

Sympathetic ophthalmitis following vitreoretinal surgery: Does antecedent trauma make a difference?

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Background: Sympathetic ophthalmitis (SO) has been reported following vitrectomy; however, there is a lack of data on the role of antecedent penetrating ocular trauma impacting the disease manifestation in eyes developing SO following vitrectomy. **Aim:** To report differences in the presentation and outcomes of SO in eyes with or without a history of antecedent penetrating trauma; SO being diagnosed after vitreoretinal (VR) surgery. **Design:** Comparative case series. **Methods:** Seventeen consecutive patients presenting with SO following VR surgery, diagnosed between 1995 and 2011 were included. Eyes with and without prior penetrating injury were included in Group I ($n = 7$) and Group II ($n = 10$), respectively. All Group I patients had received systemic steroids prior to presentation. Demographic and clinical parameters were evaluated. **Results:** Differences were observed between Group I and Group II mainly with regards to time interval between VR surgery and diagnosis of SO (1.5 months vs. 8 months, $P = 0.10$), presence of neurosensory detachments (100% vs. 30%, $P = 0.01$), and the inciting eye vision at presentation (nil light perception in 28.5% vs. 80%, $P = 0.049$). Other differences observed though not statistically significant were optic disc and retinal vessel involvement (42% vs. 70%, $P = 0.28$), Dalen-Fuchs nodules (localized vs. diffuse) and leaks on fundus fluorescein angiography (pin-head vs. pin-point leak). **Conclusion:** SO in patients with antecedent penetrating ocular trauma present early with the central serous chorioretinopathy-like picture. Prior use of systemic steroids might have a bearing on the differences in presentation and the visual acuities between the two groups.

Key words: Corticosteroids, immunosuppression, inflammation, sympathetic ophthalmitis, trauma, vitreoretinal surgery

Sympathetic ophthalmitis (SO) is a bilateral diffuse uveitis occurring as a consequence of ocular trauma or surgery in one eye. The eye sustaining the injury or undergoing surgery is called the “inciting” or the “exciting” eye while the contralateral normal eye is called the “sympathizing” eye. Mackenzie^[1] in 1840 and Fuchs^[2] in 1905 provided the earliest description of SO. The possibility of an autoimmune inflammatory response against uveal antigens as the etiology was proposed by Elschnig^[3] in 1910. Prior penetrating injury has been the most common precipitating factor for SO. However, recent studies have reported increase in the incidence following surgical procedures.^[4-10] The etiologic shift from accidental trauma to surgical trauma can probably be explained by the improved access to emergency surgical care following accidental ocular trauma. Gass^[11] has reported SO following vitrectomy and Lewis *et al.*^[12] have reported cases of SO after trauma and vitrectomy. However, there is a lack of data on the role of antecedent penetrating ocular trauma impacting the disease manifestation in eyes developing SO following vitrectomy. In this study of 17 patients with SO following vitreoretinal (VR) surgery, we analyze the impact of antecedent penetrating ocular trauma in disease manifestation and treatment outcomes.

Methods

Review of the medical records of 17 patients presenting with clinical features of SO, between 1995 and 2011 was done.

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Only those patients with a history of VR surgery prior to the episode of SO were included. SO was diagnosed if features of posterior segment involvement in the form of either classic chorioretinal lesions or exudative retinal detachment (RD) or optic disc edema and sunset glow fundus were present, with or without bilateral anterior uveitis.^[4] The minimal diagnostic criteria was the presence of multiple pin-point areas of hyperfluorescence with or without late dye pooling and disc leakage on fundus fluorescein angiography (FFA) and/or the presence of diffuse choroidal thickening of the posterior pole on ultrasound B scan (USG). The patients were divided into two groups; those with prior penetrating injury constituting Group I and those without, constituting Group II.

