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Original Article

Sympathetic Ophthalmia: Demographic Characteristics, Clinical Findings, and Treatment Results

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Abstract

Objectives: To evaluate the demographic characteristics, clinical findings, and treatment approach of patients with sympathetic ophthalmia (SO).

Materials and Methods: The records of 14 patients with SO between 2000 and 2020 were retrospectively reviewed. The patients' Snellen best corrected visual acuity (BCVA), detailed ophthalmological examination, optical coherence tomography (OCT), enhanced depth imaging-OCT (EDI-OCT), fundus fluorescein angiography findings, and treatment approaches were recorded.

Results: The study included the 14 sympathizing eyes of 14 patients with SO (7 female, 7 male). The mean age was 48.5±15.4 years (range: 28-75), and the mean follow-up duration was 55.1±48.7 months (range: 6-204). Ten patients (71%) had a history of ocular trauma and 4 (29%) had a history of ocular surgery. The time to symptom onset in the sympathizing eye after trauma or ocular surgery ranged from 15 days to 60 years. The most common posterior segment findings were optic disc edema (36%) and exudative retinal detachment (36%). In the acute period, the mean choroidal thickness value on EDI-OCT was 716.5±63.6 µm (range: 635-772) and decreased to 296±81.6 µm (range: 240-415) after treatment. Treatment with high-dose systemic corticosteroid was given to 8 patients (57%), azathioprine (AZA) to 7 (50%), AZA and cyclosporine-A combination to 7 (50%), and tumor necrosis factor-alpha inhibitors to 3 patients (21%). Recurrence was observed in 4 patients (29%) during follow-up. At last follow-up, BCVA values were better than 20/50 in 11 (79%) of the sympathizing eyes. Remission was achieved in 13 patients (93%), but 1 patient (7%) lost her vision due to acute retinal necrosis.

Conclusion: SO is a bilateral inflammatory disease that presents with granulomatous panuveitis after ocular trauma or surgery. Favorable functional and anatomical results can be obtained with early diagnosis and initiation of appropriate treatment.

Keywords: Imaging, optical coherence tomography, sympathetic ophthalmia, treatment, Vogt-Koyanagi-Harada

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Introduction

Sympathetic ophthalmia (SO) is a bilateral granulomatous panuveitis occurring after trauma or ocular surgery of one eye. Although its pathogenesis is not completely known, it is thought to result from an autoimmune hypersensitivity reaction to the eye itself.[1] The traumatized or operated eye is referred to as the "sympathetic" or "inciting" eye and the other as the "sympathizing" eye.[2] SO can develop within the first few days after ocular injury or surgery or even years later. Cases developing between 5 days and 66 years later have been reported in the literature.[2,3] Penetrating trauma was previously considered to be the main cause of SO. However, intraocular surgical procedures, especially vitreoretinal surgeries, have been recognized as an important risk factor in recent years.[2,4] Cases of SO have also been reported after intravitreal injection, laser iridotomy, cryocyclodestructive procedures, plaque therapy, and rarely after fungal keratitis.[1,2]

As SO is a rare disease, most studies conducted to date have documented case series. Therefore, its incidence has been reported in different studies at rates varying between 0.2% and 0.5% following injury, and 0.01% after intraocular surgery.[1] In this study, we aimed to evaluate the clinical and demographic characteristics, treatment, and visual results of patients diagnosed with SO in a tertiary referral hospital.

