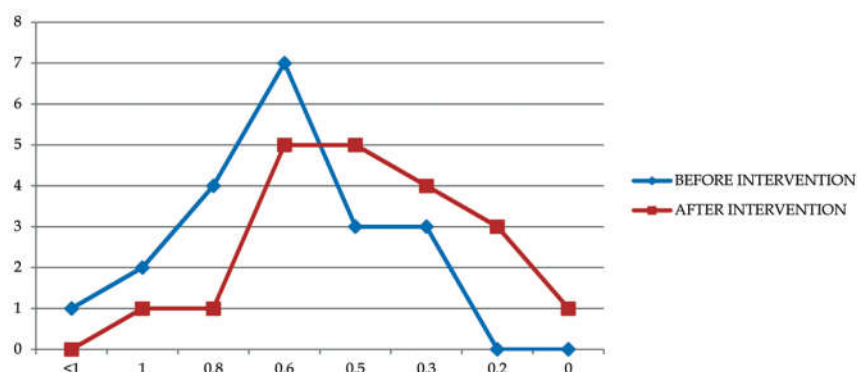
12% patients had early visual field defects, 40% patients had arcuate scotoma and 36% patients had severe damage with extensive visual field loss to small residual field. (Table 4)

The mean diurnal baseline IOP was 24.34 ± 2.82

mmHg. The mean diurnal IOPs at 3rd and 5th week were 18.64 ± 3.87 and 17.67 ± 3.73 respectively. The mean reduction in IOP from baseline to 3rd week was 5.70 ± 1.88 and mean % reduction was $23.93\% \pm 9.06\%$. Similarly, the mean reduction in IOP from baseline to 5th week was 6.67 ± 2.15 and mean % reduction was $27.77\% \pm 9.50\%$. In group II, the mean diurnal baseline IOP was 24.16 ± 2.76 , the mean

diurnal baseline IOPs at 3rd and 5th week were 18.39 ± 3.52 and 16.92 ± 3.47 respectively.

The mean reduction in IOP from baseline to 3rd week was 5.76 ± 1.65 and mean % reduction was $24.28\% \pm 7.99$. Similarly, the mean reduction in IOP from baseline to 5th week was 7.23 ± 2.33 and mean % reduction was $30.21\% \pm 9.71\%$. (Graph 1)



Graph 1: Mean iop and mean reduction in IOP

5 patients had adverse events, of which 3 had burning / stinging in the eyes and 2 had conjunctival hyperemia. In group II, 2 patients had foreign body sensation in the eyes. A total of 7 patients (14%) had adverse events of which 5 patients (20%) were in group I and 2 (8%) were in group II.

23 patients (46%) had complete success, 9 (36%) of these belonged to group I and 14 (56%) to group II. 18 patients (36%) had partial success. 11 (44%) of these belonged to group I and 7 (28%) to group II. 9 patients (18%) had complete failure, of which 5 patients (20%) belonged to group I and 4 patients (16%) belonged to group II.

Discussion

In a study to measure the 4 years risk of open angle glaucoma found that, incidence rate of primary open angle glaucoma increased from 1.2% at age 40 – 49 years to 4.2% at age of 70 or more [2]. Another study noted that one of the factors that predict the onset of primary open angle glaucoma is older age [3]. In the present study, 8% patients were below 40 years of age, POAG is by no means limited to those over 40 years.

Our study suggests higher prevalence among Men.

Our study shows majority of patients had poor visual acuity, which determines glaucoma as one of the leading causes of blindness. A study done at

Arvind Eye Hospital, Madurai included 5150 patients to determine the prevalence of blindness and vision impairment in a rural population of Southern India. Visual impairment was defined as best corrected visual acuity $< 6/18$, and blindness was defined using both Indian ($< 6/60$) and World Health Organization ($< 3/60$) definition [4]. Authors concluded that 4.3% patients had visual acuity $< 3/36$ and 11.4% patients had visual acuity $< 6/60$ [5].

Pupillary reaction is an important factor in diagnosing primary open angle glaucoma. Pupillary dynamics in 13 patients with primary open angle glaucoma was evaluated. Out of 13 patients, abnormal light reflex was detected in all eyes of 6 patients, afferent papillary defect pattern was detected in 13 eyes and only one patient was found to be normal [6].

Khadikova E. V. (1997), in their study of papillary reactions in normal subjects aged over 40 and in patients with POAG, found that in POAG the changes in the papillary reaction are more when compared to normal subjects which is due to dystrophy of the iris and ciliary body.

Majority of the patients (68%) had mean diurnal baseline intraocular pressure between 21 to 25 mm Hg and 30% had between 26 to 30 mm Hg. Several studies have shown an incidence of new onset glaucomatous damage in previously unaffected patients, was about 2.6%- 3% for IOP 21 to 25 mm Hg, 12 to 26% incidence for IOP 26 to 30 mm Hg and approximately 42% for those higher than 30mm Hg.

Thus, chances of glaucoma damage increases with increase in IOP in accordance to the study conducted at which was studied relationship between Intraocular pressure and primary open angle glaucoma in 5308 patients and found that the risk of glaucomatous damage increases with the height of the IOP, particularly at levels of 21 to 29 and 30 mm Hg and above [7].

74% patients had Cup-Disc ratio above 0.6. Increased Cup-Disc ratio is one of the risk factors for the development of glaucomatous visual field loss. This is in accordance with a study showing eyes with the combination of IOP consistently above 20mm Hg and Cup-Disc ratio of 0.5 or more, were at higher risk of developing glaucomatous damage [8].

In this study, there is no much difference of mean diurnal baseline IOP between the two groups. ($P > 0.8$). In group I, at the end of 3rd week follow-up the mean diurnal IOP was 18.64 ± 3.87 mm Hg, thus effecting a fall of 5.7 ± 1.88 mm Hg (which is 23.93% of the initial levels). In group II, at the end of 3rd week follow-up, the mean diurnal IOP was 18.39 ± 3.52 mm Hg, thus effecting a fall of 5.76 ± 1.65 mm Hg which is 24.28% of the initial levels.

The intraocular pressure lowering was similar in both Timolol and Brimonidine groups. At baseline, the mean IOP was 24.34 in Timolol group and 24.16 in Brimonidine group showing no statistically significant difference. ($P > 0.8$).

The IOP readings after treatment were significantly lower than baseline in both groups. The application of paired 't' test showed that the mean reduction in diurnal IOP at 3rd and 5th week of group II was significant ($P < 0.001$).

The majority of patients in both treatment groups achieved clinical success with their 5 week treatment regimen. The clinical success rate was 80% in Timolol group and 84% in Brimonidine group. There was no statistically significant difference in both groups.

In a study to evaluate the efficacy of Brimonidine and Timolol for glaucoma it was found that with mean baseline IOP was 24.48 ± 2.29 mm Hg with Brimonidine and 23.32 ± 0.82 mm Hg with Timolol group, significantly lower IOP readings were noted when baseline values were compared to values at all visits (weeks 2 and 4).

In a study of 483 patients, Brimonidine produced significantly greater mean decreases of IOP ($P \leq 0.007$), when compared to Timolol at all follow up visits (12 month study)[9].

A reduction of IOP of 7.7 mm Hg with Timolol and

6.9 mm Hg with Brimonidine, was seen which intended hence showing almost similar clinical effectiveness in reducing the intraocular pressure.

A study of 30 patients revealed that, within group differences, reduction of IOP was significant, but the mean reduction of IOP when brimonidine (19.8 ± 3.1) and Timolol (17.7 ± 2.9) were compared was statistically not significant [10].

Another animal study on rats showed a very significant reduction of retinal ganglion cell loss with Brimonidine when compared to Timolol, thus indicating the neuroprotectiveness of brimonidine [11].

7 patients had reported mild adverse events. An extensive study reported that 17% patients of Brimonidine group and 9% patients of Timolol group had have mild adverse events 10% patients of Brimonidine group had ocular allergy [12].

An overall similar incidence of adverse events in both treatment groups, with no serious adverse event in either of the groups has been reported. Significantly more ocular burning and stinging was reported in Timolol group (43.6%) ($P < 0.001$)[13].

Patients receiving Timolol had significantly ($P < 0.04$) lower heart rates than did patients receiving Brimonidine.

A compilation of review of more than 3,000 reports of adverse events was attributed to topical Timolol maleate, which included 267(55%) patients experiencing cardiac arrhythmia or a bronchospasm related event^[14].

We had complete success in 9 patients (36%) in group I and in 14 patients (56%) in group II. 11 patients (44%) in group I and 7 patients (28%) in group II had partial success.

Five patients (20%) with complete failure were in group I and four patients (16%) were in group II.

30% reduction or more in mean diurnal IOP was achieved by 71% of patients in Brimonidine group and by 64% of patients in Timolol group.

Another study found after 6 weeks of treatment that the diurnal IOP measured for Timolol maleate (17.7 ± 2.7 mm Hg) and Brimonidine tartarate (19.0 ± 2.4 mm Hg) had a statistical difference between the two groups [15].

In a study of 40 patients for a period of one year showed clinical success rate of 81.8% was seen in the Timolol group and 86.2% in the Brimonidine group making no statistically significant difference between them ($P = 0.817$) [16].

Conclusion

Old age, male gender, high intraocular pressure, increased cup-disc ratio are high risk factors for the development of primary open angle glaucoma.

Abnormal pupillary reaction is a good predictor for this disease. Systemic diseases like hypertension and diabetes are predisposing factors for primary open angle glaucoma. As the disease remains symptomless for long and majority of the patients are being diagnosed only on routine examination, it is recommended to perform applanation tonometry in all individuals above 40 years of age, as a preliminary screening method.

This study indicates that in a small population, both Timolol maleate 0.5% and Brimonidine tartrate 0.2% eye drops are equally effective in lowering intraocular pressure and also showed sustained ocular hypotensive efficacy in the study period. However, the clinical success rate showed no significant statistical difference.

The treatment related adverse events were all ocular and none were severe in intensity. Both these drugs had a safe prescribing profile but Brimonidine has an added advantage of providing neuroprotection to ganglion cells; and Timolol has a guarded usage in patients with co-morbid respiratory and cardiovascular conditions.

