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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] by suggesting 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.

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

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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].

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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

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 $375 \mu\text{m}$ 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

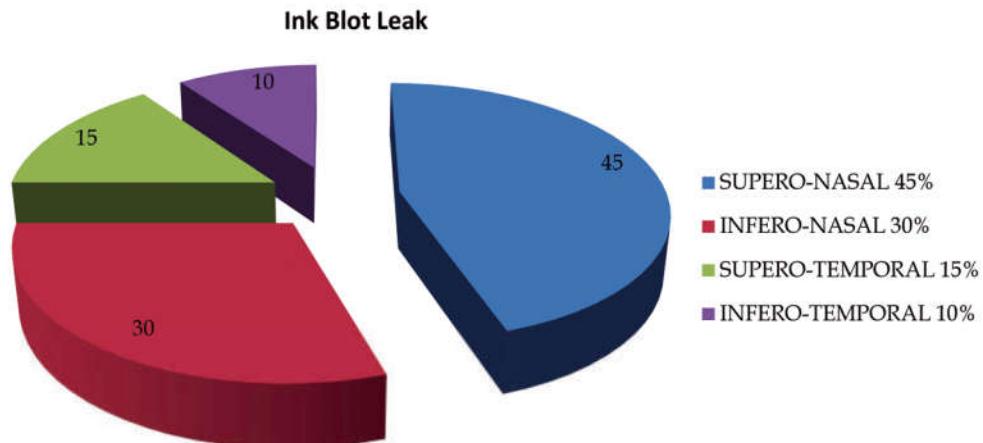


Fig. 1: Showing pattern of leak of ink blot type on Fluorescein Angiography (FA) in 20 eyes

Fig. 1: above shows different patterns of leaks of ink blot type encountered during FFA at time of presentation. Figure 2 shows 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

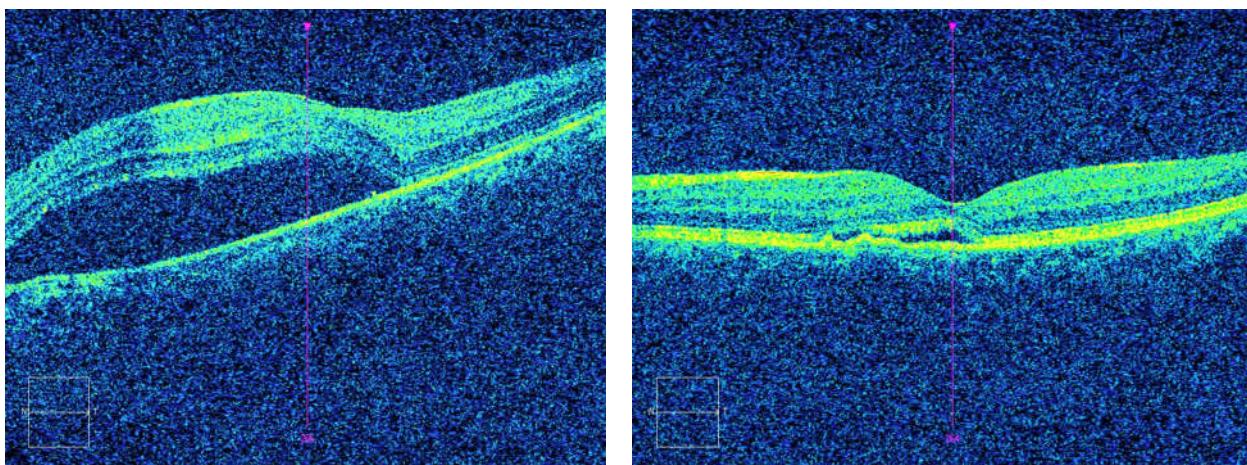


Fig. 3: CMT (central macular thickness) of 380 μm and post injection shows CMT of 250 μm of same patient at 08 weeks follow up post Inj Ranibizumab

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

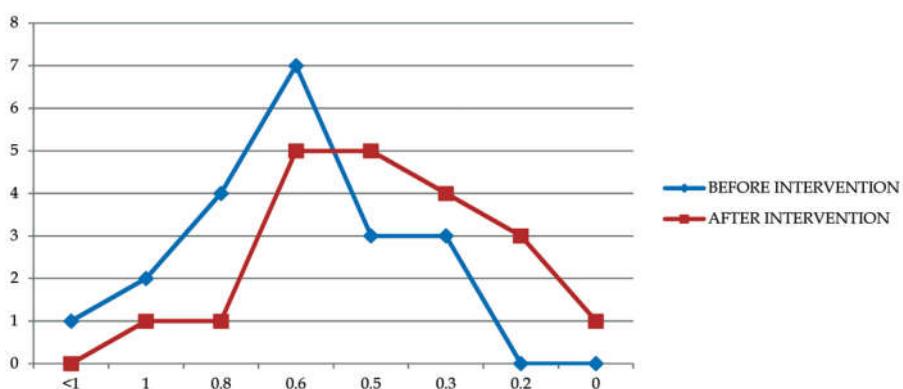


Fig. 4: Showing improvement in BCVA over a period of 02 months

um at baseline and decreased to 259 um. 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 μm and at 2 month was 259 μm . 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.