Collected data included age, sex, presenting complaints, history of antecedent penetrating ocular trauma or any other ocular surgery, timing of the surgical procedures performed, the time interval between the VR surgery(ies) and the onset of symptoms, and the duration of follow-up and the final visual outcome. The clinical parameters recorded included the best corrected visual acuity measured by Snellen’s chart,

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at each visit, anterior and posterior segment manifestations with FFA and ultrasound B-scan features. Therapeutic response to steroids (oral, intravenous, and topical) and immunosuppressants (azathioprine and cyclosporine) and complications during the course of the treatment were also noted. The final visual outcome between the two groups was compared using nonparametric statistical tests. Statistical software (SPSS for Windows, version 13.0 SPSS Science, Chicago, IL, USA) was used for statistical analysis. The statistical significance was assumed at $P \leq 0.05$.

Results

Seven patients were included in Group I, whereas 10 patients were included in Group II. Demographic features of 7 patients in Group I are shown in Table 1. Demographic characteristics of 10 patients in Group II are shown in Table 2. Overall, the mean age at presentation was 37 ± 14.96 years (range 18–65 years); the mean age at presentation in Group I being 30.28 years (range 18–44 years) and that in Group II being 39.4 years (range 20–65 years). Vitrectomy was performed using

Table 1: Demographic features of 7 patients in Group I (VR surgery with antecedent trauma)

Age (years)/sex	Interval between 1° surgery and SO	Indication for VR surgery	Surgeries done in the IE	BCVA at presentation		BCVA at final visit		Follow-up (months)
				IE	SE	IE	SE	
40/male	20 days	VH dislocated nucleus	1°: Scleral wound repair 2°: V + L + BB + EL + SOI	CFCF	CF 1 m	6/60	6/6	8
18/male	15 days	Posttraumatic endophthalmitis	1°: Corneal tear repair 2°: V + L	3/60	2/60	6/18	6/6	24
38/female	11 months	Rheg RD	1°: Corneal tear suturing + L + V + SOI 2°: SB	PL	6/36	NLP	6/6	9
23/male	40 days	Hemorrhagic choroidals	1°: Corneoscleral repair 2°: Choroidal drainage + L + V + BB	CFCF	6/12	HM	6/6	2
44/male	10 months	IOFB	V + IOFB removal + SB	CF 2 m	CF 2 m	NLP	6/6	192
22/male	1 month	IOFB	Corneal wound repair + L + V + IOFB removal+SOI	CF 1 m	6/9	HM	6/6	30
27/male	45 days	IOFB with VH	1°: Scleral tear repair 2°: L + V + RR + SOI	CF 2 m	6/9	PL	6/6	24

VR: Vitreoretinal surgery, 1°: Primary, 2°: Secondary, SO: Sympathetic ophthalmitis, BCVA: Best corrected visual acuity, RD: Retinal detachment, IOFB: Intraocular foreign body, V: Vitrectomy, L: Lensectomy, BB: Belt buckling, EL: Endolaser, SOI: Silicone oil injection, RR: Relaxing retinotomy, SE: Sympathizing eye, IE: Inciting eye, CF: Counting finger, VH: Vitreous hemorrhage, SB: Scleral buckling, Rheg: Rhegmatogenous, m: Meter, CFCF: Counting fingers close to face

Table 2: Demographic characteristics of 10 patients in Group II (VR surgery without antecedent trauma)