Materials and Methods

The records of 14 patients who were followed for SO between 2000 and 2020 in the uvea unit of the University of Health Sciences Türkiye Ulucanlar Eye Training and Research Hospital were evaluated retrospectively. This study was conducted in accordance with the principles of the Declaration of Helsinki. Ethics approval was obtained from the Ankara Training and Research Hospital Ethics Committee. The diagnosis of SO was made in the presence of typical clinical findings and a history of trauma and/or surgery after the exclusion of other known causes.[5,6]

The diagnostic criteria for SO included mutton-fat keratic precipitates (KPs), cells and flare in the anterior segment, and acute stage findings (multifocal retinochoroidal nodular lesions, choroidal thickening, vitritis, papillitis, exudative retinal detachment [RD]) and chronic stage findings (subretinal fibrosis, optic and retinochoroidal atrophy, multiple yellow-white round subretinal lesions located in the mid-equatorial region [Dalen-Fuchs (D-F) nodules], and sunset glow fundus) in the posterior segment. Other causes of granulomatous uveitis such as syphilis, tuberculosis, sarcoidosis, and Vogt-Koyanagi-Harada (VKH) disease were considered in the differential diagnosis and ruled out with relevant clinical and laboratory examinations when necessary.

History of trauma or ocular surgery, demographic data such as age and gender, follow-up duration, detailed ocular examination findings (Snellen visual acuity, slit-lamp examination, applanation tonometry, and dilated fundoscopy), fundus fluorescein angiography (FA), optical coherence tomography (OCT), enhanced depth imaging-OCT (EDI-OCT) findings, and applied treatments were retrieved from the patients' medical files. Initial and final best corrected visual acuities (BCVA) were recorded. All patients underwent regular blood counts and liver and kidney function tests during follow-up. Indocyanine green angiography imaging could not be performed.

Statistical Analysis

Data were analyzed using the SPSS version 22.0 software (IBM Corp., Armonk, NY, USA). Qualitative data were expressed as percentages and quantitative data as mean ± standard deviation. Frequency and percentage comparisons of categorical variables were made using the Pearson chi-square test and comparisons of group means were made with the Mann-Whitney U test.

Results

The study included 14 eyes of 14 SO patients, comprising 7 (50%) women and 7 (50%) men. The mean age at admission was 48.5±15.4 years (range: 28-75). The mean follow-up duration was 55.1±48.7 months (range: 6-204). The demographic and clinical characteristics of the patients are presented in Table 1. Eight patients (57.1%) had a history of penetrating eye injury and repair, and 2 patients (14.3%) had blunt eye trauma. Of the patients with blunt eye trauma, 1 (7.1%) (case 10) had undergone vitreoretinal surgery due to RD development and 1 (7.1%) (case 3) had undergone cataract surgery during follow-up. Of the remaining 4 patients (28.6%), 1 (7.1%) had a history of vitreoretinal surgery and 3 (21.4%) of evisceration surgery (due to endophthalmitis, congenital cataract surgery, and uveitis). The mean ages of patients who developed posttraumatic and postsurgical SO were 49.6±17.1 years and 46.0±11.6 years, respectively. There was no statistically significant difference in terms of age or gender between patients with posttraumatic and postsurgical SO (p=0.571 and p=0.559, respectively). The inciting eye was the left eye in 7 (50.0%) and the right eye in 7 (50.0%) of our patients. The time from trauma or ocular surgery to the onset of symptoms in the sympathizing eye ranged from 15 days to 60 years, with a mean of 202.6±289.0 months (range: 0.5-720). The time to onset of SO was 211.1±278.4 months after trauma and 181.1±359.0 months after ocular surgery. There was no statistically significant difference between the time to symptom onset after trauma and ocular surgery (p=0.352). Extraocular findings were not observed in any patient.

The initial BCVA of the sympathizing eyes was 20/25 in 4 eyes (28.6%), 20/32 in 1 eye (7.1%), 20/100 in 2 eyes (14.3%), and 20/200 in 7 eyes (50.0%) (Table 1). Among the posttraumatic sympathizing eyes (Figure 1a), initial BCVA was 20/25 in 2 eyes (14.3%), 20/32 in 1 eye (7.1%), 20/100 in 1 eye (7.1%), and 20/200 in 6 eyes (42.8%). In the postsurgical sympathizing eyes, initial BCVA was 20/25 in 2 eyes (14.3%), 20/100 in 1 eye (7.1%), and 20/200 in 1 eye (7.1%) (Table 1).