References

1. Resnikoff S, Pascolini D, Etya'ale D et al. Global data on visual impairment in the year 2002. *Bull World Health Organ.* 2004; 82 : 844-851.
2. Leske MC, Connell AMS, Wu SY, Nemesure B, Li X, Schachat A et al. Incidence of open - angle glaucoma. *Arch Ophthalmol.* 2001 ; 119: 89-95.
3. Gordon MO, Beiser JA, Brandt JD, Heuer DK, Higginbotham EJ, Johnson CA et al. The ocular hypertension treatment study group. The Ocular Hypertension Treatment Study: Baseline Factors That Predict the Onset of Primary Open - Angle Glaucoma. *Arch Ophthalmol.* 2002 ; 120: 714-20.
4. Thulasiraj RD, Nirmalan PK, Ramakrishnan R, Krishnadas R, Manimekalai TK, Baburajan NP et al. Blindness and vision impairment in a rural south Indian population: the Arvind Comprehensive Eye Survey. *Ophthalmology.* 2003; 110: 1491-8.
5. Duke Elder. *System of ophthalmology.* Vol. XI Disease of the lens and vitreous: glaucoma and hypotony London: Henry Kimpton; 1976.
6. Hashimoto - Takahashi E. Pupillary dynamics in patients with primary open - angle glaucoma. *Bull Osaka Med Coll.* 1990; 36(1-2): 71-7.
7. Sommer A, Tielsch JM, Katz J, Quigley HA, Gottsch JD, Javitt J et al. Relationship between intraocular pressure and primary open glaucoma among white and black Americans. The Baltimore Eye Survey. *Arch Ophthalmol.* 1991; 109: 1090-5.
8. Hart WM, Yablonski M, Kass MA, Becker B. Multivariate analysis of the risk of glaucomatous visual field loss. *Arch Ophthalmol.* 1979; 97(8): 1455-8.
9. LeBlanc RP. Twelve-month results of an ongoing randomized trial comparing Brimonidine Tartrate 0.2% and Timolol 0.5% given twice daily in patients with glaucoma or ocular hypertension. Brimonidine Study Group 2. *Ophthalmology.* 1998; 105(10): 1960-7.
10. Konstas AGP, Stewart WC, Topouzis F, Tersis I, Holmes KT, Stangos NT. Brimonidine 0.2% given two or three times daily versus Timolol Maleate 0.5% in Primary Open Angle Glaucoma. *Am J Ophthalmol.* 2001; 131: 729-33.
11. WoldeMussie E, Ruiz G, Wijono M, Wheeler LA. Neuroprotection of Retinal Ganglion Cells by Brimonidine in Rats with Laser-induced chronic ocular hypertension. *Invest Ophthalmol Vis Sci.* 2001; 42(12): 2849-55.
12. Chen MJ, Chou JC, Hsu WM, Liu JH. The efficacy and safety of Brimonidine 0.2% compared with Timolol 0.5% in Glaucoma: A randomised clinical trial on Taiwanese patients. *J Chin Med Assoc.* 2003; 66: 276-81.
13. Katz LJ. Brimonidine tartrate 0.2% twice daily v/s timolol 0.5% twice daily: 1- year results in glaucoma patients. *Am J Ophthalmol.* 1999; 127: 20-6.
14. Carlsson AM, Chaucham BC. The effect of brimonidine tartrate on blood flow in patients with ocular hypertension. *Am J Ophthalmol.* 2000; 129: 297-301.
15. Waldock A, Snape J. Effects of glaucoma medications on the cardiorespiratory and intraocular pressure status on newly diagnosed glaucoma patients. *Br J Ophthalmol.* 2000; 84: 710-3.
16. Nelson WL, Fraunfelder FT, Sills JM, Arrowsmith JB, Kuritsky JN. Adverse respiratory and cardiovascular events attributed to timolol ophthalmic solution, 1978-1985. *Am J Ophthalmol.* 1986; 102(5): 606-11.

An Atypical Case of Sympathetic Ophthalmia Following Zone 1 Corneal Injury

Praveen Subudhi*, B.N.R. Subudhi**

Authors Affiliation: *Consultant, Ruby Eye Hospital and Research Center, Govinda Vihar, Berhampur, Odisha. **Professor, M.K.C.G Medical College, Berhampur, Odisha, India.

Abstract

Purpose: To report a case of atypical sympathetic ophthalmia following limbal corneal laceration. *Methods and Results:* An eleven year old child had a successful left eye (OS) corneal laceration repair at the temporal limbus with excision of exposed non necrotic iris tissue, resulting in good visual acuity of 20/80 and 20/25 postoperative day 1 and 7 respectively. The patient was prescribed 1mg/kg oral prednisolone in a tapering dose as prophylaxis. Post operative day 21, patient presented with acute onset decreased vision in both eyes. Visual acuity was counting fingers 3 feet in both eyes. On examination, anterior segment examination was quiet without any inflammation, anterior vitreous face showed 1+ cells and dilated funduscopy revealed bilateral symmetrical serous retinal detachments along the posterior pole. Optical coherence tomography (OCT) demonstrated separation and elevation of inner neurosensory layers from the outer segment marking presence of hyperreflective material along with subretinal fluid between detached surfaces. There was stippled hyperfluorescence along the posterior pole as seen in fluorescein angiography. With a diagnosis of sympathetic ophthalmia confirmed, oral prednisolone (2 mg/kg body weight) was instituted following which, there was gradual decrease in macular elevation with corresponding improvement in visual acuity with no recurrence for last 6 months. *Conclusion:* To our knowledge, this is the first reported instance of an atypical presentation of sympathetic ophthalmia and antecedent corticosteroid therapy would have mitigated robust anterior segment findings usually associated with the condition.

Keywords: Corneal Laceration; OCT; Open Globe Injury; Exudative Macular Elevation; Uveal Prolapse.

Introduction

Sympathetic ophthalmia is a rare phenomenon with an incidence of 0.03 per 100,000 per year [1]. Penetrating injuries involving uveal tissue and retinal surgeries are common causes [2-4]. Plaque brachytherapy [5], fungal keratitis [6] and cyclodestructive procedures [7] have been reported to be rarely associated. There is a delayed hypersensitivity reaction to sequestered uveal antigen leading to the damage of outer RPE layer of retina [8,9]. Sympathetic ophthalmia has biphasic peaks in children and the elderly because of greater incidence of accidental trauma and ocular surgery respectively.¹⁰ Hereby we present a case of accidental corneal injury

that developed sympathetic ophthalmia in spite of prophylactic systemic steroid therapy. The efficacy of optical coherence tomography (OCT) in following the course of the disease and correlating visual recovery with that of anatomic normalcy is also reported [11].

Case Presentation

An 11 year old male child presented with complaints of pain and decreased vision in left eye

Reprint Request: Praveen Subudhi,

Ruby Eye Hospital and Research center, Govinda Vihar,
Berhampur-760001, Ganjam, Odisha India.
E-mail: subudhipraveen@gmail.com,

for 3 days following penetrating pencil injury. His visual acuity in right eye was 20/20 and in left eye was 20/120. Examination of the left eye revealed full thickness corneal laceration at the temporal limbus with iris prolapse, clear lens and normally appearing fundus. Corneal laceration repair was performed followed by excision of exposed normal appearing iris tissue and apposition of corneal margins (Figure 1). The patient was treated with oral prednisolone 1 mg/kg body weight, a plan to gradually taper the dose over 6 weeks. Visual acuity rapidly improved from 20/80 on Post operative day 1 to 20/25 on Post operative day 7.

On postoperative day 21, patient presented with sudden onset, rapidly progressive visual loss in both eyes (OU) over last 2 days. His visual acuity was counting fingers at 3 feet in OU. He was still on oral prednisolone therapy with a dose of 10 mg/day. Dilated fundus examination showed clear optical media with bilateral gross serous elevation of macula (Figure 2a & b) and occasional cells in anterior vitreous face. Optical coherence tomography (Stratus OCT, Carl ZEISS Meditech, Dublin, CA) revealed separation of inner neurosensory layer from outer hyper-reflective area (RPE layer) with accumulation of subretinal fluid along with exudation but there was no evidence of cystoid spaces in inner neurosensory layer (Figure 3a & b). Fundus fluorescein angiogram (figure 2c & d) demonstrated stippled hyperfluorescence in the posterior pole. Analyzing above features a diagnosis of sympathetic ophthalmia was made, however it was quite atypical owing to absence of keratic precipitates and anterior chamber reaction and

posterior synechiae. The patient was prescribed higher dose of oral prednisolone (2mg/kg body weight), which was tapered by 10 mg every 10 days and terminated at 12 weeks. On day 3 of increased steroid usage, OCT revealed reduction of macular elevation in both eyes with corresponding improvement in visual acuity (20/200 in OU) (Figure 3c & d). On 15 days of increased steroid usage, his visual acuity was 20/20 in OU and there was complete resolution of macular elevation with restoration of normal foveal contour. (Figure 3e & f). Subsequent follow up for 6 months vision of the patient was well preserved and there was no evidence of recurrence of clinical signs of sympathetic ophthalmia.



Fig. 1:

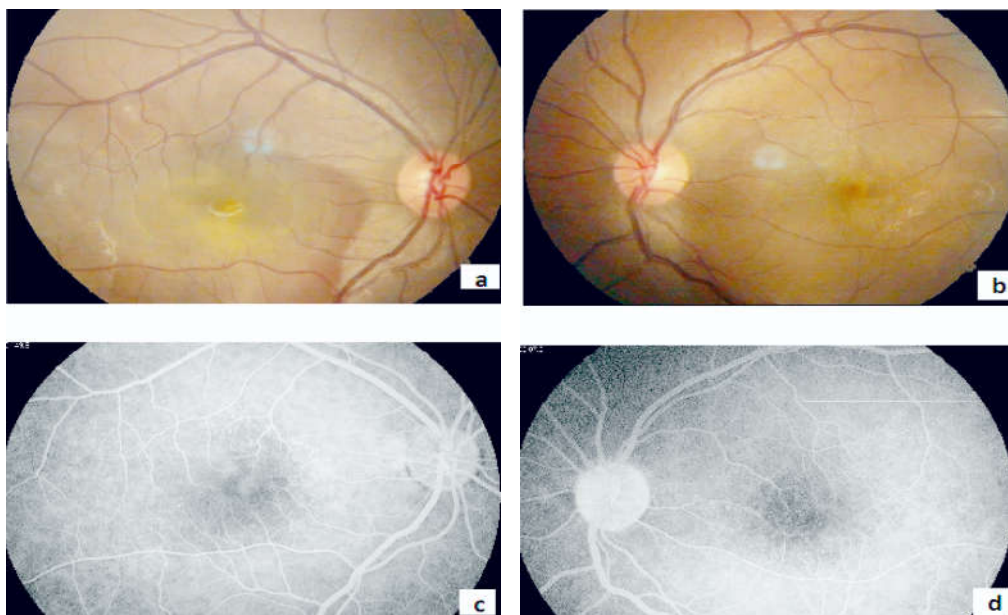


Fig. 1: A&B Colour fundus photographs exhibiting serous macular elevation. C&D Fundus fluorescein angiogram (FFA) photographs in peak arteriovenous filling exhibiting stippled hyperfluorescence in the posterior pole

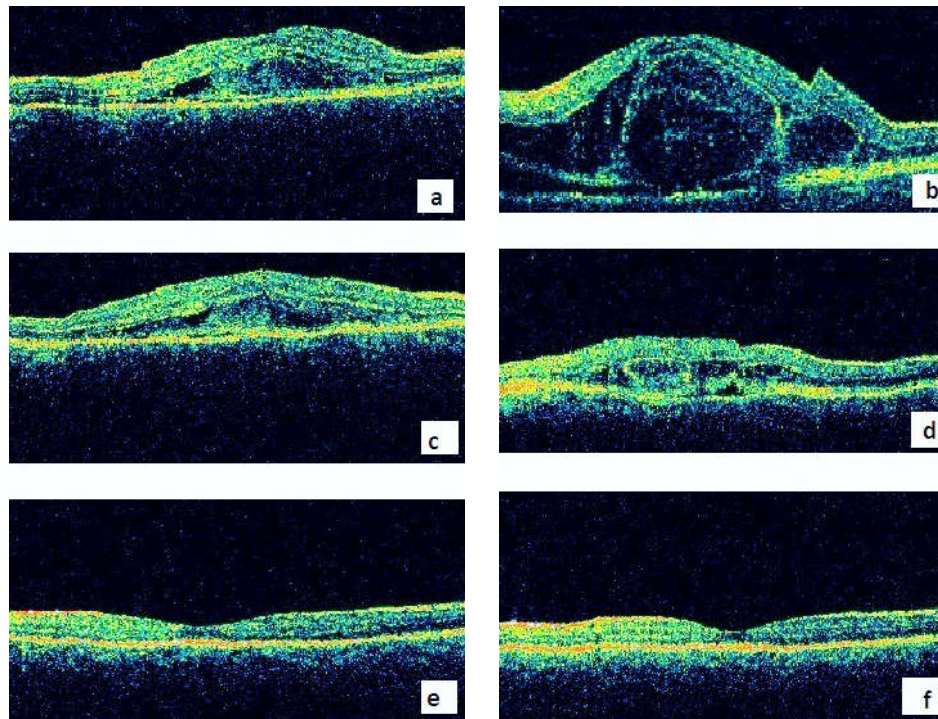


Fig. 3: A&B: Optical coherence tomography both eyes exhibiting exudative retinal detachment, C&D: showing gradual reduction of macular elevation, E&F: showing complete resolution of serous macular elevation with attainment of normal contour with 15 days of high corticosteroid therapy