Case number	Age (years)/sex	Interval between surgery and SO (months)	Indication for VR surgery	VR surgery (ies) in the IE	BCVA at presentation		BCVA at final visit		Follow-up (months)
					IE	SE	IE	SE	
8	65/male	1	Rheg RD	SB with SRF drainage	CF 2 m	CF 2 m	6/18	6/6	12
9	36/female	24	Rheg RD	Phaco + V + IOL	NLP	6/18	NLP	6/6	16
10	20/male	10	Rheg RD	SB + V + IOL removal	6/36	6/12	NLP	6/9	180
11	22/female	3	Rheg RD	1°: SB 2°: L + V + SOI	PL	6/9	NLP	6/6	60
12	58/female	6	Dislocated nucleus	L + V + SOI	PL	CF 1 m	PL	6/9	8
13	45/male	120	Rheg RD	1°: SB with SRF drainage 2°: Transscleral cryopexy	NLP	6/9	NLP	6/9	12
14	48/male	12	Recurrent Rheg RD	1°: SB with SRF drainage 2°: PPV + SOI 3°: Silicone oil removal	NLP	CFCF	NLP	6/9	24
15	37/male	144	Rheg RD	SB + V + FGE	NLP	6/24	NLP	6/6	60
16	28/male	60	Rheg RD	1°: SB 2°: Infected SB removal	PL	6/6	NLP	6/6	66
17	35/female	1	Rheg RD	1°: BB + L + V + SOI 2°: SOR + Re -V + SOI	PL	6/24	NLP	1/60	12

VR: Vitreoretinal, SO: Sympathetic ophthalmitis, IE: Inciting eye, BCVA: Best corrected visual acuity, CF: Counting finger, PL: Perception of light, NLP: No light perception, SRF: Subretinal fluid, SE: Sympathizing eye, Rheg: Rhegmatogenous, RD: Retinal detachment, SB: Scleral buckling, BB: Belt buckling, V: Vitrectomy, L: Lensectomy, FGE: Fluid gas exchange, SOI: Silicon oil injection, SOR: Silicon oil removal, Phaco: Phacoemulsification, m: Meter

20 g instrumentation in all the patients in the study. Indications for VR surgery are depicted in Fig. 1. Presenting symptoms and their timing of presentation are shown in Table 3. In Group I, 4 (57.1%) eyes underwent repeated surgeries within 2–4 weeks and all of them presented with SO within 1–1.5 months of the last surgery.

All except 1 patient (Case 13) presented in the acute phase of the disease. Clinical presentation in sympathizing eyes (SEs) is shown in Table 4. Patients in Group I predominantly presented with neurosensory detachment [Figs. 2a and 3a] resembling “central serous retinopathy-like picture” ($n = 4$) along with mild anterior uveitis. Massive exudative RD was seen in the other patients ($n = 3$). Dalen-Fuchs nodules were smaller, less numerous, and mostly limited to the posterior pole ($n = 1$). On disease resolution, atrophic retinal pigment epithelial (RPE) changes were observed more at the macula [Fig. 2b]. However, patients in Group II presented with more significant anterior uveitis along with the posterior segment findings ($n = 9$). Classical granulomatous uveitis with large mutton fat keratic precipitates were seen in 2 patients. However, iris nodules were not seen in any of the patients. Posterior segment manifestations included disc hyperemia/edema ($n = 9$), peripapillary choroidal nodules ($n = 7$), and retinal vascular caliber changes ($n = 5$) [Figs. 4a and 5a]. Dalen-Fuchs nodules were numerous, coalescent, and present diffusely over the posterior pole and extending to the periphery as well ($n = 3$). On disease resolution, RPE atrophy was classically seen in the peripapillary area [Fig. 5b]. Thus, the Group I eyes differed from Group II eyes mainly with respect to the presence of neurosensory detachments (100% vs. 30%, $P = 0.01$). Other differences noted though not statistically significant were disc and vessel involvement (42% vs. 70%, $P = 0.28$), Dalen-Fuchs nodules (localized vs. diffuse) and the areas of RPE atrophy on resolution (macular vs. peripapillary).