BCVA in the sympathetic eye (Figure 1b, 2a, 3a) was at the level of hand movements in 1 patient (7.1%) and light perception in 4 patients (28.6%), while 9 patients (64.3%) had no light perception.

Table 1. Clinical and demographic characteristics of patients

Patient/ sex	Age	Type of injury	Time to development of SO after trauma/surgery	Initial visual acuity in Snellen		Clinical features of the sympathizing eye at presentation		Duration of follow-up	Final visual acuity in Snellen
				Sympathetic eye	Sympathizing eye	Anterior chamber	Fundus		Sympathizing eye
1/M	68	Penetrating trauma	60 years	NLP	20/200	Active Granulomatous KPs, 1+ cells	Serous RD, disc edema	14 months	20/32
2/M	75	Penetrating trauma	35 years	LP	20/32	Granulomatous KPs	D-F nodules	9 years	20/25
3/M	44	Blunt trauma and cataract surgery	30 years	NLP	20/200	-	Serous RD, disc edema	15 months	20/25
4/M	68	Penetrating trauma	2 years	NLP	20/25	Granulomatous KPs	Sunset glow fundus	2 years	20/63
5/F	58	Vitrectomy surgery	4 months	LP	20/200	Active Granulomatous KPs, 2+ cells	VH 2+	21 months	20/40
6/M	43	Penetrating trauma	8 years	LP	20/200	-	Sunset glow fundus, D-F nodules	2 years	20/25
7/F	62	Penetrating trauma	15 days	NLP	20/200	Active granulomatous KPs, 1+ cells	VH 2+	7 years	NLP
8/F	29	Penetrating trauma	10 months	NLP	20/25	Active granulomatous KPs, 1+ cells	Vitritis 2+, serous RD, disc edema	7 years	20/20
9/F	42	Penetrating trauma	1 month	LP	20/200	Granulomatous KPs	D-F nodules	17 years	20/800
10/M	37	Blunt trauma and vitreoretinal surgery	21 months	NLP	20/100	-	Serous RD, disc edema	2 years	20/20
11/F	54	Evisceration	1 month	NLP	20/25	-	Sunset glow fundus	6 months	20/25
12/F	28	Penetrating trauma	5 years	HM	20/200	-	Serous RD, disc edema	6 years	20/50
13/M	36	Evisceration	1 month	NLP	20/25	1+ cells	D-F nodules	14 years	20/20
14/F	66	Evisceration	60 years	NLP	20/100	Active granulomatous KPs, 2+ cells	VH 2+	6 months	20/25

SO: Sympathetic ophthalmia, M: Male, F: Female, NLP: No light perception, LP: Light perception, HM: Hand movements, KPs: Keratic precipitates, RD: Retinal detachment, VH: Vitreous haze, D-F: Dalen-Fuchs

Anterior segment inflammation was observed in 9 (64.3%) of the 14 sympathizing eyes at admission (anterior chamber cells in 1 eye, active granulomatous KPs and anterior chamber cells in 5 eyes, and pigmented mutton-fat KPs in 3 eyes). In the dilated fundus examination at presentation, 8 eyes (57.1%) had acute stage findings (Figure 1c-e, 2b-d) and 6 eyes (42.9%) had chronic stage findings (Figure 3b-e). Acute stage findings included optic disc edema in 5 eyes (35.7%) and exudative

macular detachment in 5 eyes (35.7%) (Figure 1c, 2c), 2+ vitreous haze in 3 eyes (21.4%), and 2+ vitritis without vitreous haze in 1 eye (7.1%). Chronic stage findings consisted of D-F nodules in 4 eyes (28.6%) and sunset glow fundus in 3 eyes (21.4%) (Figure 3b-e).