Discussion

The diagnosis of sympathetic ophthalmia is based on clinical examination and evaluation of history [12,13]. However ocular investigations like fundus fluorescein angiogram and optical coherence tomography are useful adjuncts in establishing the diagnosis [14,15]. It classically manifest as bilateral granulomatous pan-uveitis with a definitive history of penetrating trauma and rarely by blunt trauma [16]. Posterior segment shows moderate to dense vitritis, choroiditis and papillitis with multiple exudative retinal detachments [18,19]. Onset of disease is within 1 year in 90% of cases and 17% present within 1 month [17,18]. Our case presented on 28th day of traumatic repair and 30th day of trauma. None of the anterior segment findings could be elucidated in our patient possibly attributed to prior steroid therapy. Kumar et al [20] showed 30% of isolated posterior segment findings in their case series on sympathetic ophthalmia. Gupta et al [21] demonstrated that 22 of their 40 cases presented with exudative retinal detachment with no evidence of anterior segment inflammation, leading to the conclusion that lone posterior segment findings may be an indicative of early diagnosis where anterior segment has not yet involved or it is an atypical presentation. Our case presented with lone posterior segment findings which is very consistent with 2 of

the previous case series [20,21]. Isolated posterior segment findings could be explained by prior immunosuppression in the immediate post operative period.

OCT is a useful noninvasive tool in the diagnosis and determining efficacy of treatment in sympathetic ophthalmia [22,23]. OCT demonstrates exudative retinal detachments and its reduction marks the response to treatment. Our patient too had gradual reduction in exudative retinal separation in OCT following steroid therapy. Sympathetic ophthalmia is treated with immunosuppressive therapy. Because of high risk of recurrence, patients needs timely follow up. Recurrence calls for institution of other immunosuppressive therapy such as chlorambucil and azathioprine [24]. In our case there was complete resolution of exudative retinal detachment with high dose steroids which was maintained for 6 months and showed no signs of recurrence undermining the need of immunosuppressants.

Conclusion

Sympathetic ophthalmia is a rare phenomenon can still occur despite attempted prophylaxis with corticosteroid therapy and that OCT findings parallel clinical improvement. The present case is reported owing to its rarity and unusual presentation.

References

1. Kilmartin DJ, Dick AD, Forrester JV. Prospective surveillance of sympathetic ophthalmia in the United Kingdom and Republic of Ireland. *Br J Ophthalmol*. 2000; 84: 259-263.
2. Towler HMA, Lightman S. Sympathetic ophthalmia. *Int Ophthalmol Clin*. 1995; 35: 31-42.
3. Rao NA, Forster DJ, Spalton DJ. Sympathetic ophthalmia. In: Podos SM, Yanoff M (eds). *The Uvea Uveitis and Intraocular Neoplasms*, Vol. 2, Chapter 8. Mosby-Wolfe: USA. 1995; pp 8.10-8.13.
4. Nussenblatt RB. Sympathetic Ophthalmia. In: Nussenblatt RB, Whitcup SM (eds). *Uveitis Fundamentals and Clinical Practice*, 3rd ed., Chapter 22. Elsevier: USA. 2004; pp 311-323.
5. Ahmad N, Soong TK, Salvi S, Rudle PA, Rennie IG. Sympathetic ophthalmia after ruthenium plaque brachytherapy. *Br J Ophthalmol*. 2007; 91: 399-401.
6. Buller AJ, Doris JP, Bonshek R, Brahma AK, Jones NP. Sympathetic ophthalmia following severe fungal keratitis. *Eye*. 2006; 20: 1306-1307.
7. Jonas JB, Back W, Sauder G, Junemann U, Harder B, Spandau UH. Sympathetic ophthalmia in vater association combined with persisting hyperplastic primary vitreous after cyclodestructive procedure. *Eur J Ophthalmol*. 2006; 16: 171-172.
8. Chan CC, Benezra D, Rodrigues MM, Palestine AG, Hsu SM, Murphree AL, Nussenblatt RB. Immunohistochemistry and electron microscopy of choroidal infiltrates and Dalen-Fuchs nodules in sympathetic ophthalmia. *Ophthalmology*. 1985; 92: 580-90.
9. Jakobiec FA, Marboe CC, Knowles DM 2nd, Iwamoto T, Harrison W, Chang S, Coleman DJ. Human sympathetic ophthalmia. An analysis of the inflammatory infiltrate by hybridoma-monoclonal antibodies, immunochemistry, and correlative electron microscopy. *Ophthalmology*. 1983; 90: 76-95.
10. Albert DM, Diaz-Rohena R. A historical review of sympathetic ophthalmia and its epidemiology. *Surv Ophthalmol*. Jul-Aug 1989; 34(1): 1-14.
11. Chan RV, Seiff BD, Lincoff HA, Coleman DJ. Rapid recovery of sympathetic ophthalmia with treatment augmented by intravitreal steroids. *Retina*. 2006 Feb; 26(2): 243-7.
12. Lubin JR, Albert DM, Weinstein M. Sixty-five years of sympathetic ophthalmia. A clinicopathologic review of 105 cases (1913-19788). *Ophthalmology*. Feb 1980; 87(2): 109-121.
13. Damico FM, Kiss S, Young LH. Sympathetic ophthalmia. *Semin Ophthalmol*. 2005; 20: 191-197.
14. David Fleischman, Emil Anthony T. Say, John D. Wright, and Maurice B. Landers Multimodality Diagnostic Imaging in a Case of Sympathetic Ophthalmia. August 2012; 20(4): 300-302.
15. Castiblanco, Claudia, Adelman, Ron A. Imaging for Sympathetic Ophthalmia: Impact on the Diagnosis and Management International Ophthalmology Clinics Issue: 2012 Fall; 52(4): 173-181.
16. Castiblanco CP, Adelman RA. Sympathetic ophthalmia. *Graefes Arch Clin Exp Ophthalmol*. 2009 Mar; 247(3): 289-302. doi: 10.1007/s00417-008-0939-8. Epub 2008 Sep 16.
17. Goto H, Rao NA. Sympathetic ophthalmia and Vogt-Koyanagi-Harada syndrome. *Int Ophthalmol Clin*. 1990 Fall; 30(4): 279-85.
18. Xi K Chu and Chi-Chao Chan Sympathetic ophthalmia: to the twenty-first century and beyond *J Ophthalmic Inflamm Infect*. 2013; 3: 49.
19. Arevalo JF, Garcia RA, Al-Dhibi HA, Sanchez JG, Suarez-Tata L. Update on sympathetic ophthalmia. *Middle East Afr J Ophthalmol*. 2012 Jan; 19(1): 13-21.
20. Kumar K, Mathai A, Murthy SI, Jalali S, Sangwan V, Reddy Pappuru R, Pathangay A. Sympathetic ophthalmia in pediatric age group: clinical features and challenges in management in a tertiary center in southern India. *Ocul Immunol Inflamm*. 2014 Oct; 22(5): 367-72.
21. Gupta V, Gupta A, Dogra MR. Posterior sympathetic ophthalmia: a single centre long-term study of 40 patients from North India. *Eye (Lond)*. 2008 Dec; 22(12): 1459-64.
22. Puliafito C. Acute sympathetic ophthalmia. In: Schuman, Puliafito, Fujimoto, eds. *Optical Coherence Tomography of Ocular Diseases*. 2nd ed. New York: Slack; 2003: 386-393
23. Gupta V, Gupta A, Dogra MR, Singh I. Reversible retinal changes in the acute stage of sympathetic ophthalmia seen on spectral domain optical coherence tomography. *Int Ophthalmol*. 2011 Apr; 31(2): 105-10.
24. Maruyama Y, Kishi S. Tomographic features of serous retinal detachment in Vogt-Koyanagi-Harada syndrome. *Ophthalmic Surg Lasers Imaging* 2004; 35: 239-242.

Cystoid Macular Oedema Due to Cancer Associate Retinopathy: A Rare Presentation and Its Response to Intravitreal Bevacizumab Injection

Gupta Sanjiv K.*, Kumar Ajai**, Sharma Arun***, Katiyar Vishal****, Agrawal Siddharth****

Authors Affiliation: *Professor, **Associate Professor, ***Assistant Professor, Department of Ophthalmology, King George's Medical University, Chowk, Lucknow, Uttar Pradesh 226003 U.P., India. **Ophthalmologist, Jan Kalyan Eye Hospital, Lucknow, Uttar Pradesh 226016.

Abstract

We here present a cancer associated retinopathy with gradually progressive moderate diminution of vision in a patient of gall bladder carcinoma. There was bilaterally symmetrical cystoids macular oedema (CME) and was treated with intravitreal Bevacizumab injection with partial improvement in the vision and CME.

Keywords: Cancer Associated Retinopathy; Gall Bladder Carcinoma; Cystoids Macular Oedema; Intravitreal Injection; Bevacizumab; Anti-VEGF.

Introduction

Cancer associated retinopathy (CAR) is the most prevalent paraneoplastic retinopathy of the spectrum of diseases called autoimmune retinopathy. Still being a rare disease approximately only 100 cases of cancer-associated retinopathy have been reported in the literature [1].

The case reported here by us is a CAR patient with gall bladder carcinoma with local metastasis as the primary contributory disease. This association has not yet been reported in literature and another unusual presentation was of Cystoid Macular Oedema (CME) as manifestation of CAR. we present here the Optical Coherence Tomography (OCT) findings of the case and response to treatment by intravitreal Bevacizumab injection.

Key Messages

Cancer associated retinopathy (CAR) can present as gradually progressive bilateral moderate diminution of vision with cystoids macular oedema and gall bladder carcinoma can be one of the causes of CAR.

Case History

A 62 year old female patient presented in September 2014 with painless progressive diminution of vision in both eyes for last 4 months. At the time of presentation the patient did not have any systemic illness.

On ocular examination the patient vision had visual acuity of 6/24 (Log MAR 0.6) in both the eyes with no improvement with refractive correction. Early bilateral immature senile cataracts were present but did corroborate the significant vision loss. On fundus evaluation bilateral cystoids macular oedema (CME) was seen with no other associate findings. The OCT showed bilateral CME with central macular thickness of 503 micron and 514 micron in right eye and left eye respectively. (Figure 1. Figure 2. OCT macula Right eye and Left eye respectively showing increased macular thickness with typical cystoids spaces). There was no known systemic disease at the time of presentation.

