Posterior Scleritis Case Series: Clinical Findings and Treatment Approach

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ABSTRACT

Purpose: To present clinical and demographic features, imaging findings, diagnosis and treatment approach of cases with posterior scleritis.

Material and Methods: We retrospectively reviewed data of six patients who were diagnoses with posterior scleritis and followed up between 2010 and 2020. In addition to detailed ophthalmological examination, optical coherence tomography (OCT), enhanced depth imaging-OCT, B-scan ultrasonography (US), fundus fluorescein angiography and magnetic resonance imaging, if needed, were obtained.

Results: Of the patients, 4 (66.7%) were female and 2 (33.3%) were male with mean age of 33.3±12.2 (15-49) years. The mean follow-up was 26.8±21.32 (6-58) months. All cases had unilateral involvement at presentation. The most common symptoms were pain exacerbated with eye movements and impaired vision. There was comorbid anterior scleritis in only 2 cases (33.3%). Serous retinal detachment was observed in 5 patients (83.3%) while optic disc edema in 3 patients (50%), scleral thickening and T-sign in all cases. There was comorbid autoimmune disease in 3 patients (%50). High-dose corticosteroids were given in all cases at acute phase of the disease. In 4 patients (66.7%), systemic immunosuppressive therapy was given concurrently. In one patient (16.7%), biological agents were given in addition to combined immunosuppressive therapy. Remission was achieved in all cases.

Conclusion: Posterior scleritis is an uncommon and vision threatening disease which can easily be misdiagnosed. Good visual and clinical outcomes can be achieved with early diagnosis and appropriate treatment.

Keywords: Posterior Scleritis, Scleritis, Imaging, Treatment, Prognosis.

INTRODUCTION

Scleritis is a serious, vision-threatening ocular inflammation characterized by edema and cellular infiltration of sclera.[1] Based on classification described by Watson and Hayreh in 1976, scleritis is classified as anterior and posterior in anatomic manner.[2] Posterior scleritis is defined as inflammation of sclera posterior to insertion of rectus muscles.2 The posterior scleritis is a rare form of the cases with scleritis and it has been reported to account for 2-12% of all cases with scleritis.[3]

Impaired vision and periocular pain are most common presenting complaints in posterior scleritis.4 The clinical findings may vary based on localization and severity of scleral involvement. The posterior scleritis may manifest either as isolated posterior scleral involvement or red eye where comorbid anterior scleritis is present.[1] There may be accompanying findings including serous retinal detachment at posterior segment, optic disc edema and macular edema. However, fundus can be observed as normal in some cases. The posterior scleritis is mostly idiopathic; however, association with systemic autoimmune diseases have been reported by 10-40%.[1, 5, 6] The posterior scleritis is mostly overlooked or misdiagnosed due to clinical characteristics resembling ocular tumors and other ocular or orbital inflammatory diseases.[7, 8] In a study from tertiary clinic in India, it was reported that posterior scleritis was detected in 8.5% of 94 cases with scleritis and that it was failed to establish the diagnosis in primary ophthalmological care settings.9 In another study, it was reported that 74% of cases with posterior scleritis were misdiagnosed at presentation.10 In these patients, in addition to history and clinical findings, imaging studies including B-scan ultrasonography, optic coherence tomography (OCT),

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enhanced depth imaging-OCT (EDI-OCT), fundus fluorescein angiography (FFA) and magnetic resonance imaging can be helpful in establishing diagnosis.[1, 10]

In posterior scleritis, diagnostic challenges lead delay in treatment, vision loss and complications. In this study, it was aimed to assess clinical and demographic characteristics, imaging findings, diagnostic and therapeutic approaches in 6 cases with posterior scleritis.

MATERIALS AND METHODS

We retrospectively reviewed clinical and demographic characteristics of 6 patients who were diagnosed with posterior scleritis and followed at Ulucanlar Eye Training and Research Hospital of University of Health Sciences between 2010 and 2020. The study was conducted in accordance with tenets of Helsinki Declaration. In all patients, detailed ocular and systemic history and comprehensive ophthalmological examination including best-corrected visual acuity (BCVA) using Snellen chart at presentation and during follow-up, intraocular pressure (IOP), anterior segment examination and fundus examination were performed. The diagnosis of posterior scleritis was made based on symptoms such as ocular pain exacerbating with eye movements as well as typical clinical findings and presence of posterior scleral thickening and subtenon fluid accumulation (T-sign) on B-scan ultrasonography. In all patients, routine blood tests including complete blood count, biochemistry assays, acute phase reactants (erythrocyte sedimentation rate, C-reactive protein), serological tests for syphilis and ELISA tests for hepatitis viruses and HIV as well as additional tests (if needed) were performed. All patients were consulted with rheumatology department for systemic rheumatoid diseases.