Fundus FA findings in the acute stage included multiple vascular leakage areas in 2 eyes (14.3%), optic disc hyperfluorescence in 5 eyes (35.7%), and dye pooling in 5 eyes (35.7%) with exudative RD (Figure 1d, 2d). Serous macular detachment and choroidal folds were detected on OCT in 5 eyes (35.7%) (Figure 1e, 2b). The mean choroidal thickness values at initial and last visits were 716.5±63.6 µm (range: 635-772) and 296±81.6 µm (range: 240-415), respectively, in patients with acute stage disease. In patients with chronic stage disease, mean choroidal thicknesses at initial and last visits were 302.0±9.8 µm (range: 295-309), and 258.3±64.6 µm (range: 166-341), respectively.

Topical corticosteroid (CS) (prednisolone acetate, Allergan Pharmaceuticals, Co., Mayo, Ireland) and cycloplegic (cyclopentolate HCL, Abdi Ibrahim Pharmaceuticals, Istanbul, Türkiye) drops were given to patients with anterior segment inflammation. The 8 patients (57.1%) who presented with acute stage disease were started on either 1 mg/day intravenous pulse methylprednisolone (Mustafa Nevzat Pharmaceuticals, Istanbul, Türkiye) (first 3-5 days) followed by oral treatment or high-dose (1.5-2 mg/kg/day) oral methylprednisolone (Mustafa Nevzat Pharmaceuticals, Istanbul, Türkiye). Based on the clinical findings during follow-up, the dosage of systemic CS therapy was gradually reduced by 5-10 mg per week for 6 months. All of our patients received systemic immunosuppressive agents simultaneously with CS therapy. Seven patients (50.0%) received azathioprine (AZA; Aspen Port Elizabeth [Pty] Pharmaceuticals, Port Elizabeth, South Africa) (100-150 mg/day) and 7 patients (50.0%) received AZA (100-150 mg/day) and cyclosporine-A (CSA; Novartis Pharmaceuticals, Eberbach, Germany) (200-300 mg/day) combination therapy. Systemic treatment was switched to the anti-tumor necrosis factor-a (TNF-a) agents infliximab (5-10 mg/kg) (Merck Sharp Dohme Pharmaceuticals, Singapore) in 2 patients (14.3%) and adalimumab (40 mg every 2 weeks) (AbbVie Pharmaceuticals, Ravensburg, Germany) in 1 patient (7.1%) due to refractory and/or recurrent inflammation despite combined immunosuppressive therapy (cases 6, 7, and 11).

Recurrence was observed in 4 patients (28.5%) during the first 4 months of follow-up. Optic disc edema, serous macular detachment, and vitreous haze were observed in 1 patient (7.1%) (case 6) after 3 months of treatment. Another patient (7.1%) (case 7) had recurrence with vitreous haze after 4 months of treatment. In these 2 patients (14.3%), who were both receiving anti-TNF treatment and developed vitreous haze during follow-up, AZA was added to systemic treatment and an intravitreal dexamethasone implant (Allergan Pharmaceuticals, Co., Mayo, Ireland) was injected. Their vitreous haze regressed within the first month of treatment. One patient (7.1%) (case 9) who was on AZA had recurrence with anterior segment inflammation after 2 months of treatment. Topical CS and CSA were added to her treatment. In 1 patient (7.1%) using CS and AZA who did not adhere to treatment (case 10), choroidal thickness was increased