Reprint Request: Sanjiv Kumar Gupta, Professor,
Department of Ophthalmology, King George's Medical
University, Chowk, Lucknow, Uttar Pradesh 226003 U.P., India.
E-mail: sanjiv204@gmail.com

Patient's blood investigations including blood sugar, hemogram, and kidney function tests were normal. However, the Liver function tests revealed marginally raised serum glutamic oxaloacetic transaminase (SGOT) (58.0 IU/Lt) and Alkaline Phosphatase enzymes (163.6 IU/Lt).

Patient was administered intravitreal Bevacizumab for cystoid macular oedema in both

the eyes and was kept on follow up. Repeat OCT during follow up after 6 weeks of the intervention, showed reduction in CME (Figure 3. Figure 4. OCT macula of right and left eyes respectively after 6 weeks of intravitreal Bevacizumab injections) and the visual acuity improved to 6/18 in both the eyes.

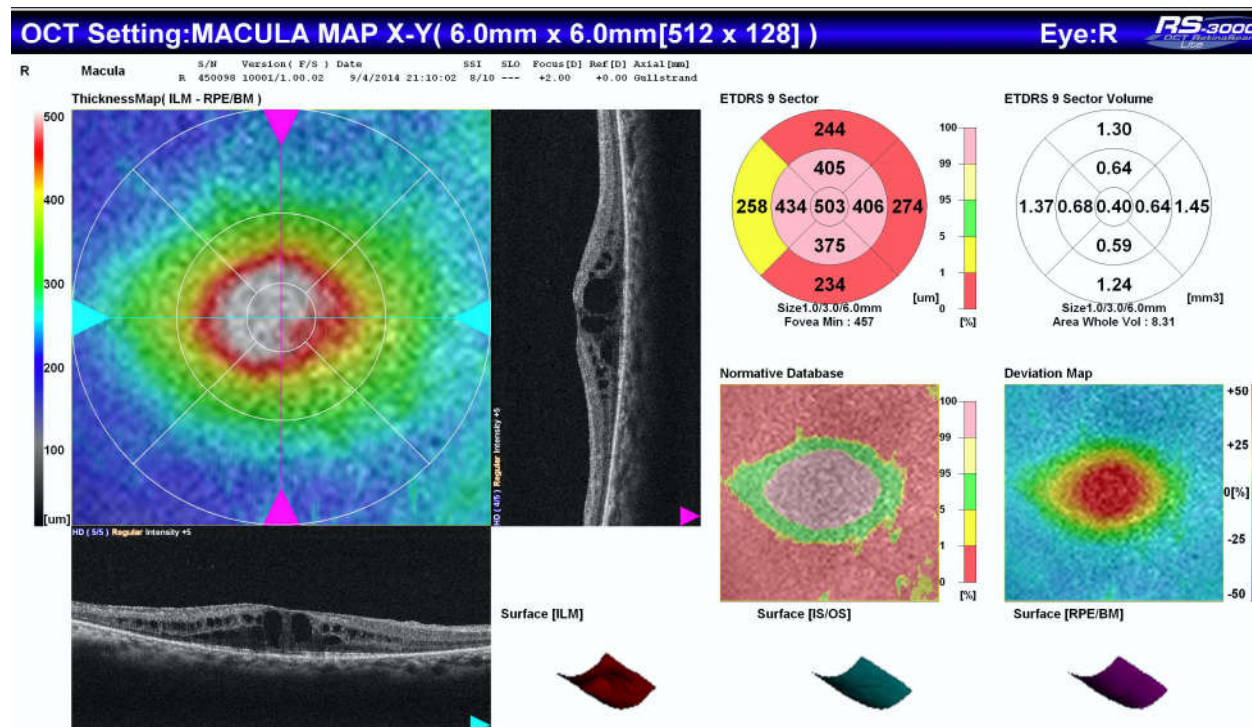


Fig. 1:

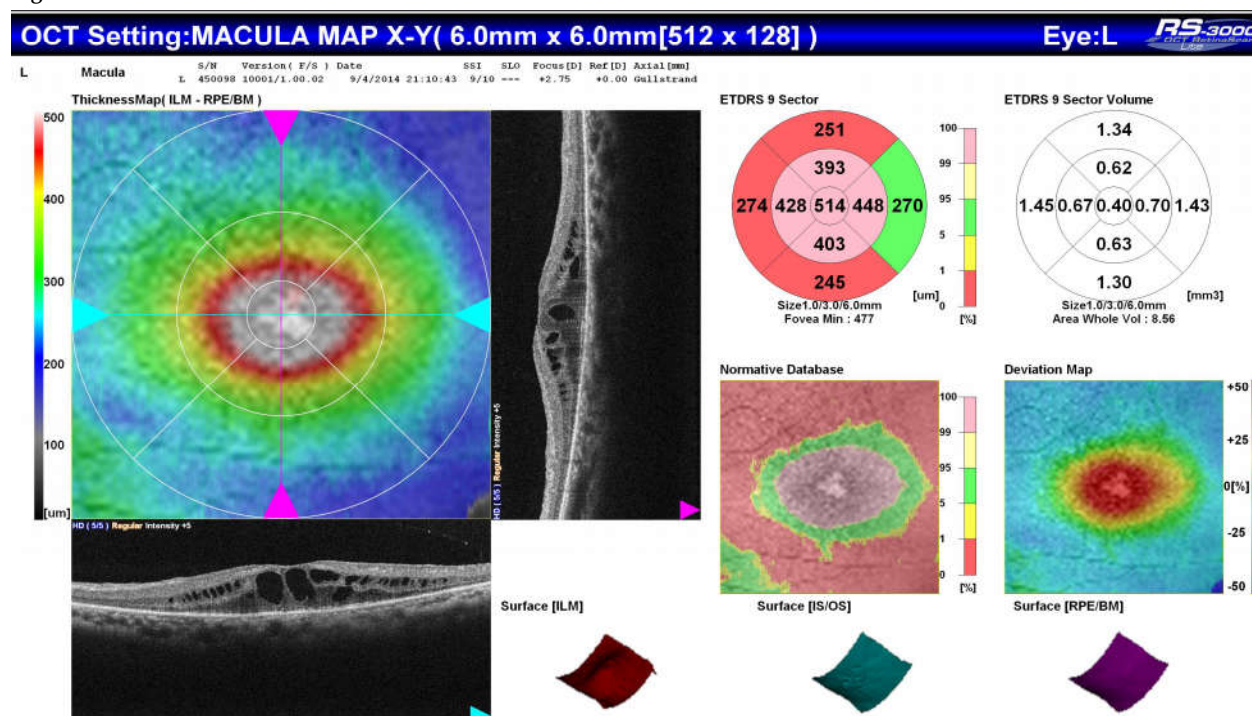


Fig. 2:

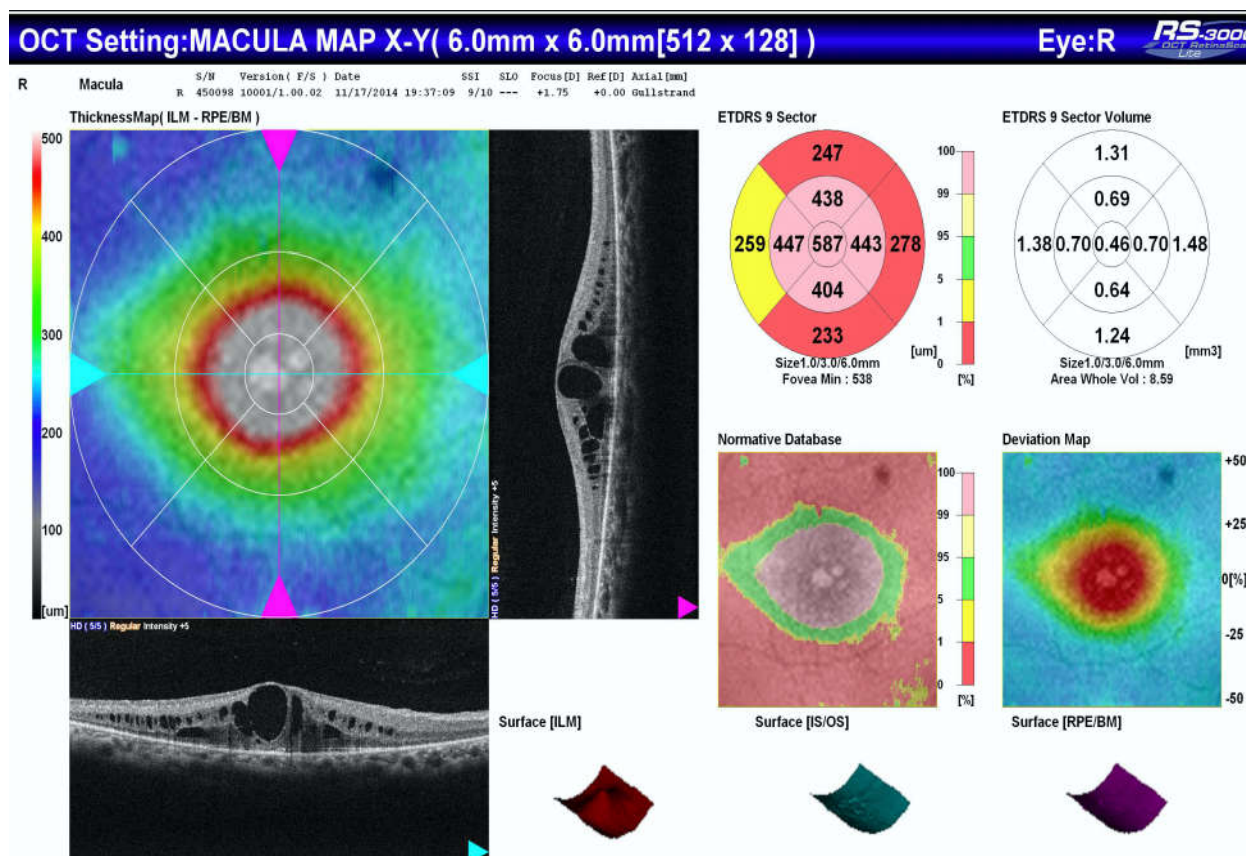


Fig. 3:

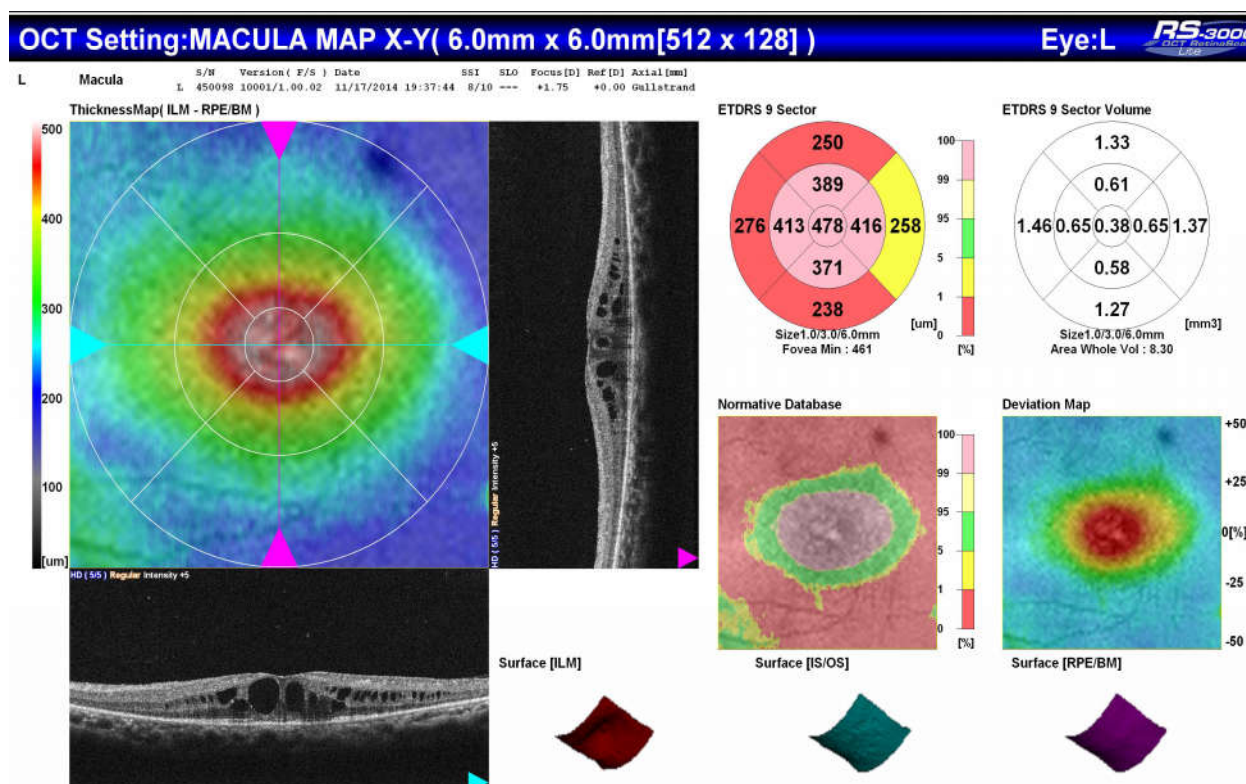


Fig. 4:

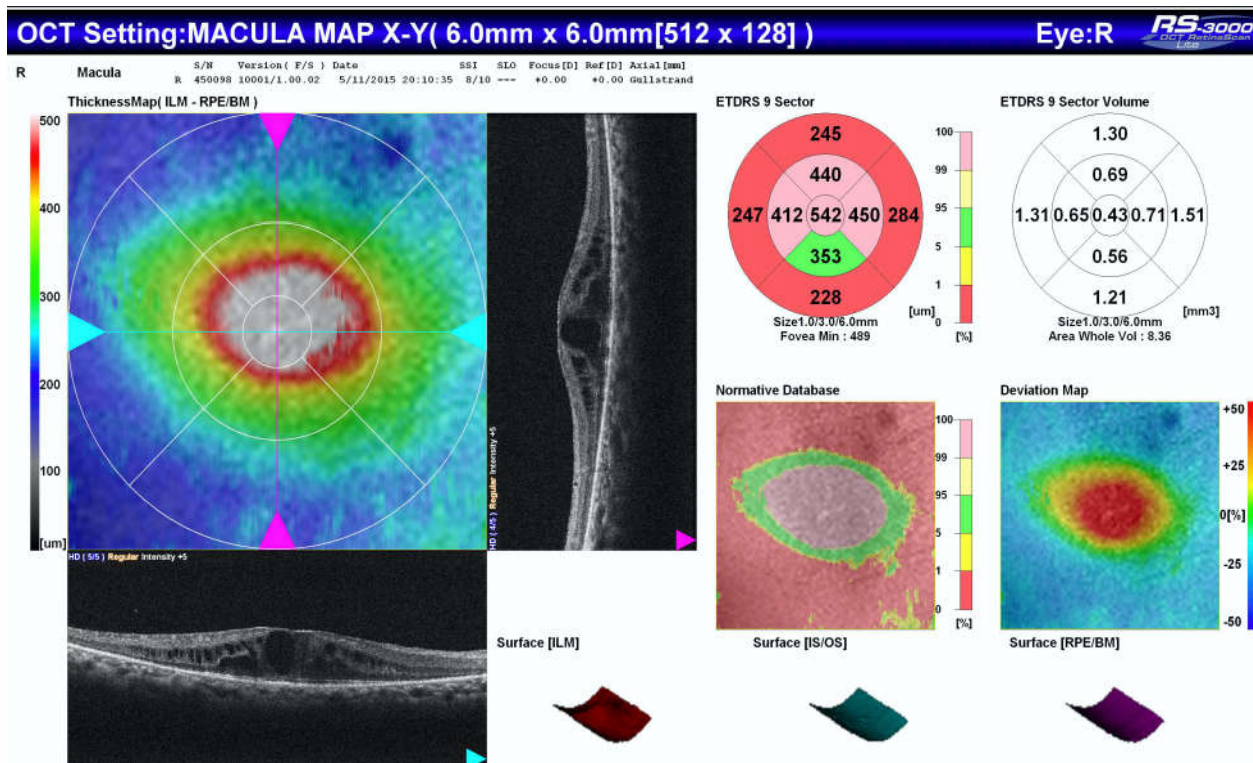


Fig. 5:

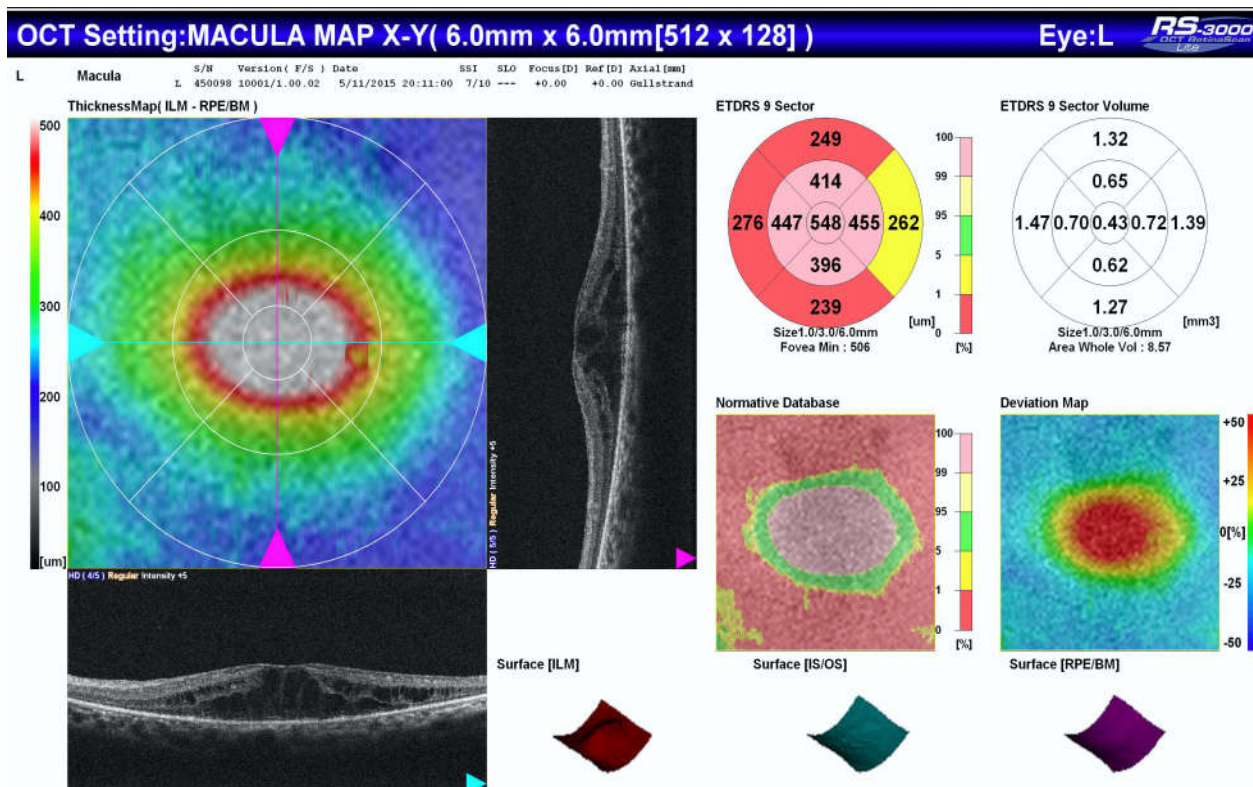


Fig. 6:



Fig. 7:

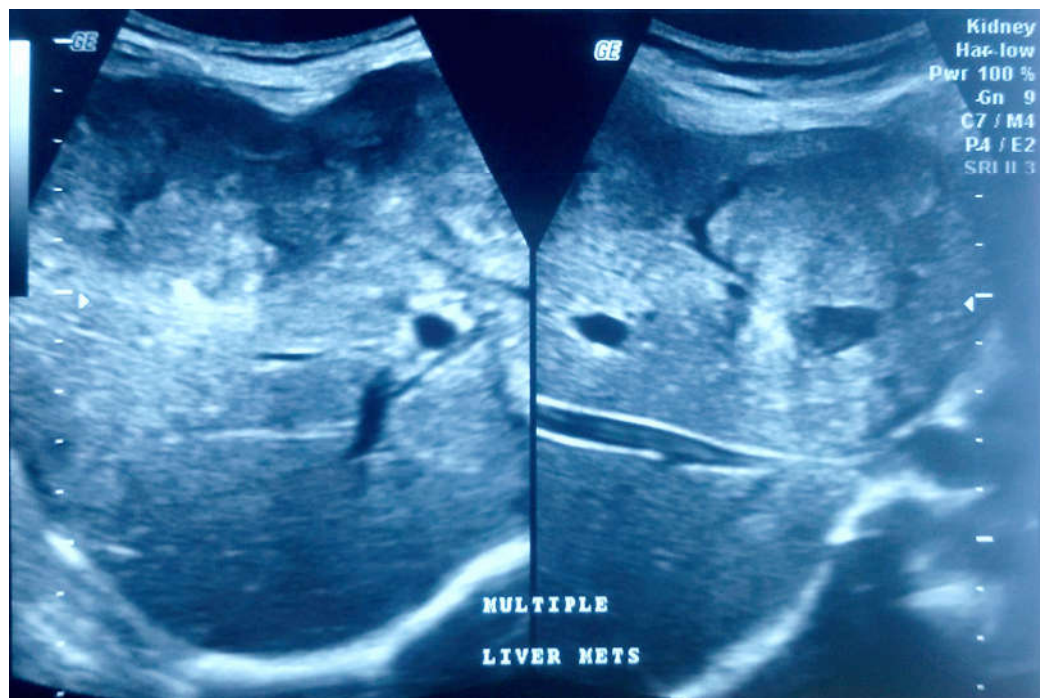


Fig. 8:

In May 2015 patient again presented with similar symptoms of painless progressive diminution of vision with Best Corrected Visual Acuity of 6/24 and recurrence of CME in both the eyes (Figure 5. Figure 6. OCT macula of right and left eyes respectively showing increased macular thickness and cystoids

changes indicating recurrence of CME). Patient was planned for intravitreal steroids for recurrent CME as there had been only partial response to earlier intravitreal injections of Bevacizumab.