Topical corticosteroid (prednisolone acetate) was given to patients with accompanying anterior segment inflammation. As initial treatment, pulse intravenous methylprednisolone (1 g/day for 3-5 days) was given to all patients; followed by oral methylprednisolone at does of 1 mg/kg/day or 1.5-2 mg/kg/day (high dose corticosteroid therapy). The systemic corticosteroid therapy was tapered gradually. Based on clinical status, systemic azathioprine (AZA) and/or cyclosporine-A (CSA) were given if needed. Biological agents were used if combined immunosuppressive therapy was failed.

FINDINGS

In this case series, there were 4 women (67%) and 2 men (33%). The mean age was 33.3±12.2 years (15-49 years). The mean follow-up was 26.8±1.32 months (6-58 months). Unilateral involvement was detected in 5 cases (83.3%) while bilateral involvement in one case (16.7%) (case 4) during follow-up. The presenting complaints included ocular pain exacerbated with eye movements in 6 patients (100%), impaired vision in 5 patients (83.3%), red eye in 2 patients (33.3%) and concurrent headache in 1 patient (16.7%). At presentation, BCVA was <0.3 in 4 patients (66.7%), 0.6 in 1 patient (16.7%) and 1.0 in 1 patient (16.7%). There was concurrent hyperemia with anterior scleritis findings in 2 patients (33.3%) (Figure 1a) while anterior segment findings were normal in 4 patients (66.6%). There was serous retinal detachment in 5 patients (Figure 1b, 2a, 3b) and optic disc edema in 3 patients (50%) Serous retinal detachment was confirmed in 5 patients (83.3%) with OCT imaging (Figure 1c, 2c, 3f). At presentation, choroidal thickening was detected in 5 patients (83.3%) by EDI-OCT (Figure 3f). It was failed to obtain EDI-OCT in one patient. The mean choroidal thickness was found as 656±79.6 μm (420-857) in 5 patients with available EDI-OCT imaging. On FFA, it was observed that there was perifoveal leakage in 4 eyes (66.6%), optic disc leakage in 4 eyes (66.6%), hyperflorescence due to accumulation at serous retinal detachment areas in 5 eyes, choroidal folding in 2 eyes (33.3%) and choroidal hypoflorescent lines in 1 eye (16.7%) (Figure 1d, 2d, 3d). In all cases, scleral thickening and T-sign were observed on B-scan ultrasonography (Figure 1e, 2d, 3g, 3h). In one case (case 6), there was chronic central serous chorioretinopathy (CCSR) in intact eye (right eye); fundus examination and imaging were found to be normal in intact eyes of remaining 5 cases (Case 1-5). There was comorbid systemic autoimmune disease in 3 cases (50%) including rheumatoid arthritis in one (case 4) and Familial Mediterranean Fever (FMF) in two patients (Case 5 and 6). No systemic disease was detected in remaining 3 patients who were diagnosed as idiopathic posterior scleritis.

In the treatment, intravenous pulse corticosteroid therapy was initially given; followed by oral maintenance therapy in 5 patients. In one case, oral corticosteroid was started at a dose of 1.5 mg/kg/day. In 4 patients (66.7%), immunosuppressive therapy was added. Recurrence was detected in 3 patients (50.0%; Case 2, 3 and 4) during follow-up. The patient with FMF(Case 6) was previously received systemic corticosteroid therapy with diagnosis of optic neuropathy in another facility; the patient had history of CCSR developed following systemic corticosteroid therapy. Due to risk for corticosteroid-related CCSR in the eye with posterior scleritis, rapid-acting anti-tumor necrosis factor (anti-TNF) was offered to the patient; however, the patient declined treatment with an anti-TNF agent. Thus, AZA was added to systemic corticosteroid therapy in this patient. The corticosteroid dose was tapered rapidly. No recurrence was detected in control visit. Recurrence