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Strabismus Surgery: Difficult Situations Simplified

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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

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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.

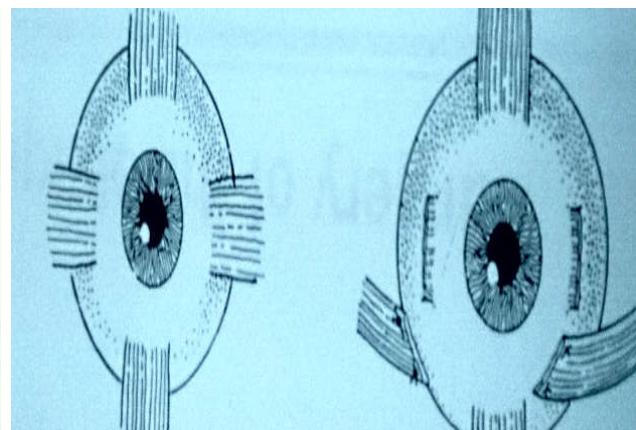
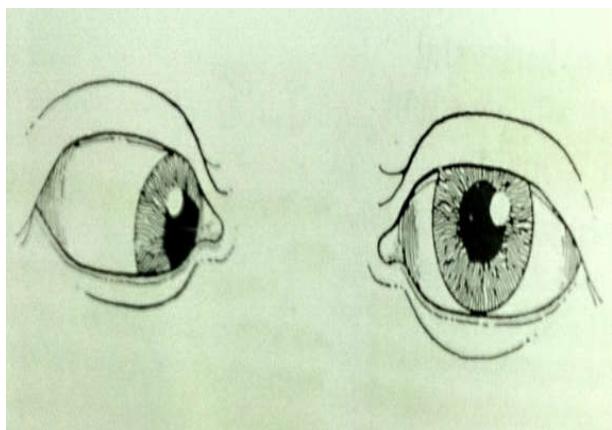
Group 2 (8 cases)

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 group1.

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

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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

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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

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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 lipoxygenase 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, descemets' 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.

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

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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.

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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%
Biacruate 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)

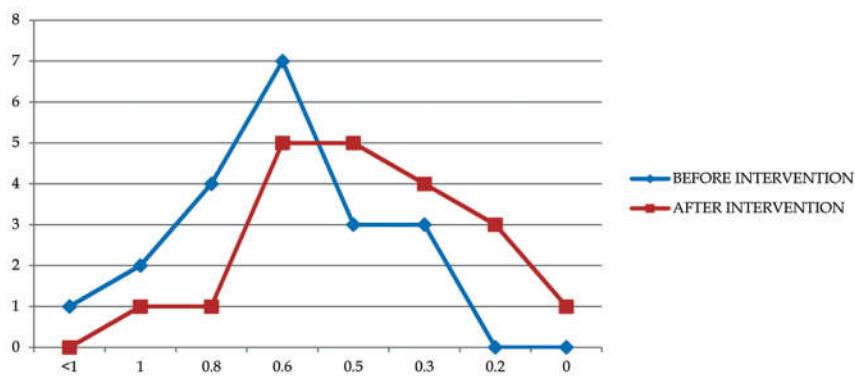
12% patients had early visual field defects, 40% patients had arcuate scotoma and 36% patients had severe damage with extensive visual field loss to small residual field. (Table 4)

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 pupillary reactions in normal subjects aged over 40 and in patients with POAG, found that in POAG the changes in the pupillary 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 tartarate 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.

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An Atypical Case of Sympathetic Ophthalmia Following Zone 1 Corneal Injury

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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 fundoscopy 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 inspite 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 complains of pain and decreased vision in left eye

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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 (StratusOCT, 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:

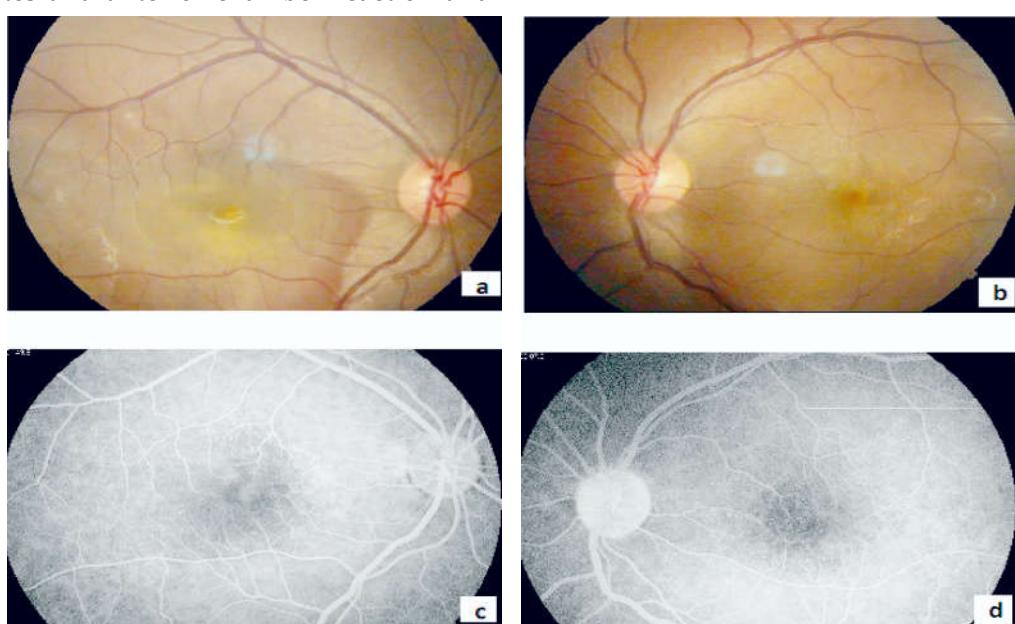


Fig. 1: A&B Colour fundus photographs exhibiting serous macular elevation. C&D Fundus fluorescein angiogram (FFA) photographs in peak arteriovenous filling exhibiting stippled hyperfluorescence in the posterior pole

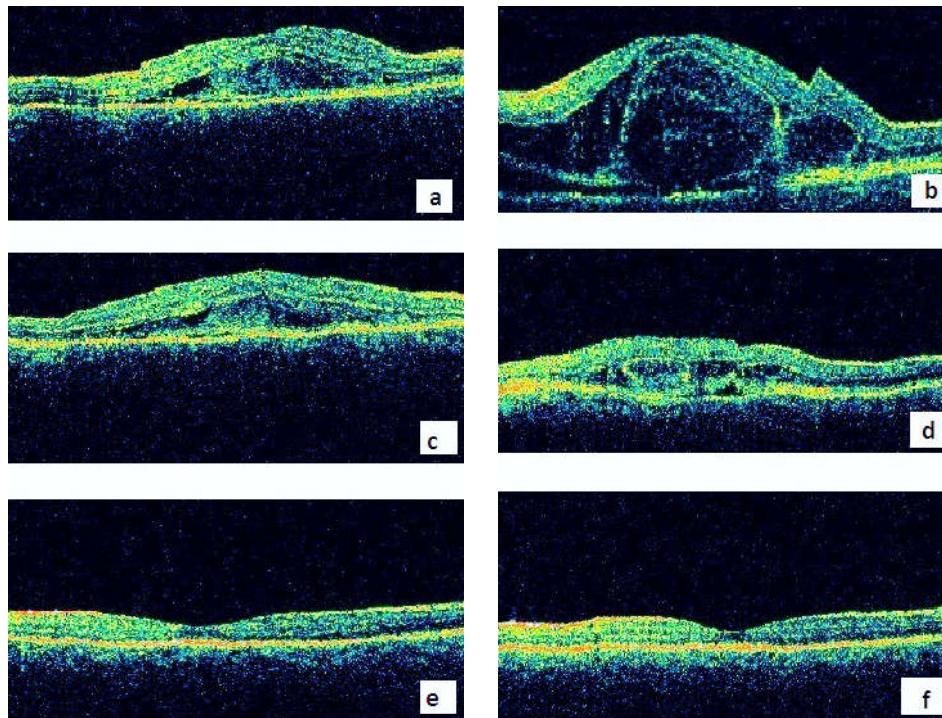


Fig. 3: A&B: Optical coherence tomography both eyes exhibiting exudative retinal detachment, C&D: showing gradual reduction of macular elevation, E&F: showing complete resolution of serous macular elevation with attainment of normal contour with 15 days of high corticosteroid therapy

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.

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

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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.

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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.