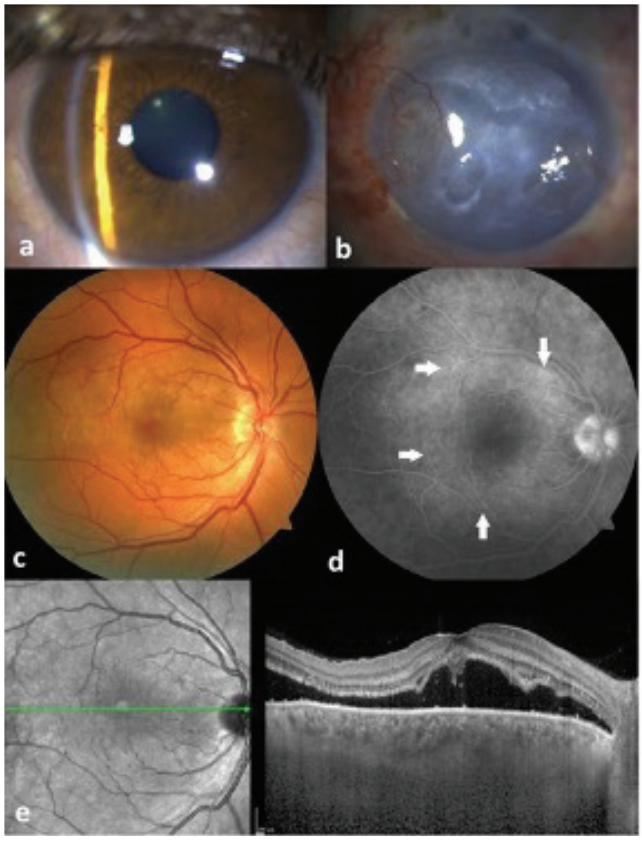

Figure 1. Case 3: Anterior segment images of the sympathizing (a) and sympathetic (b) eye. Fundus image of the sympathizing eye in the acute stage showing hyperemia and edema of the optic disc, and exudative macular detachment (c). Fluorescein angiography showing multiple pinpoint leakage areas, optic disc hyperfluorescence, and dye pooling with exudative retinal detachment (white arrows) (d). Enhanced depth imaging-optical coherence tomography showing serous macular detachment and diffuse choroidal thickening (e)

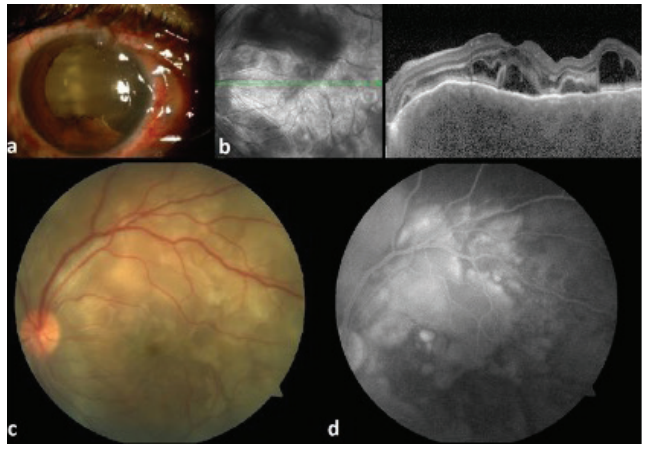

Figure 2. Case 10: Anterior segment image of the sympathetic eye (a). Enhanced depth imaging-optical coherence tomography of the sympathizing eye in the acute stage showing serous macular detachment with septa and choroidal folds, and diffuse choroidal thickening (b). Fundus image showing the optic disc hyperemia and exudative retinal detachment (c). Fluorescein angiography showing optic disc hyperfluorescence and dye pooling due to exudative retinal detachment (d)