Before this therapy could be administered, patient started having symptoms of abdominal pain and loss

of appetite with weight loss. Patient was seen by the family physician and was diagnosed to have lump in abdomen in relation to liver. Ultrasonography of abdomen was done on in June 2015 which revealed a complex mass of mixed ecogenecity with embedded calculi (mass measured 3.4 x 4.8 x 3.5 cm). The liver was normal in size but showed multiple hypo echoic masses of variable sizes in both the lobes (Figure 7). Ultrasound abdomen showing gall bladder lump with calculi, Figure 8. Ultrasound abdomen showing Multiple liver metastasis). A provisional diagnosis of gall bladder carcinoma was made and the patient was referred to an oncologist.

The disease was too advanced at the time of presentation for any curative treatment and hence patient was put on palliative treatment consisting of Opioid analgesics and other supportive treatment to aid nutrition. The patient succumbed to the disease in august 2015.

Discussion

Cancer Associated Retinopathy (CAR) was first described by Sawyer et al. in 1976 with three cancer patients with blindness caused by diffuse retinal degeneration. In CAR, retinal degeneration occurs in the presence of auto-antibodies that cross react with tumour-tissue and retinal-tissue antigens which are recognized as foreign. In many instances, visual loss from CAR precedes the diagnosis of cancer [2].

CAR is Commonly associated with small-cell lung cancer, followed by gynaecologic and breast cancers, non-small-cell lung cancer, Hodgkin lymphoma, and pancreatic, prostate, bladder, laryngeal, and colon cancers [3]. Association of CAR with gall bladder carcinoma has not been reported as per the literature search at the time of reporting.

The temporal association of CAR with reported malignancies is very variable (from years before the malignancy is detected to months after the diagnosis of malignancy) [4]. The gall bladder carcinoma is known to have median survival period of 3 months [5] and in most patients the disease is incurable at the time of presentation, and many patients can be offered only palliative treatment [6]. Similarly our case was also diagnosed late, when there was local spread of the tumour and only palliative treatment was possible.

CME due to CAR can be considered a very rare presentation of autoimmune retinopathy due to paraneoplastic syndromes because majority of patients of CAR have been reported to have normal

fundus findings at presentation, rest had vascular attenuation, or RPE changes [10]. Thinning of the inner retinal layers has been demonstrated with optical coherence tomography (OCT) in CAR [7]. On literature search we could find only one single case report by Moyer Ket al describing cystoid macular oedema as a manifestation of CAR in a case of small cell carcinoma of lungs in a male patient [8].

The visual loss was painless progressive and was moderate at the time of presentation in contrast to the progressive optic atrophy and rapid severe vision loss as reported in autoimmune cancer related retinopathies in other series [9].

There was partial response to the anti-VEGF therapy with moderate improvement in vision and reduction in the CME as seen on OCT done 6 weeks after the therapy. The role of intra-ocular anti-VEGF (Bevacizumab) has not been reported in these patients yet.

We could not test the patient for anti-retinal antibodies for various reasons but though the presence of antibodies to various antigens (recoverin, enolase, transducin, carbonic anhydrase, arrestin, retinal bipolar cells, and transducin) [10] have been identified but the value of these in establishing the diagnosis is still on [11, 12] and in majority of cases the diagnosis is based on clinical findings.

The Case Report of this Patient with CAR Highlights That

1. CME can be a presenting sign of CAR and the associated vision loss is moderate with gradual progression.
2. The gall bladder carcinoma can be a cause of CAR and CME may be the presenting condition.
3. The treatment of such cases of CME is partially responsive to intraocular anti-VEGF (Bevacizumab) therapy in form of improvement in vision and changes as seen on OCT.
4. A check up by a physician should be recommended in patients with CME, in the absence of any contributory / risk factor. We could have probably diagnosed the malignancy earlier significantly altering the eventual outcome had search for cause been undertaken at first presentation.

References

- 1 Arnold AC, Lee AG. Systemic disease and neuro-ophthalmology: Annual update 2000. J

- Neuroophthalmology. 2001; 21: 46-61.
 2. Khan N, Huang JJ, Foster CS. Cancer associated retinopathy (CAR): An autoimmune-mediated paraneoplastic syndrome. *Semin Ophthalmol.* 2006; 21: 135-41.
 3. Adamus G. Autoantibody targets and their cancer relationship in the pathogenicity of paraneoplastic retinopathy. *Autoimmun Rev.* 2009; 8: 410-4.
 4. Saito W, Kase S, Ohguro H, Furudate N, Ohno S. Slowly progressive cancer-associated retinopathy. *Arch Ophthalmol.* 2007; 125: 1431-3.
 5. Chan SY, Poon RT, Lo CM, Ng KK, Fan ST. Management of carcinoma of the gallbladder: a single-institution experience in 16 years. *J Surg Oncol.* 2008; 97: 156-64.
 6. Ouchi K, Suzuki M, Saijo S, Ito K, Matsuno S. Do recent advances in diagnosis and operative management improve the outcome of gallbladder carcinoma? *Surgery.* 1987; 101: 731-7.
 7. Mohamed Q, Harper CA. Acute optical coherence tomographic findings in cancer-associated retinopathy. *Arch Ophthalmol.* 2007; 125: 1132-3.
 8. Moyer K, DeWilde A, Law C. Cystoid macular edema from cancer-associated retinopathy. *Optom Vis Sci.* 2014; 91: 66-70.
 9. Keltner JL, Thirkill CE, Yip PT. Clinical and immunologic characteristics of melanoma-associated retinopathy syndrome: eleven new cases and a review of 51 previously published cases. *J Neuroophthalmol.* 2001; 21: 173-87.
 10. Potter MJ, Adamus G, Szabo SM, Lee R, Mohaseb K, Behn D. Autoantibodies to transducin in a patient with melanoma-associated retinopathy. *Am J Ophthalmol.* 2002; 134: 128-30.
 11. Forooghian F1, Macdonald IM, Heckenlively JR, Héon E, Gordon LK, Hooks JJ, Detrick B, Nussenblatt RB. The need for standardization of antiretinal antibody detection and measurement. *Am J Ophthalmol.* 2008; 146: 489-95.
 12. Braithwaite T, Holder GE, Lee RW, Plant GT, Tufail A. Diagnostic features of the autoimmune retinopathies. *Autoimmun Rev.* 2014; 13: 534-8.
-

Subscription Form

I want to renew/subscribe international class journal **“Ophthalmology and Allied Sciences”** of Red Flower Publication Pvt. Ltd.

Subscription Rates:

- Institutional: INR5500/USD550
- Individual: Contact us

Name and complete address (in capitals):

Payment detail:

Demand Draft No.

Date of DD

Amount paid Rs./USD

1. Advance payment required by Demand Draft payable to Red Flower Publication Pvt. Ltd. payable at Delhi.
2. Cancellation not allowed except for duplicate payment.
3. Agents allowed 10% discount.
4. Claim must be made within six months from issue date.

Mail all orders to

Red Flower Publication Pvt. Ltd.

48/41-42, DSIDC, Pocket-II

Mayur Vihar Phase-I

Delhi - 110 091(India)

Phone: 91-11-45796900, 22754205, 22756995, Fax: 91-11-22754205

E-mail: customer.rfp@rfppl.co.in, customer.rfp@gmail.com

Website: www.rfppl.co.in

A Rare Case of Unilateral Star Shaped Cataract Following Electric Shock

Meena Ashok Kr*, Soni Akshar**, Gupta Tarun***

Authors Affiliation: *Professor & Head, **3rd Yr Resident ***2nd Yr Resident, Department of Ophthalmology, Government Medical College, Kota, Rajasthan.

Abstract

A 25 year old male reported to eye- opd with complaint of diminution of vision of left eye for 20 days following electric shock (11,000v) approximately three months back. On examination he had a unilateral star shaped anterior subcapsular cataract in his left eye. He was operated for cataract and achieved 6/6 and N6 visual acuity 3 weeks after surgery. This case highlights rare unilateral cataract following electric shock and excellent outcome following surgery provided fundus and optic nerve are unaffected. Need for awareness of this complication and screening of all cases of electric injury is emphasized.

Keywords: Anterior Subcapsular Cataract Electric Shock; Unilateral Star Shaped Cataract.

Introduction

Systemic complications from electrical injury can be multisystemic, varied, debilitating, and are frequently fatal[1]. It can result in a wide range of ocular injuries with resultant ocular complications [2,3]. Cataracts develop in approximately 6% of cases of high-voltage injuries, especially whenever electrical injury occurs in the vicinity of the head [4]. Of these, electrical cataract can occur after a latent period and then progress with startling rapidity [5]. However proper surgical management can result in good and stable visual acuity as is seen in this case. The need for awareness of the possibility of this complication and screening of all cases of electrical injuries is stressed.

This is a case report of unilateral star shaped cataract in a 25 year old male secondary to electric shock.

Key Message

Unilateral star shaped cataract following high voltage electric shock and excellent outcome

following surgery provided fundus and optic nerve are unaffected. Need of screening of all cases of electric shock so that this complication can be managed early and effectively.

Case Report

A 25yr old male with a history electric shock reported to eye-opd with complaint of diminution of vision in his left eye for 20 days. Patient had sustained an electric shock from a high tension line (11,000 volts) approximately three months back while he was working in fields. Patient got unconscious and was admitted in our hospital for burns on his body. The electric current passed from his left hand and exited through his right foot (Figure 1). He received burn on his left arm, trunk, right hand and right foot for which his left arm had to be amputated below elbow.

Reprint Request: Ashok Kumar Meena,
27, Atwal Nagar, Kota-324001, Rajasthan.
E-mail: dr.ashokmeena10@gmail.com

On examination visual acuity of his right eye was 6/6 and left eye has only finger counting at 2m improving to 6/36 with pinhole. Right eye examination was within normal limits whereas left eye had a characteristic anterior subcapsular star shaped cataract (Figure 2) and posterior subcapsular cataract (Figure 3) with normal depth of anterior chamber and fundus was not clearly visible.

He was operated for left eye cataract and a posterior chamber IOL was placed. The surgery was

uneventful.

His first post-op day vision was 6/36 improving to 6/18 with pinhole. Cornea was clear, anterior chamber was deep, and good red reflex was seen.

On review 3 weeks later, his best corrected visual acuity is 6/6, N6 in left eye.