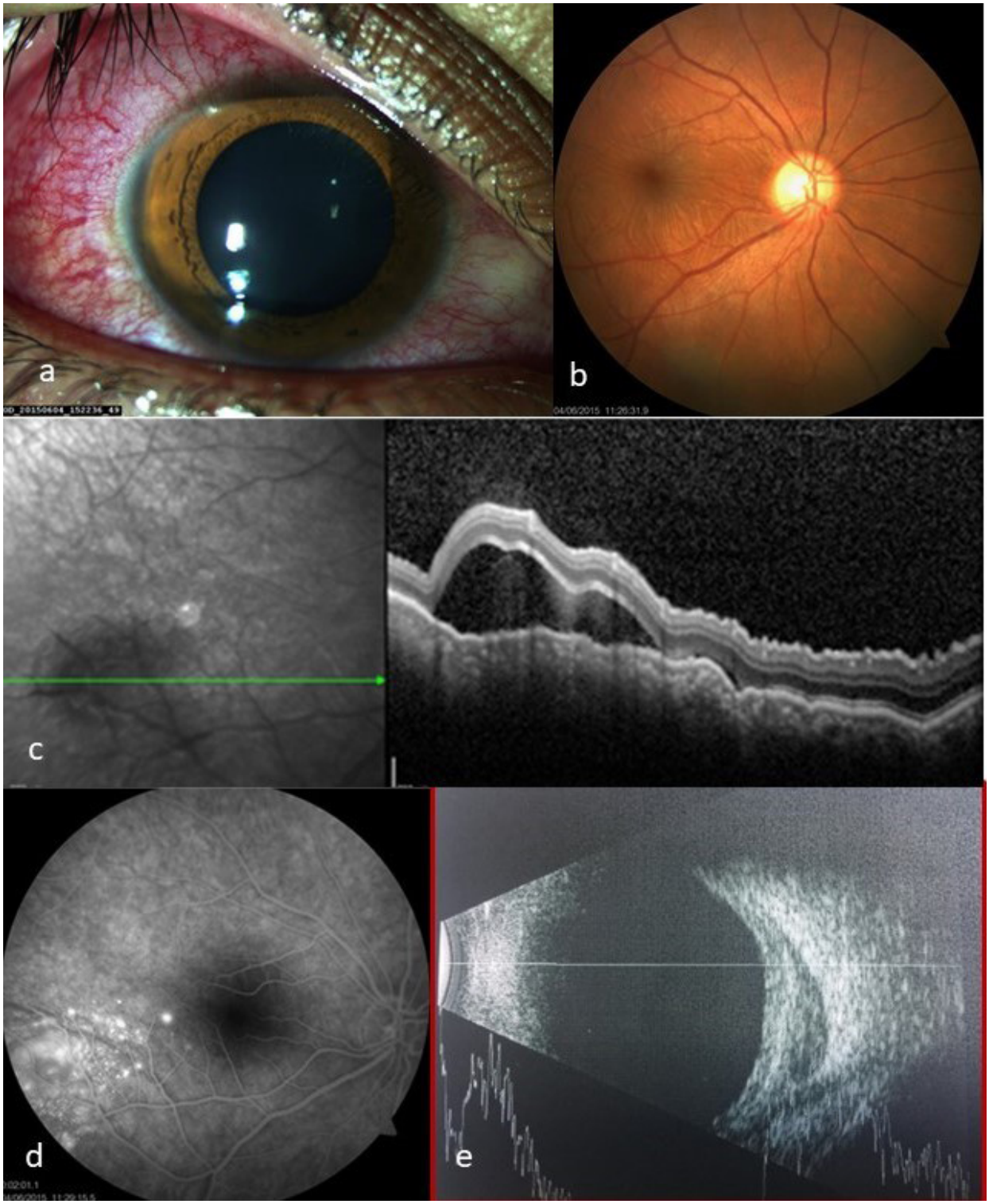

Figure 1 a-e: *Case 2. Marked congestion and shallow anterior chamber in the anterior segment of the right eye (a) Macular wrinkle in the fundus and serous retinal elevation in the inferotemporal macula (b) Serous retinal detachment and RPE undulation on OCT (c) Pinpoint leakage and dye pooling in the area corresponding to the serous retinal detachment on FFA (d) Choroidal and scleral thickening, and marked subscleral fluid on B-scan ultrasonography (e)*

with anterior scleritis and serous retinal detachment was observed in one patient with poor compliance (Case 2); AZA was added to topical and systemic corticosteroid therapy. In one patient (Case 3), AZA was added to systemic corticosteroid therapy due to presence of increased choroidal thickness with pain during follow-up. AZA plus CSA combination was added as the patient with RA (Case 4) showed poor response to corticosteroid therapy. In this patient, different biological agents (rituximab, infliximab, golimumab, tocilizumab) were given in consultation with