Though in small numbers, distinct fluorescein angiographic features were noted between the two groups. SEs in Group I revealed RPE leakages ($n = 7$). The RPE leaks were larger [Figs. 2c and 3b], associated with late pooling of dye ($n = 5$) [Figs. 2d and 3c, d], and usually confined to the posterior pole (except in 3 patients where the leaks were present beyond the equator). Retinal vessel changes and peripapillary

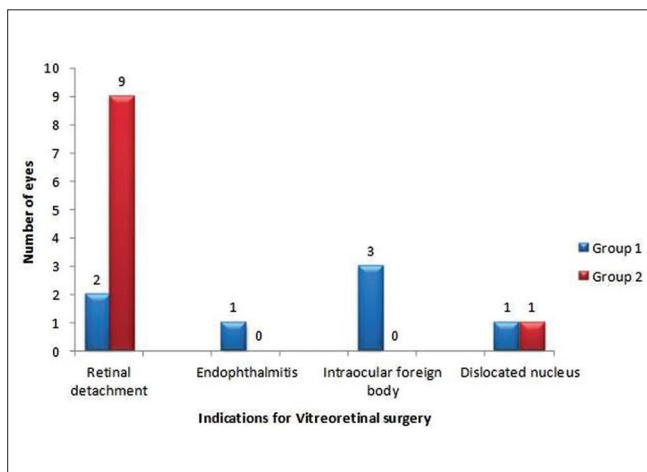


Figure 1: Bar diagram depicts the indications for vitreoretinal surgeries in 17 eyes that developed sympathetic ophthalmitis following surgery

hyperfluorescence were conspicuously absent. In contrast, Group II SEs predominantly showed early hypofluorescence and late hyperfluorescence in the area corresponding to the peripapillary choroidal nodules ($n = 7$) [Figs. 4b-d and 5c, d], disc leakage in eyes with disc edema/hyperemia ($n = 9$), segmental staining of the retinal veins and arterioles ($n = 2$) [Fig. 5c,d], retinal venous beading and tortuosity ($n = 6$), and RPE leaks that were pin-point, multiple, and scattered all over the postpole extending beyond arcades up to the equator. Pooling of dye was seen in a limited number of patients ($n = 2$).

Medical management, duration of treatment, follow-up, and recurrence of inflammation are shown in Table 5. Oral

Table 3: Differences in presenting symptoms and timing of presentation in the two groups

	Group I (n=7)	Group II (n=10)
Symptoms (%)		
Diminution of vision	5 (71.4)	9 (90)
Headache	0	4 (40)
Eye pain and redness	0	3 (30)
Metamorphopsia	3 (42)	0
Floaters	3 (42)	0
Timing of presentation		
Duration of symptoms	Median 15 days (range: 2-90 days)	Median 22.5 days (range: 2-240 days)
Interval between VR surgery and onset of SO	Median 1.5 months (range: 10 days-11 months)	Median 8 months (range: 20 days-12 years)

VR: Vitreoretinal, SO: Sympathetic ophthalmitis

Table 4: Clinical presentation in SEs in the two groups

Clinical features	Group I (n=7) (%)	Group II (n=10) (%)
Anterior segment findings		
Absent/mild anterior nongranulomatous uveitis	5 (71)	4 (40)
Anterior nongranulomatous uveitis	1 (14)	4 (40)
Granulomatous uveitis	1 (14)	2 (20)
Posterior segment findings		
Vitritis	4 (57)	6 (60)
Neurosensory detachment	7 (100)	3 (30)
Disc involvement	4 (57)	7 (70)
Subretinal infiltrates	2 (28.5)	4 (40)
Fundus fluorescein angiography		
FFA performed	6 (85.74)	7 (70)
Pin-point hyperfluorescence	6 (100)	5 (71.4)
Disc leakage	3 (50)	6 (85.74)
Retinal vessel wall staining	2 (28.5)	3 (42.8)
Ultrasound		
Ultrasound B scan performed	6 (85.74)	9 (90)
Exudative RD	6 (100)	5 (71.4)
Increased choroidal thickness	3 (50)	7 (77.7)