Fig. 1: Amputated left hand and arrow showing exit wound

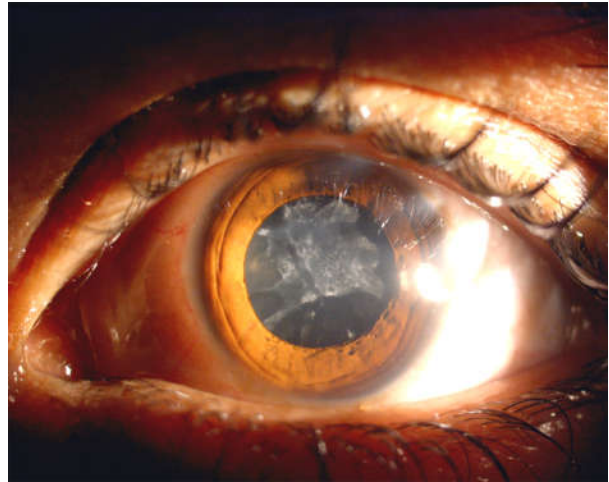


Fig. 2: Star shaped anterior subcapsular cataract

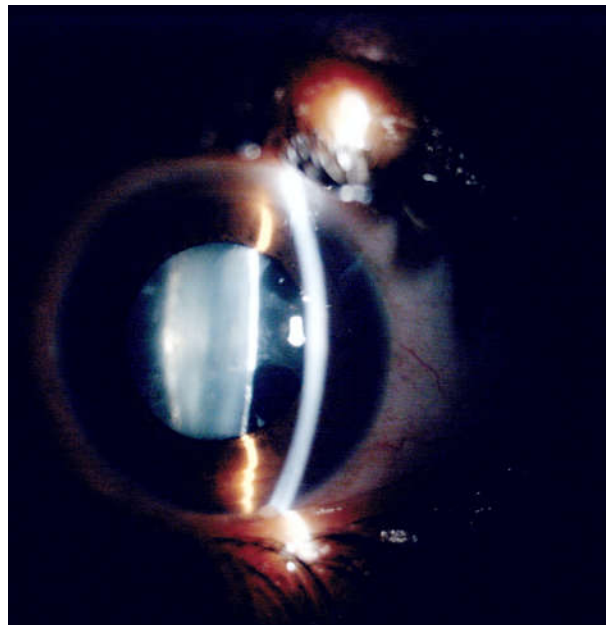


Fig. 3: Anterior and posterior subcapsular cataract

Discussion

High voltage electric burns can cause various ocular injuries and may manifest in the form of conjunctival hyperemia, corneal opacities, uveitis, miosis, spasm of accommodation, cataract, retinal edema, papilledema, chorio-retinal necrosis/atrophy, retinal detachment and optic atrophy.

Choroidal rupture, optic neuritis and retinal detachment may also be seen. Macular edema may progress to macular cysts or holes [2,3,6]. Although a number of these ocular changes occur immediately after injury [7], many develop days and even years after [4,6]. The cataract may develop immediately after injury or be delayed a few days; the latency varies from 1 to 18 months [8] although a latent period of 11 years has also been reported [9]. It is usually bilateral [2] but can also occur unilaterally [7]. If the point of contact is near to one side, the cataract may develop on that side first and then on the other side. The interval between cataracts occurring in the 2 eyes can vary from 3 weeks to 2 years. As in this case who developed unilateral cataract. Cataract usually occurs 1-12 month [4] after the accident and is frequently associated with no other observable ocular damage. The exact pathogenesis of these cataracts is unknown, but direct coagulation of proteins and osmotic changes following damage to the subcapsular epithelium are thought to be responsible [2]. The earliest changes seen in the lens after electrical injury are a collection of multiple fine vacuoles beneath the anterior capsule, usually in the midperiphery of the lens, requiring dilation of the pupil for visualization. These collections are always present in the anterior subcapsular area and show no apparent relationship to lens fiber configuration. Over intervals varying from weeks to months, these vacuoles are replaced with flake-like opacities that coalesce and migrate into the line of vision. Electrical burn can cause scar formation in the anterior capsule, leading to impairment of lens nutrition and, eventually, cataract formation.

Rarely, the cataract may become complicated by secondary glaucoma in the intumescent stage [10].

Thus, proper surgical management of electric cataract will result in a good visual rehabilitation if the eye has otherwise escaped damage as in this case.

Conclusion

Electric injuries can cause unilateral or bilateral cataracts. Proper and timely management of electric cataract have excellent outcome provided fundus and optic nerve are unaffected.

References

1. Demling RH. Electrical trauma: pathophysiology and clinical management. In: Lee RC, Cravalho EG, Burke JF, eds. *Electrical Trauma: The pathophysiology, manifestations and clinical management*. Cambridge: Cambridge University Press. 1992: 122-132.
2. Boozalis GT, Purdu GF. Ocular changes from electric burn injuries: a literature review and report of cases. *J Burn Care Rehabil*. 1991; 12: 458-62.
3. Albert, Jakobiec. *Principles and Practice of Ophthalmology, Posterior Segment Trauma*. Philadelphia: Paul Dieckert, B Saunders Company; 1994: 3419.
4. Saffle J R, Crandall A :Cataracts a long term complication of electrical injury. *J Trauma*. 1985 Jan; 25(1): 17-21.
5. Stephen V, John SR, Chakraborty A, Chakrabarti M. Bilateral cataract following electrical injury. *Kerala J Ophthalmol*. 2006; 18(3): 252e254.
6. Grewal DS, Jain R, Brar GS, Grewal SPS. Unilateral electric cataract: Scheimpflug imaging and review of the literature. *J Cataract Refract Surg*. 2007; 33(6): 1116e1119.
7. Mutlu FM, Duman H, Cil Y. Early-onset unilateral electric cataract: a rare clinical entity. *J Burn Care Rehabil*. 2004; 25: 363-365.
8. Duke-Elder S, MacFaul PA. Injuries; Non-Mechanical Injuries. In: Duke-Elder, ed, *System of Ophthalmology*. London, Henry Kimpton. 1972; 14(2): 813-835.
9. Skoog T. Electrical injuries. *J Trauma*. 1970; 10: 816-830.
10. Reddy SC. Electric cataract: a case report and review of the literature. *Eur J Ophthalmol*. 1999; 9: 134-138.

Instructions to Authors

Submission to the journal must comply with the Guidelines for Authors.

Non-compliant submission will be returned to the author for correction.

To access the online submission system and for the most up-to-date version of the Guide for Authors please visit:

<http://www.rfppl.co.in>

Technical problems or general questions on publishing with OAS are supported by Red Flower Publication Pvt. Ltd's Author Support team (<http://www.rfppl.co.in>)

Alternatively, please contact the Journal's Editorial Office for further assistance.

Editorial Manager

Red Flower Publication Pvt. Ltd.

48/41-42, DSIDC, Pocket-II

Mayur Vihar Phase-I

Delhi - 110 091(India)

Phone: 91-11-22754205, 45796900, 22756995, Fax: 91-11-22754205

author.rfp@rfppl.co.in

author.rfp@gmail.com

Manuscripts must be prepared in accordance with "Uniform requirements for Manuscripts submitted to Biomedical Journal" developed by international committee of medical Journal Editors.

Types of Manuscripts and Limits

Original articles: Up to 3000 words excluding references and abstract and up to 10 references.

Review articles: Up to 2500 words excluding references and abstract and up to 10 references.

Case reports: Up to 1000 words excluding references and abstract and up to 10 references.

Online Submission of the Manuscripts

Articles can also be submitted online from http://rfppl.co.in/customer_index.php.

1) First Page File: Prepare the title page, covering letter, acknowledgement, etc. using a word processor program. All information which can reveal your identity should be here. use text/rtf/doc/PDF files. Do not zip the files.

2) Article file: The main text of the article, beginning from Abstract till References (including tables) should be in this file. Do not include any information (such as acknowledgement, your name in page headers, etc.) in this file. Use text/rtf/doc/PDF files. Do not zip the files. Limit the file size to 400 Kb. Do not incorporate images in the file. If file size is large, graphs can be submitted as images separately without incorporating them in the article file to reduce the size of the file.

3) Images: Submit good quality color images. Each image should be less than 100 Kb in size. Size of the image can be reduced by decreasing the actual height and width of the images (keep up to 400 pixels or 3 inches). All image formats (jpeg, tiff, gif, bmp, png, eps etc.) are acceptable; jpeg is most suitable.

Legends: Legends for the figures/images should be included at the end of the article file.

If the manuscript is submitted online, the contributors' form and copyright transfer form has to be submitted in original with the signatures of all the contributors within two weeks from submission. Hard copies of the images (3 sets), for articles submitted online, should be sent to the journal office at the time of submission of a revised manuscript. Editorial office: Red Flower Publication Pvt. Ltd., 48/41-42, DSIDC, Pocket-II, Mayur Vihar Phase-I, Delhi - 110 091, India, Phone: 91-11-45796900, 22754205, 22756995, Fax: 91-11-

22754205, E-mail: author.rfp@gmail.com, author.rfp@gmail.com, Website: www.rfppl.co.in

Preparation of the Manuscript

The text of observational and experimental articles should be divided into sections with the headings: Introduction, Methods, Results, Discussion, References, Tables, Figures, Figure legends, and Acknowledgment. Do not make subheadings in these sections.

Title Page

The title page should carry

- 1) Type of manuscript (e.g. Original article, Review article, Case Report)
- 2) The title of the article, should be concise and informative;
- 3) Running title or short title not more than 50 characters;
- 4) The name by which each contributor is known (Last name, First name and initials of middle name), with his or her highest academic degree(s) and institutional affiliation;
- 5) The name of the department(s) and institution(s) to which the work should be attributed;
- 6) The name, address, phone numbers, facsimile numbers and e-mail address of the contributor responsible for correspondence about the manuscript; should be mentioned.
- 7) The total number of pages, total number of photographs and word counts separately for abstract and for the text (excluding the references and abstract);
- 8) Source(s) of support in the form of grants, equipment, drugs, or all of these;
- 9) Acknowledgement, if any; and
- 10) If the manuscript was presented as part at a meeting, the organization, place, and exact date on which it was read.

Abstract Page

The second page should carry the full title of the manuscript and an abstract (of no more than 150 words for case reports, brief reports and 250 words for original articles). The abstract should be structured and state the Context (Background), Aims, Settings and Design, Methods and Materials, Statistical analysis used, Results and Conclusions. Below the abstract should provide 3 to 10 keywords.

Introduction

State the background of the study and purpose of the study and summarize the rationale for the study or observation.

Methods

The methods section should include only information that was available at the time the plan or protocol for the study was written such as study approach, design, type of sample, sample size, sampling technique, setting of the study, description of data collection tools and methods; all information obtained during the conduct of the study belongs in the Results section.

Reports of randomized clinical trials should be based on the CONSORT Statement (<http://www.consort-statement.org>). When reporting experiments on human subjects, indicate whether the procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional or regional) and with the Helsinki Declaration of 1975, as revised in 2000 (available at http://www.wma.net/e/policy/17-c_e.html).

Results

Present your results in logical sequence in the text, tables, and illustrations, giving the main or most important findings first. Do not repeat in the text all the data in the tables or illustrations; emphasize or summarize only important observations. Extra or supplementary materials and technical details can be placed in an appendix where it will be accessible but will not interrupt the flow of the text; alternatively, it can be published only in the electronic version of the journal.

Discussion

Include summary of key findings (primary outcome measures, secondary outcome measures, results as they relate to a prior hypothesis); Strengths and limitations of the study (study question, study design, data collection, analysis and interpretation); Interpretation and implications in the context of the totality of evidence (is there a systematic review to refer to, if not, could one be reasonably done here and now?, What this study adds to the available evidence, effects on patient care and health policy, possible mechanisms)? Controversies raised by this study; and Future research directions (for this particular research collaboration, underlying

mechanisms, clinical research). Do not repeat in detail data or other material given in the Introduction or the Results section.

References

List references in alphabetical order. Each listed reference should be cited in text (not in alphabetic order), and each text citation should be listed in the References section. Identify references in text, tables, and legends by Arabic numerals in square bracket (e.g. [10]). Please refer to ICMJE Guidelines (http://www.nlm.nih.gov/bsd/uniform_requirements.html) for more examples.

Standard journal article

[1] Flink H, Tegelberg Å, Thörn M, Lagerlöf F. Effect of oral iron supplementation on unstimulated salivary flow rate: A randomized, double-blind, placebo-controlled trial. *J Oral Pathol Med* 2006; 35: 540-7.