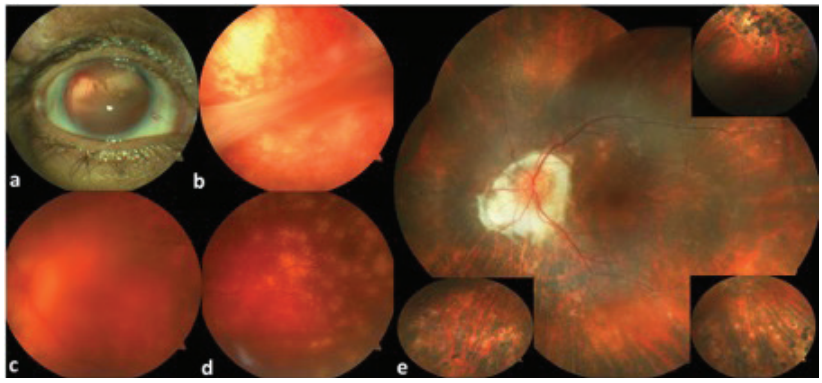

Figure 3. Case 6: Anterior segment (a) and fundus (b) images of the sympathetic eye. Fundus images of the sympathizing eye in the chronic stage showing sunset glow fundus (c) and Dalen-Fuchs (D-F) nodules (d) at presentation. Fundus image 2.5 years after treatment showing peripapillary chorioretinal atrophy, retinal pigment epithelial changes, and D-F nodules with hyperpigmentation (e)

after 3 months of treatment and CSA was added. During the follow-up period (ranging from 6 months to 10 years), remission was achieved in 13 of 14 patients and no new recurrence was observed. Acute retinal necrosis (ARN) due to herpes simplex virus 2 occurred in the remaining 1 patient (case 7). Systemic immunosuppressive treatment has been continued with reduced dosages in 11 patients (78.6%) and discontinued in only 3 patients (21.4%). One of these was the patient who developed ARN (case 7). The other 2 patients have been in drug-free remission for more than 5 years (cases 9 and 13).

Final BCVA was better than 20/50 in 11 of the sympathizing eyes. The most common ocular complications in our series were complicated cataracts (4 patients, 28.5%) and glaucoma (3 patients, 21.4%). One patient (7.1%) who developed glaucoma (case 4) had a final BCVA of 20/63. Corneal dellen and leucoma occurred in 1 patient (7.1%) (case 9), who needed corneal transplantation and had a final BCVA of 20/800. One patient (7.1%) (case 7) lost her vision due to the development of ARN during the course of the disease.

Discussion

SO is a rare but severe ocular inflammatory disease characterized by bilateral diffuse granulomatous uveitis. It occurs as a result of an autoimmune reaction against the eye's own antigens. In recent years, studies have focused on associated human leukocyte antigens and genetic susceptibility.[1] The inflammatory process begins primarily with the involvement of the choroid or anterior segment and progresses to panuveitis.[5,6]

Although intraocular surgical procedures have become an important risk factor for the development of SO in recent years, penetrating trauma has been classically accepted as the most common cause.[2,6,7,8,9] In the analysis of a multicenter SO case series, it was reported that 72% of the patients had a history of ocular trauma and 28% had a history of intraocular surgery.[7] Among SO cases associated with surgery, vitreoretinal surgery (36%) was the most common cause, followed by cataract surgery (30%) and glaucoma surgery (16%).[7] In another study, ocular surgery, especially retinal surgery, was reported to be the most common precipitating cause of SO.[8] Galor et al.[10] reported the rate of posttraumatic SO development as 38%. In our study, the proportion of posttraumatic SO was higher, at 71%, while postsurgical SO occurred in 29% of our patients. Our study emphasizes that trauma remains a major cause of SO.

Vitreoretinal surgery has been reported as another important cause of SO in recent years. Dutta Majumder et al.[11] reported that 14 of 197 patients had postoperative SO and that the surgical intervention most commonly associated with SO was vitreoretinal surgery (57%). Although our hospital is a tertiary referral center with a high volume of ocular and vitreoretinal surgeries (mean 3 thousand, 23-25 gauge vitreoretinal surgeries per year), the development of SO after vitreoretinal surgery was observed in only one patient. This may be related to the use of minimally invasive vitreoretinal surgery in recent years.

In most studies, SO has been reported to be more common in males due to their greater exposure to ocular injury.[2,12,13] Guzman-Salas et al.[13] reported that 65% of patients were male and attributed this to the higher frequency of trauma in men.[1] Galor et al.[10] reported that patients who developed posttraumatic SO were younger than patients with postsurgical SO. However, although posttraumatic SO development was more common in our series, female and male patients were in equal proportion. In addition, patients with posttraumatic SO and postsurgical SO were similar in mean age.