SEs: Sympathizing eyes, RD: Retinal detachment, FFA: Fluorescein angiography

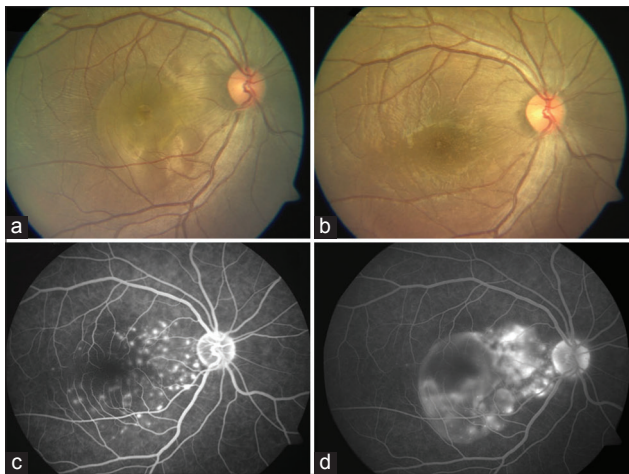


Figure 2: (a) Color fundus photograph of the right eye of a Group I patient showing a neurosensory detachment at the macula without any retinal vascular changes: A central serous retinopathy like picture. (b) Color fundus photograph 1 month after treatment. Resolution of subretinal fluid, with retinal pigment epithelial changes at the macula are noted. (c) Early arteriovenous phase and (d) late arteriovenous phase: Fluorescein angiography showing numerous pronounced retinal pigment epithelial leaks which are bigger and confined to the macula with late pooling of dye

Table 5: Medical management: Therapeutic agents, duration of treatment, follow-up and recurrence of inflammation in the two groups

Treatment details	Group I (n=7) (%)	Group II (n=10) (%)
Drugs		
Oral prednisolone (1.5-2 mg/kg/day; tapered)	7 (100)	9 (90)
Intravenous methyl prednisolone (1 g IV for 3 days, followed by oral steroids)	6 (85.7)	5 (50)
Additional immunosuppressives	6 (85.7)	8 (80)
Azathioprine (50 mg TID - 1 month; tapered)		
Cyclosporine (175 mg BID; tapered)		
Average duration of treatment (months)	7.8	9
Follow-up period	Median 24 months	Median 34 months
Eyes with recurrence of inflammation	1 (14.7)	4 (40)

IV: Intravenous, TID: Three times a day, BID: Twice a day

and topical corticosteroids were the mainstay of treatment. Treatment with oral steroids was initiated with a dose of 1.5–2 mg/kg, tapered and followed-up with a maintenance dose of 5–10 mg/day. Multiple drug combinations were found necessary to control the inflammation in patients with inciting eyes (IEs) having \leq PL vision in Group II and in all patients (except 1 who presented in the resolving stage of the disease) in Group I. Eyes with recurrent inflammation were managed with repeat cycles of immunosuppressive and systemic steroid therapy. No light perception (NLP) was noted in 2 (28.5%) and 8 (80%) of the IEs in Group I and II, respectively ($P = 0.049$). The final visual acuity in the SE was observed to be better in Group I, though not statistically significant (100% vs. 70%, $P = 0.33$) [Fig. 6].

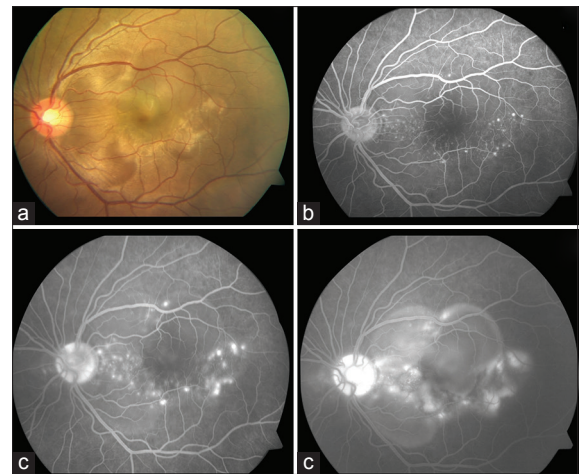


Figure 3: (a) Color fundus photograph of the left eye of a Group I patient showing multiple pockets of subretinal fluid with internal limiting membrane striae. (b) Early arteriovenous phase, (c) late arteriovenous phase and (d) late phase: Fluorescein angiography showing numerous retinal pigment epithelial leaks which increase in size with pooling of dye in late phase

Two SEs (Case numbers 16, 17) in Group II showed visual deterioration due to secondary glaucoma and optic atrophy, respectively.