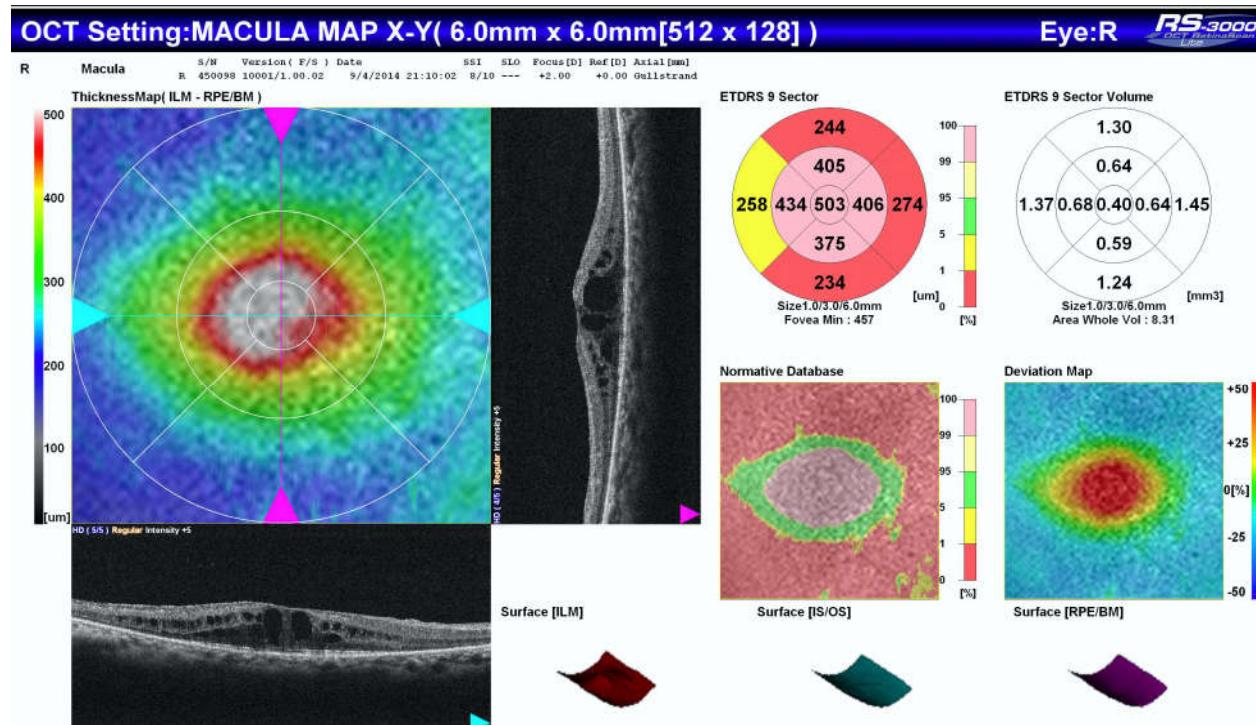


Fig. 1:

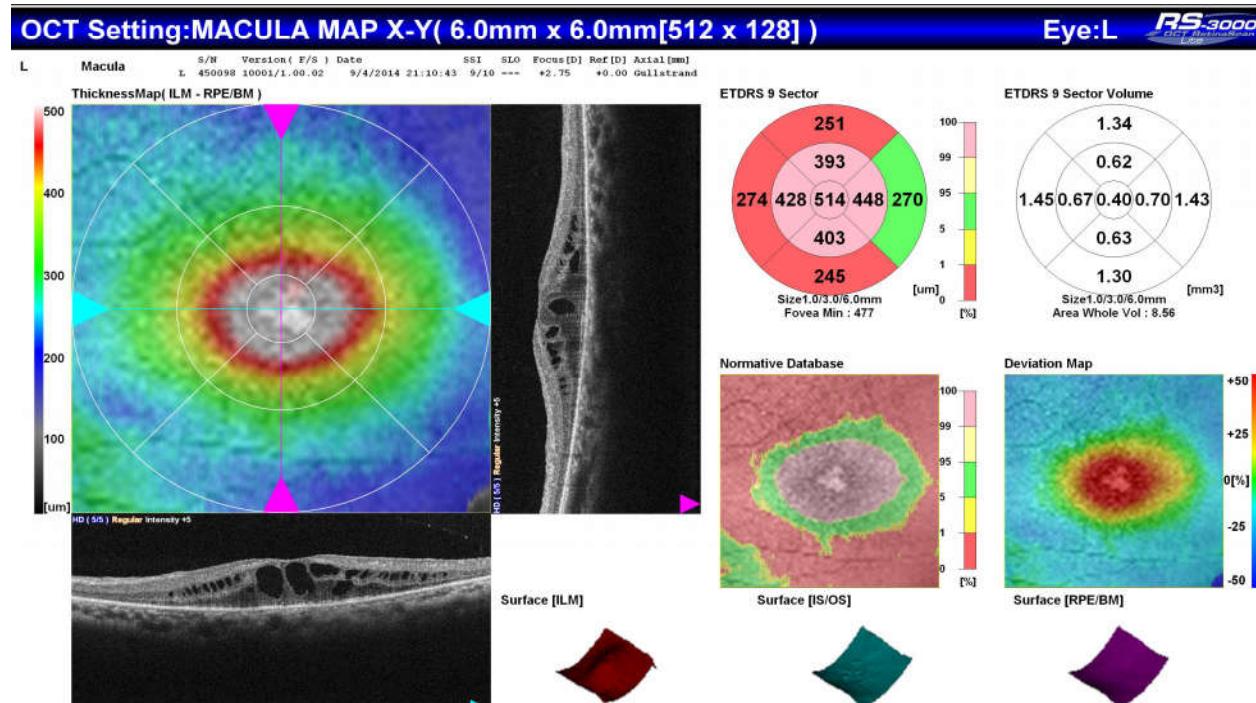


Fig. 2:

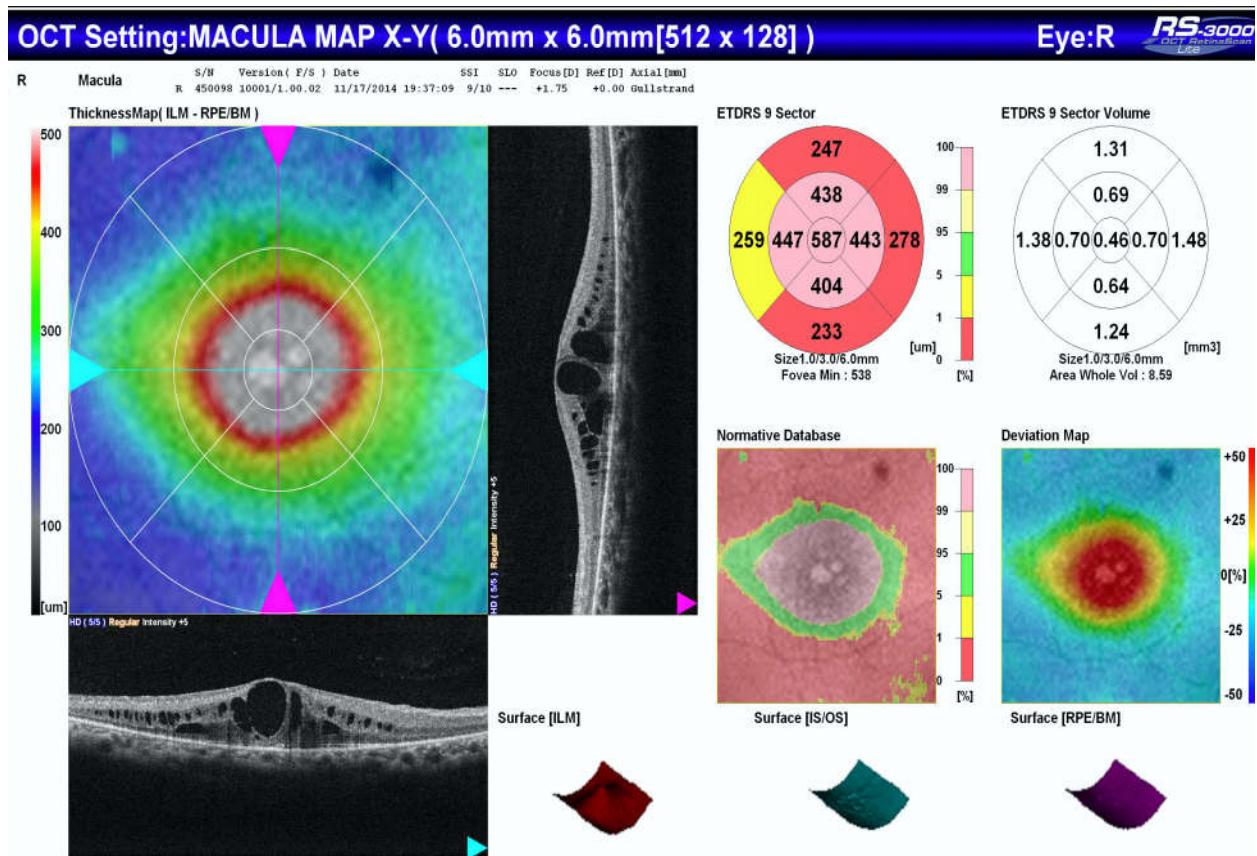


Fig. 3:

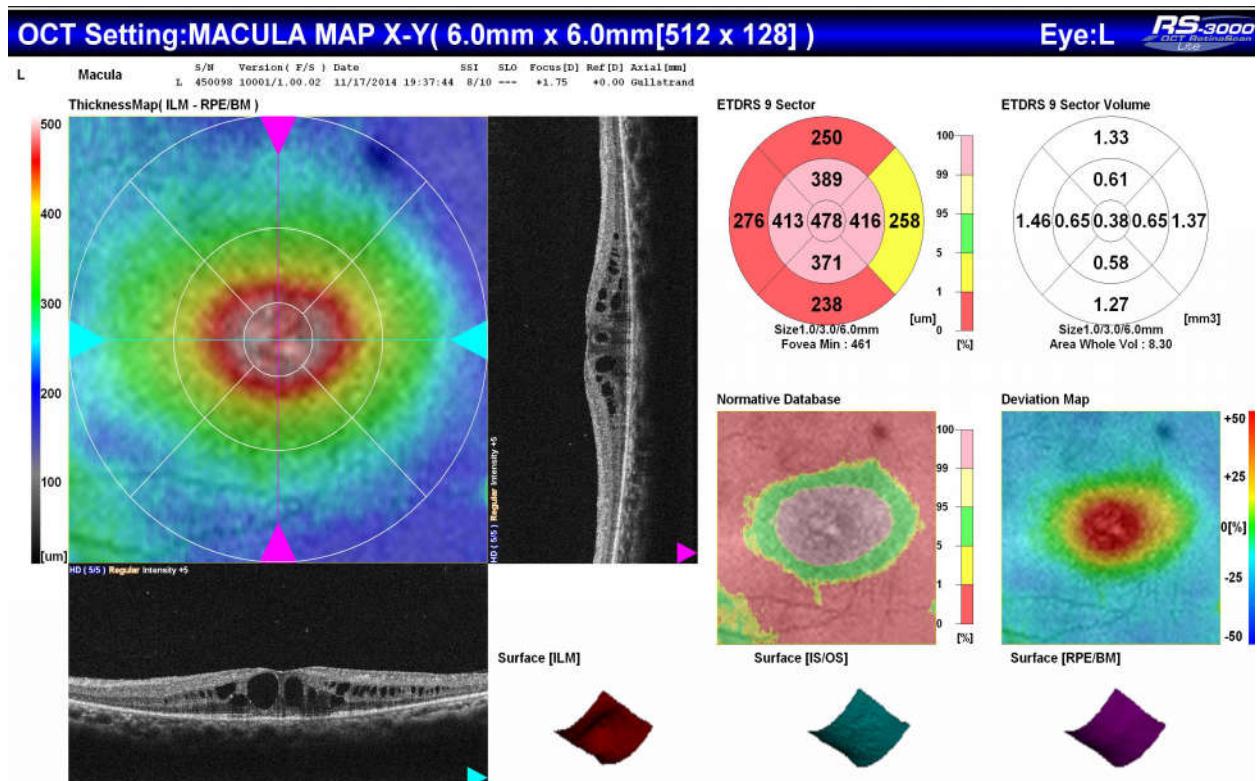


Fig. 4:

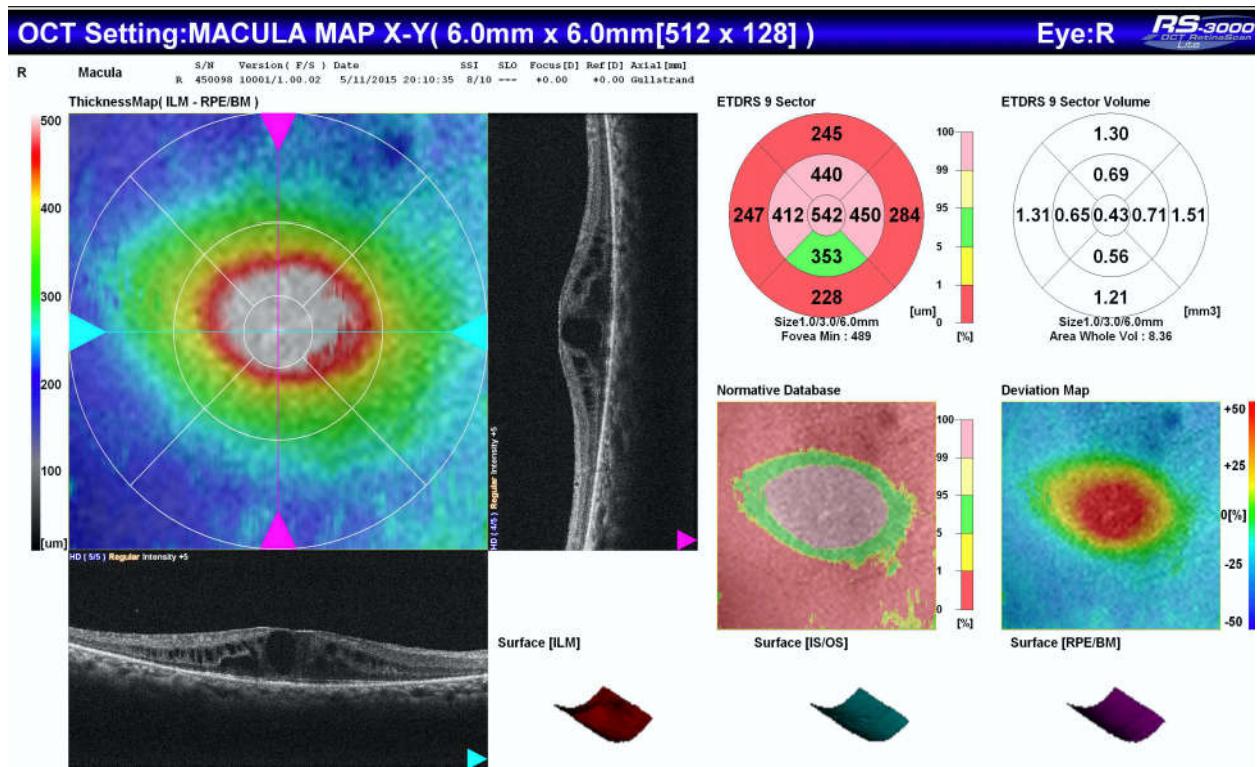


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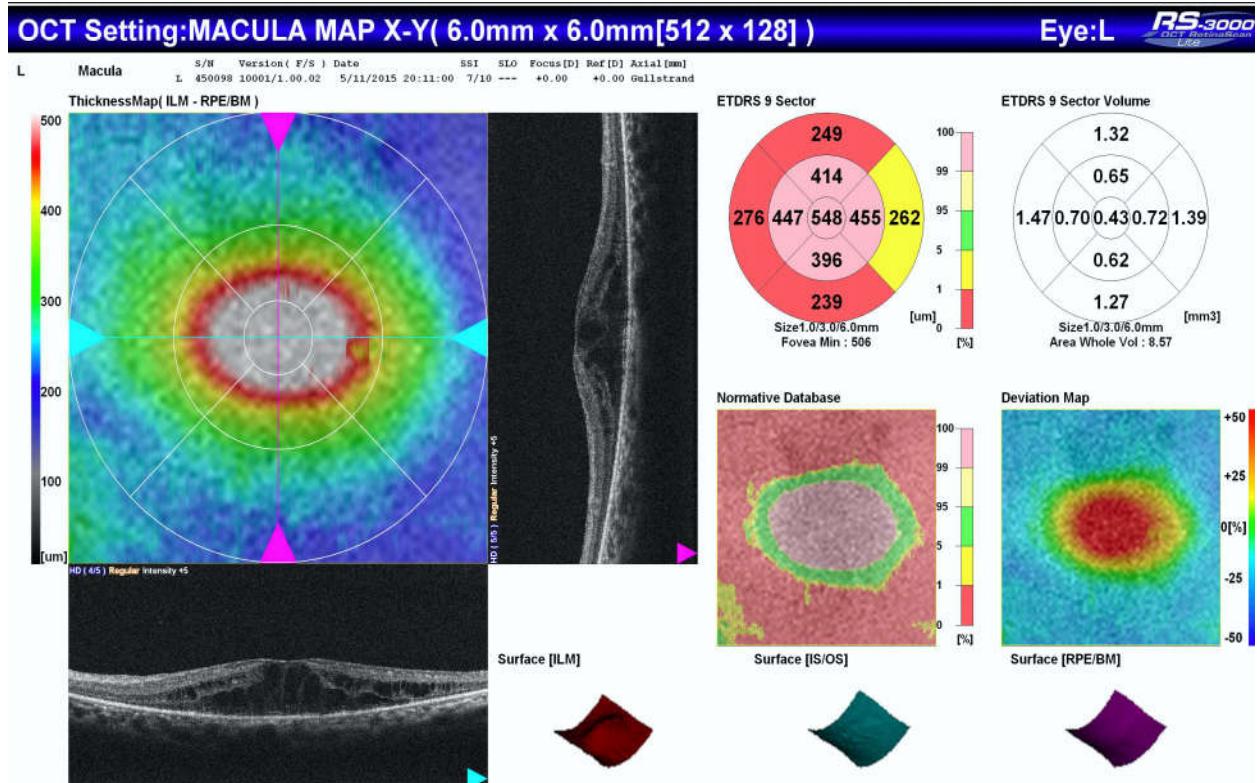


Fig. 6:

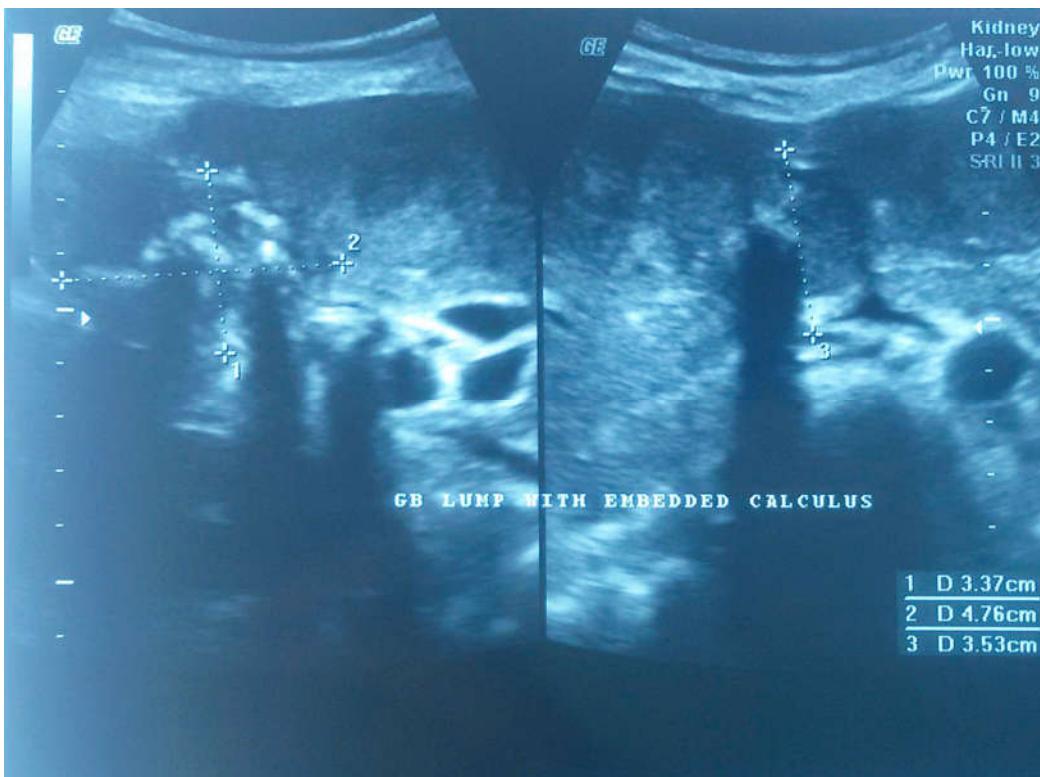


Fig. 7:



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 ecogenicity with embedded calculi (mass measured $3.4 \times 4.8 \times 3.5$ 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.

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A Rare Case of Unilateral Star Shaped Cataract Following Electric Shock

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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.

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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



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

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.

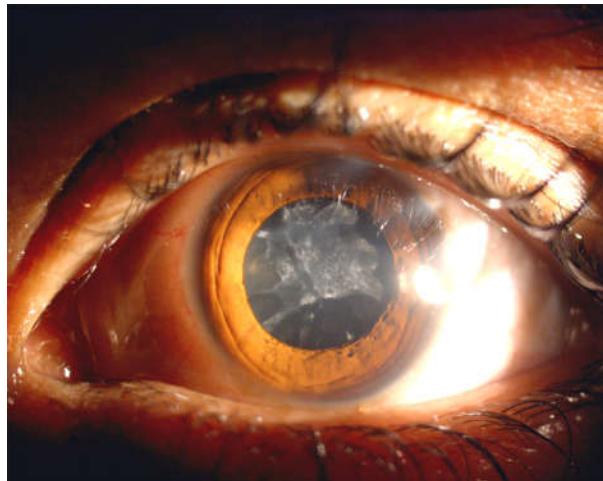


Fig. 2: Star shaped anterior subcapsular cataract

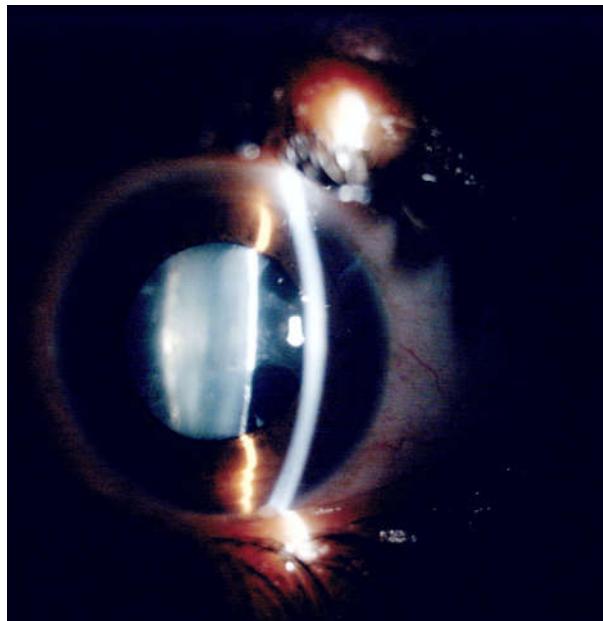


Fig. 3: Anterior and posterior subcapsular cataract

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.

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