SO may develop in a period of time varying from the first few days to years after trauma or surgery.[5,14] It is reported that 80% of cases develop within the first 3 months and 90% within the first year after the triggering event.[1,15] However, in the literature, the longest period for the development of SO has been reported as 66 years after trauma.[3] In our study, the time between the triggering event and the onset of the disease ranged from 15 days to 60 years. SO developed after 35 years in one case and after 60 years in two cases in our series. The reason for the variable time intervals in the development of SO is not known exactly. SO is a rare pathology and patients with a history of eye trauma or surgery should be followed carefully throughout their lifetime. The possibility of SO should be kept in mind, considering that most of these patients have only one seeing eye.

SO presents clinically with acute and chronic ocular findings.[1,5,16] Its clinical features in different stages have not been well defined as in VKH. In their review of the literature, Yang et al.[2] determined the most common accompanying clinical finding was anterior chamber inflammation (69%), similar to other reports.[12,13] The most common clinical manifestation in our series was also anterior segment inflammation, which was granulomatous in most cases. Serous macular detachment and optic disc edema were the second most common clinical findings in our study. SO is usually confused with VKH disease because of the similarity of both acute and chronic findings. The absence of trauma or surgery history is important in the differential diagnosis of VKH; moreover, extraocular findings such as tinnitus, headache, sensorineural deafness, and vitiligo are less frequent in SO.[5] In our study, no extraocular findings were observed in any patient.

The assessment of fundus findings with multimodal imaging methods has an important role in both diagnosis and follow-up.[6] Fundus FA is helpful in evaluating retinal alterations in the acute and chronic stage.[1,6] OCT and EDI-OCT should also be used to visualize serous RD, measure choroidal thickness, and monitor treatment response.[17,18,19] In a study by Mahajan et al.,[6] serous RD with septa and choroidal folds were observed on OCT in SO patients, and choroidal thickening was observed in EDI-OCT, as in VKH disease. Studies have emphasized the importance of using EDI-OCT in SO patients, both in evaluating the response to treatment in patients presenting with exudative RD and in detecting early changes in asymptomatic patients.[17,19,20] Choroidal thickness was also reported to be a good biomarker for monitoring disease activity in VKH.[18] Behdad et al.[19] detected choroidal thickening during the acute phase in a young man with SO and observed that choroidal thickness decreased with treatment. Similarly, a recent study detected a significant increase in choroidal thickness in six acute SO patients compared to control eyes and reported that the assessment of choroidal thickness could be a quantitative parameter in the diagnosis and follow-up of patients.[20] EDI-OCT, which became a commonly used noninvasive imaging modality in recent years, was used in the follow-up of all our SO patients. We observed that the increase in choroidal thickness in the acute period regressed significantly with treatment. We also suggest that the evaluation of choroidal thickness by EDI-OCT is very useful in monitoring disease activity and treatment response in SO patients, especially when indocyanine green angiography is unavailable.

The use of high-dose systemic CS is still the gold standard in the initial treatment of SO. Discontinuing CS therapy with careful dose tapering over a period of 3-6 months after inflammation is controlled has been recommended.[1,5] Yang et al.[2] reported that 96% of patients were receiving systemic CSs, and 48% were also using an immunosuppressive therapy. An improvement in visual acuity at the last examination was detected in 77% of their patients.[2] Similar to previous studies, in our center, systemic CSs were preferred as the initial treatment in the acute stage in all patients, and steroid-sparing immunosuppressive agents were used for maintenance treatment. Depending on the patient's general condition and clinical manifestations, a single immunosuppressive agent (AZA) was used in seven patients and combination treatment (AZA + CSA) in seven patients.