With respect to the IEs, 5 (71.4%) eyes in Group I showed deterioration of visual acuity while 1 eye remained same and 1 improved. Two eyes that worsened were prephthisical at presentation, 1 had secondary glaucoma with optic atrophy, 1 had second insult to IE in the form of another penetrating trauma and 1 developed recurrent rhegmatogenous RD. The final visual acuity in the IEs in Group II eyes remained the same in 6 (60%) eyes, worsened in 3 (30%) eyes, and improved in 1 (10%) eye. Among the 3 eyes that worsened, 1 developed secondary angle closure glaucoma eventually resulting in optic atrophy. The other 2 eyes were prephthisical at presentation and became phthisical with time. Treatment-related complications have been listed in Table 6.

Discussion

SO following VR surgery has been reported previously.^[7,9-13] There are some reports of SO developing following VR surgery in the setting of antecedent trauma and few without.^[7,10,11,13] In our series, we tried to study and analyze the presentations and outcomes in both the groups. Patients with antecedent penetrating trauma were seen to present early and had better visual outcomes with treatment.

In patients with multiple surgeries, it was the subsequent (postprimary repair) VR surgery that was seen as the inciting factor in 71.4% patients in Group I and 50% patients in Group II. This aspect has also been reported by Tamai *et al.*^[13] This could be explained by the additional insults imposed by the multiple surgeries. Thus, any kind of “recurrent” surgical manipulation in the IE can act as a trigger for SO.^[6,11] We found an earlier presentation (median 1.5 months) in Group I in comparison to Group II, though not statistically significant. Galor *et al.*^[14] also found that trauma-inflicted patients presented earlier than surgically induced SO patients.

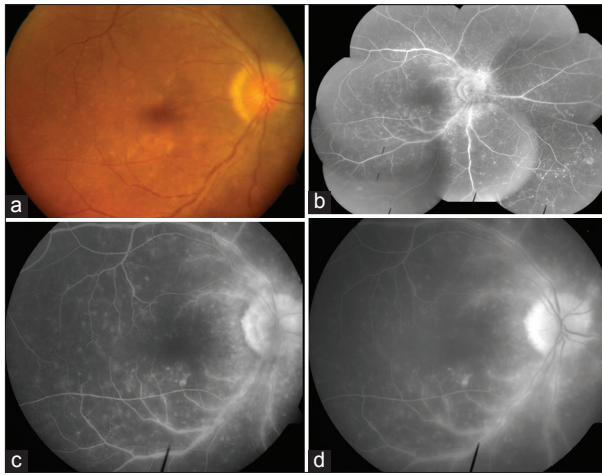


Figure 4: (a) Fundus photograph of a Group II patient showing prominent peripapillary choroidal nodules, retinal vascular tortuosity and yellowish-white subretinal infiltrates scattered over the posterior pole. (b) Fundus fluorescein angiography montage of the late arteriovenous phase showing hyperfluorescent dots corresponding to the yellowish-white subretinal nodules scattered all over the periphery with hyperfluorescence in the area corresponding to the peripapillary choroidal nodules. (c) Late venous phase and (d) recirculation phase showing disc leak and staining of the retinal veins