[2] Twetman S, Axelsson S, Dahlgren H, Holm AK, Källestål C, Lagerlöf F, et al. Caries-preventive effect of fluoride toothpaste: A systematic review. *Acta Odontol Scand* 2003; 61: 347-55.

Article in supplement or special issue

[3] Fleischer W, Reimer K. Povidone iodine antiseptics. State of the art. *Dermatology* 1997; 195 Suppl 2: 3-9.

Corporate (collective) author

[4] American Academy of Periodontology. Sonic and ultrasonic scalers in periodontics. *J Periodontol* 2000; 71: 1792-801.

Unpublished article

[5] Garoushi S, Lassila LV, Tezvergil A, Vallittu PK. Static and fatigue compression test for particulate filler composite resin with fiber-reinforced composite substructure. *Dent Mater* 2006.

Personal author(s)

[6] Hosmer D, Lemeshow S. Applied logistic regression, 2nd edn. New York: Wiley-Interscience; 2000.

Chapter in book

[7] Nauntofte B, Tenovou J, Lagerlöf F. Secretion and composition of saliva. In: Fejerskov O, Kidd EAM,

editors. Dental caries: The disease and its clinical management. Oxford: Blackwell Munksgaard; 2003. p. 7-27.

No author given

[8] World Health Organization. Oral health surveys - basic methods, 4th edn. Geneva: World Health Organization; 1997.

Reference from electronic media

[9] National Statistics Online – Trends in suicide by method in England and Wales, 1979-2001. www.statistics.gov.uk/downloads/theme_health/HSQ_20.pdf (accessed Jan 24, 2005): 7-18. Only verified references against the original documents should be cited. Authors are responsible for the accuracy and completeness of their references and for correct text citation. The number of reference should be kept limited to 20 in case of major communications and 10 for short communications.

More information about other reference types is available at www.nlm.nih.gov/bsd/uniform_requirements.html, but observes some minor deviations (no full stop after journal title, no issue or date after volume, etc).

Tables

Tables should be self-explanatory and should not duplicate textual material.

Tables with more than 10 columns and 25 rows are not acceptable.

Table numbers should be in Arabic numerals, consecutively in the order of their first citation in the text and supply a brief title for each.

Explain in footnotes all non-standard abbreviations that are used in each table.

For footnotes use the following symbols, in this sequence: *, †, ‡, §§,

Illustrations (Figures)

Graphics files are welcome if supplied as Tiff, EPS, or PowerPoint files of minimum 1200x1600 pixel size. The minimum line weight for line art is 0.5 point for optimal printing.

When possible, please place symbol legends below the figure instead of to the side.

Original color figures can be printed in color at the editor's and publisher's discretion provided the author agrees to pay.

Type or print out legends (maximum 40 words, excluding the credit line) for illustrations using double spacing, with Arabic numerals corresponding to the illustrations.

Sending a revised manuscript

While submitting a revised manuscript, contributors are requested to include, along with single copy of the final revised manuscript, a photocopy of the revised manuscript with the changes underlined in red and copy of the comments with the point to point clarification to each comment. The manuscript number should be written on each of these documents. If the manuscript is submitted online, the contributors' form and copyright transfer form has to be submitted in original with the signatures of all the contributors within two weeks of submission. Hard copies of images should be sent to the office of the journal. There is no need to send printed manuscript for articles submitted online.

Reprints

Journal provides no free printed reprints, however a author copy is sent to the main author and additional copies are available on payment (ask to the journal office).

Copyrights

The whole of the literary matter in the journal is copyright and cannot be reproduced without the written permission.

Declaration

A declaration should be submitted stating that the manuscript represents valid work and that neither this manuscript nor one with substantially similar content under the present authorship has been published or is being considered for publication elsewhere and the authorship of this article will not be contested by any one whose name (s) is/are not listed here, and that the order of authorship as placed in the manuscript is final and accepted by the co-authors. Declarations should be signed by all the authors in the order in which they are mentioned in the original manuscript. Matters appearing in the Journal are covered by copyright but no objection will be made to their reproduction provided permission is obtained from the Editor prior to publication and due acknowledgment of the source is made.

Abbreviations

Standard abbreviations should be used and be spelt out when first used in the text. Abbreviations should not be used in the title or abstract.

- Abbreviations spelt out in full for the first time. Numerals from 1 to 10 spelt out
- Numerals at the beginning of the sentence spelt out

Checklist

- Manuscript Title
- Covering letter: Signed by all contributors
- Previous publication/ presentations mentioned, Source of funding mentioned
- Conflicts of interest disclosed

Authors

- Middle name initials provided.
- Author for correspondence, with e-mail address provided.
- Number of contributors restricted as per the instructions.
- Identity not revealed in paper except title page (e.g.name of the institute in Methods, citing previous study as 'our study')

Presentation and Format

- Double spacing
- Margins 2.5 cm from all four sides
- Title page contains all the desired information. Running title provided (not more than 50 characters)
- Abstract page contains the full title of the manuscript
- Abstract provided: Structured abstract provided for an original article.
- Key words provided (three or more)
- Introduction of 75-100 words
- Headings in title case (not ALL CAPITALS). References cited in square brackets
- References according to the journal's instructions

Language and grammar

- Uniformly American English

Tables and figures

- No repetition of data in tables and graphs and in text.
- Actual numbers from which graphs drawn, provided.
- Figures necessary and of good quality (color)
- Table and figure numbers in Arabic letters (not Roman).
- Labels pasted on back of the photographs (no names written)
- Figure legends provided (not more than 40 words)
- Patients' privacy maintained, (if not permission taken)
- Credit note for borrowed figures/tables provided
- Manuscript provided on a CDROM (with double spacing)

Submitting the Manuscript

- Is the journal editor's contact information current?
- Is the cover letter included with the manuscript? Does the letter:
 1. Include the author's postal address, e-mail address, telephone number, and fax number for future correspondence?
 2. State that the manuscript is original, not previously published, and not under concurrent consideration elsewhere?
 3. Inform the journal editor of the existence of any similar published manuscripts written by the author?
 4. Mention any supplemental material you are submitting for the online version of your article. Contributors' Form (to be modified as applicable and one signed copy attached with the manuscript)

Revised Rates for 2016 (Institutional)

Title	Frequency	Rate (Rs): India	Rate (\$):ROW
Dermatology International	2	5000	500
Gastroenterology International	2	5500	550
Indian Journal of Agriculture Business	2	5000	500
Indian Journal of Anatomy	3	8000	800
Indian Journal of Ancient Medicine and Yoga	4	7500	750
Indian Journal of Anesthesia and Analgesia	3	7000	700
Indian Journal of Anthropology	2	12000	1200
Indian Journal of Biology	2	4000	400
Indian Journal of Cancer Education and Research	2	8500	850
Indian Journal of Communicable Diseases	2	8000	800
Indian Journal of Dental Education	4	4500	450
Indian Journal of Forensic Medicine and Pathology	4	15500	1550
Indian Journal of Forensic Odontology	2	4500	450
Indian Journal of Genetics and Molecular Research	2	6500	650
Indian Journal of Law and Human Behavior	2	5500	550
Indian Journal of Library and Information Science	3	9000	900
Indian Journal of Maternal-Fetal & Neonatal Medicine	2	9000	900
Indian Journal of Medical & Health Sciences	2	6500	650
Indian Journal of Obstetrics and Gynecology	3	7000	700
Indian Journal of Pathology: Research and Practice	3	11500	1150
Indian Journal of Plant and Soil	2	5500	550
Indian Journal of Preventive Medicine	2	6500	650
International Journal of Food, Nutrition & Dietetics	3	5000	500
International Journal of History	2	6500	650
International Journal of Neurology and Neurosurgery	2	10000	1000
International Journal of Political Science	2	5500	550
International Journal of Practical Nursing	3	5000	500
International Physiology	2	7000	700
Journal of Animal Feed Science and Technology	2	4100	410
Journal of Cardiovascular Medicine and Surgery	2	9100	910
Journal of Forensic Chemistry and Toxicology	2	9000	900
Journal of Microbiology and Related Research	2	8000	800
Journal of Orthopaedic Education	2	5000	500
Journal of Pharmaceutical and Medicinal Chemistry	2	16000	1600
Journal of Practical Biochemistry and Biophysics	2	5500	550
Journal of Social Welfare and Management	3	7500	750
New Indian Journal of Surgery	3	7100	710
Ophthalmology and Allied Sciences	2	5500	550
Otolaryngology International	2	5000	500
Pediatric Education and Research	3	7000	700
Physiotherapy and Occupational Therapy Journal	4	8500	850
Urology, Nephrology and Andrology International	2	7000	700

SUPER SPECIALITY JOURNALS

Indian Journal of Emergency Medicine	2	12000	1200
Indian Journal of Surgical Nursing	3	5000	500
Indian Journal of Trauma & Emergency Pediatrics	3	9000	900
International Journal of Pediatric Nursing	3	5000	500
Journal of Community and Public Health Nursing	2	5000	500
Journal of Geriatric Nursing	2	5000	500
Journal of Medical Images and Case Reports	2	5000	500
Journal of Nurse Midwifery and Maternal Health	3	5000	500
Journal of Organ Transplantation	2	25900	2590
Journal of Psychiatric Nursing	3	5000	500
Psychiatry and Mental Health	2	7500	750

Terms of Supply:

1. Advance payment required by Demand Draft payable to Red Flower Publication Pvt. Ltd. payable at Delhi.
2. Cancellation not allowed except for duplicate payment.
3. Agents allowed 10% discount.
4. Claim must be made within six months from issue date.

Order from

Red Flower Publication Pvt. Ltd., 48/41-42, DSIDC, Pocket-II, Mayur Vihar Phase-I, Delhi - 110 091 (India), Tel: 91-11-22754205, 45796900, Fax: 91-11-22754205. E-mail: customer.rfp@rfppl.co.in, customer.rfp@gmail.com, Website: www.rfppl.co.in

Advertisement





Connecting Doctors

A revolutionary mobile application that can change the lives of the doctors. It is tailored made for doctors keeping in mind their every day needs and struggles. And its free.



Stay Updated



Get your Deeam Job



Search and Connect



Discuss & Refer Cases

AVAILABLE ON





*Introducing a new sister concerned company of **Red Flower Publication Pvt. Ltd.***

RF Library Services Pvt. Ltd.

RF Library Services Pvt. Ltd. is a global market leader in managing professional information. We develop and deliver innovative services that enable the use of knowledge to its full extent. As the only information Service Company globally we play a key role in today's complex information marketplace. Founded in 1985 as a registered company under sub-section (2) of section 7 of the Companies Act, 2013 and rule 8 of the Companies (Incorporation) Rules, 2014, the business draws on more than a decade of experience within the information industry. With this knowledge, we satisfy the needs of thousands of customers from over 30 countries. We are a division of Red Flower Publication Pvt. Ltd.

Where we are based?

RF Library Services Pvt. Ltd is located in Delhi-91 in India.

RF Library Services Pvt. Ltd.

D-223/216, Laxmi Chambers, Laxmi Nagar,
Near Laxmi Nagar Metro Station,
Delhi-110092(India)

Tel: 011-22756995, Fax: 011-22756995

E-mail: custsupport@rflibraryservices.com, rflibrary.delhi@gmail.com

Website: www.rf-libraryservices.com