Studies have reported that cases refractory to immunosuppressive treatment and/or cases with recurrent inflammatory attacks have been successfully treated with biological agents (anti-TNF-α).[5,21] Gupta et al.[22] reported a 2-year persistent remission and successful visual outcome with infliximab treatment in a 7-year-old SO patient who did not respond to combined immunosuppressive treatments. Similarly, successful results have been reported with adalimumab treatment in refractory SO patients in other studies.[23,24] Intravitreal CSs can also be used and have been shown to reduce the need for systemic therapies.[25,26] In our series, systemic treatment was switched to an anti-TNF-α agent in three patients who were nonresponsive to combined immunosuppressive therapy. Intravitreal CS was administered to two patients in addition to their systemic treatment. No recurrence was observed for a period ranging from 6 months to 10 years in patients who are still being followed up. Our results showed that the use of CS combined with immunosuppressive and/or anti-TNF agents is effective in controlling intraocular inflammation and preventing recurrences.

There is no consensus regarding the duration of systemic treatment in eyes with SO. Payal and Foster[21] presented long-term (>5 years) drug-free remission results of SO patients by using a stepwise treatment approach. They reported complete drug-free remission in a group of patients treated with immunosuppressive agents for 10 to 36 months. The authors recommended immunosuppressive therapies (commonly used antimetabolites; AZA, mycophenolate, methotrexate, or CSA) after initial treatment with CS to avoid the severe adverse effects of CSs and achieve steroid-free remission.[21] Two of our patients were also able to achieve drug-free remission after more than 5 years of treatment.

If not treated properly, SO can cause complications such as cataract, glaucoma, choroidal neovascularization, chorioretinal and optic nerve atrophy, phthisis bulbi, and severe vision loss.[1,5] Complications such as glaucoma and cataract may develop due to both chronic uveitis and the long-term use of topical and systemic CSs. Similar to previous reports, the most common complications were cataract and glaucoma in our series.[7,12] In addition, corneal dellen and leucoma developed in one patient and ARN in another one. ARN characteristically occurs in immunocompromised individuals. Although rare, it has also been reported in cases with immune dysfunction due to drugs, malignancies, or other systemic disorders.[27,28] Sims et al.[28] reported that ARN developed in two patients with iatrogenic immunosuppression who were using methotrexate and mycophenolate mofetil. Our patient who developed ARN was using systemic AZA and infliximab therapy. Despite the administration of systemic and intravitreal antiviral therapy for ARN, vision loss developed during follow-up.

With appropriate treatment and follow-up, remission was achieved in 13 of our 14 (93%) patients and the final BCVA was better than 20/50 in 11 (79%) patients. In the series presented by Payal and Foster,[21] 57.9% of the patients had a BCVA above 20/40 after treatment. Yang et al.[2] reported an increase in BCVA after treatment in 81.3% of their patients. In agreement with previous studies, we believe that the visual prognosis of SO may be favorable with rapid and appropriate treatment.

Conclusion

In conclusion, our findings suggest that trauma is still an important factor in the etiology of SO. Good anatomic and functional results can be obtained with early diagnosis and aggressive treatment. Considering that these patients have only one seeing eye, long-term treatment and close follow-up are essential in order to achieve remission and improve or maintain vision.

Ethics

Ethics Committee Approval: Ethics approval was obtained from the Ankara Training and Research Hospital Ethics Committee.

Informed Consent: Retrospective study.

Peer-review: Externally peer reviewed.

Authorship Contributions

Surgical and Medical Practices: P.Ç.Ö., Y.Ö.E., Concept: P.Ç.Ö., Y.Ö.E., Design: P.Ç.Ö., Y.Ö.E., Data Collection or Processing: K.Ö.Y., Analysis or Interpretation: K.Ö.Y., P.Ç.Ö., Y.Ö.E., Literature Search: K.Ö.Y., P.Ç.Ö., Y.Ö.E., Writing: K.Ö.Y., P.Ç.Ö., Y.Ö.E.

Conflict of Interest: No conflict of interest was declared by the authors.

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