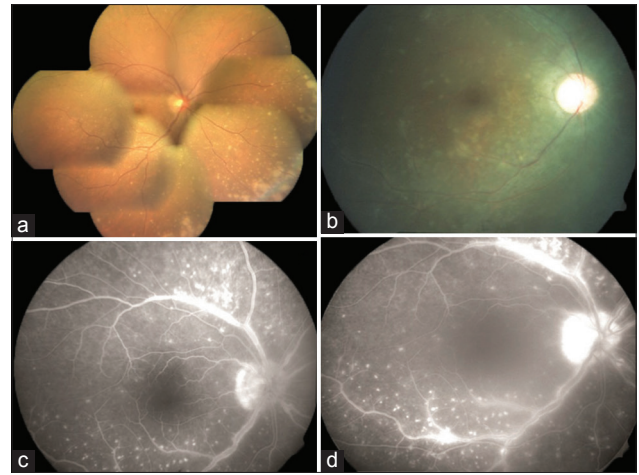


Figure 5: (a) Fundus (montage) picture of a Group II patient showing peripapillary choroidal nodules, retinal vascular tortuosity, and venous beading with yellowish-white subretinal infiltrates scattered over the posterior pole and periphery. (b) Color fundus photo of same patient after disease resolution showing retinal and optic atrophy. (c) Early arteriovenous phase and (d) late arteriovenous phase: Fluorescein angiography showing numerous hyperfluorescent dots corresponding to the yellowish-white subretinal nodules with early hypofluorescence and late hyperfluorescence in the area corresponding to the peripapillary choroidal nodules along with disc leakage and segmental staining of the retinal veins

Table 6: Treatment related complications in the two groups

Treatment related complications	Group I (n=7) (%)	Group II (n=10) (%)
Systemic		
Systemic hypertension	0	3 (30)
Diabetes mellitus	0	2 (20)
Obesity	1 (14.2)	2 (20)
Avascular necrosis of femur	0	1 (10)
Raised liver enzymes	2 (28.5)	0
Ocular		
Secondary glaucoma	3 (42.8)	4 (40)
Complicated cataract	2 (28.5)	2 (20)
Retinal and optic atrophy	0	1 (10)
Peripapillary atrophy	0	3 (30)

However, patients in Group II had varied presentation with a median of 8 months (range 20 days to 12 years) similar to the study by Pollack *et al.*^[7]

Both groups were similar with respect to the presenting visual acuity in the SEs but differed in the presenting vision among IEs ($P=0.029$), which was significantly better in Group I. This may be due to longstanding posterior segment disease in Group II and also since 4 eyes were prephthisical. Better visual acuity in Group I patients could also be explained by the earlier presentation. 40% patients presented with NLP in the IEs in Group II. The clinical features in eyes from Group II involved mainly the optic disc, peripapillary area, and the retinal vessels, whereas neurosensory detachments at the macula were conspicuous in Group I eyes. This is in contrast with the findings by Pollack *et al.*^[7] where anterior segment involvement was seen in 75% patients. Predominant

involvement of the posterior segment has also been observed in a study on the Asian-Indian population by Gupta *et al.*^[15] Isolated neurosensory detachment in Group I may easily be confused with central serous chorioretinopathy. A plausible explanation for this presentation is that these patients were treated with oral steroids at/before the presentation, which could have suppressed the inflammatory changes in the posterior segment leading to a limited disease manifestation. It is imperative to consider this differential in such eyes as patients with penetrating ocular injury are frequently treated with systemic steroids and the possibility of central serous retinopathy (CSR) masquerading as SO^[16] or vice versa cannot be ruled out. Differentiating between the two is even more essential from the treatment point of view as SO requires initiation of steroid therapy, whereas CSR warrants its discontinuation.

All patients in Group I (except 1) required additional immunosuppression with oral steroids, whereas IEs with \leq PL vision ($n=8$) in Group II required additional immunosuppression. This may imply that badly damaged and phthisical IEs require more aggressive management. Our treatment regime was comparable to that used by Su and Chee.^[6] However, despite different treatment regimens the visual outcomes were good in the SEs in about 88% of the patients. The effect of the surgical outcome of cataract extraction in SEs has been reported earlier.^[14,17] In our study too, we found that patients who underwent cataract extraction or any other ocular surgery, in the sympathizing or the IEs, in the quiescent phase of the disease under steroid cover did reasonably well. However, surgeries on the IEs during the active phase of the disease were associated with recurrences of SO.

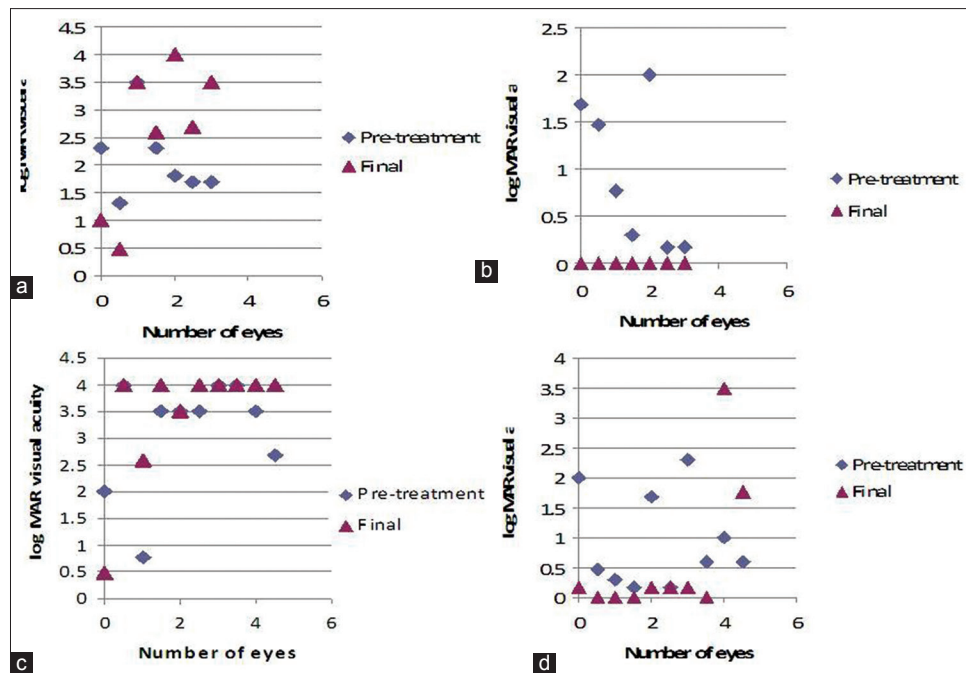


Figure 6: Scattergram showing the pretreatment and final visual acuity in the inciting eyes (a) and the sympathizing eyes (b) in Group I and the inciting eyes (c) and sympathizing eyes in Group II (d)

According to Galor *et al.*,^[14] traumatic cause, exudative RD, and active inflammation were associated with poor visual outcome. A more severe course with traumatic SO theoretically may be the result of high-doses of antigenic exposure.^[18] Patients treated promptly with prednisone and/or immunosuppression were more likely to achieve quiescence and seemed to do so more quickly.^[4-6]

Poor visual outcome was observed in the inciting and SEs in Group II as compared to Group I. Whether this difference could have been due to the earlier presentation in the trauma group, cannot be inferred. Our study sample size is not large enough to conclude a statistically significant difference in the presentation and the outcome; however, we still feel that patients with multiple surgeries, who are at higher risks of developing SO, if detected and managed at the earliest may give a good visual outcome.

Kilmartin *et al.*^[4] argued that early enucleation did not affect the visual outcome. In our study, eyes which were advised enucleation did not have any recurrences throughout the follow-up. Recurrences and severe inflammations when present were managed by stepping up the doses of steroids and use of additional immunosuppressive agents.

Conclusion

Persistent, low-grade uveitis, or isolated posterior segment features following VR surgery should alert the ophthalmologist to the possibility of SO. SO patients with antecedent penetrating trauma present early with a CSR-like picture. Prior use of systemic steroids might have a bearing on the clinical presentation and treatment outcome. The presence of superadded infection/further surgical insults to the IE in the active phase of inflammation is likely to be associated with multiple recurrences of SO and poor visual prognosis in the IEs.

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Conflicts of interest

There are no conflicts of interest.

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