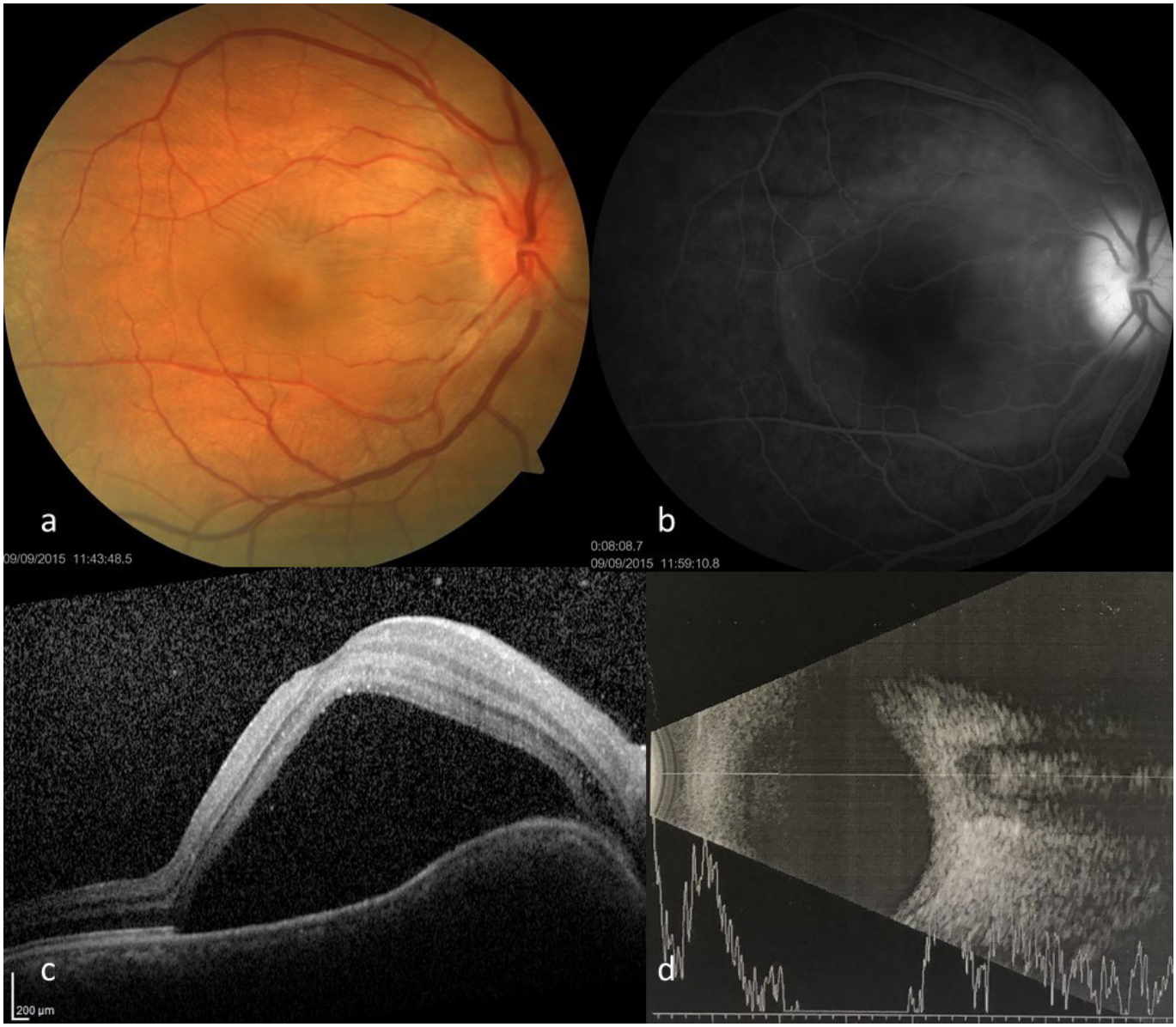

Figure2 a-d: *Case 4. Optic disc edema and hyperemia, macular serous detachment and retinal folds in the right eye color fundus image (a) Optic disc hyperfluorescence and dye pooling in the macula in the late period of FFA (b) Serous retinal detachment and RPE undulation on OCT (c) Choroidal and scleral thickening, and fluid around optic nerve sheath on B-scan ultrasonography (d)*

rheumatology clinic as multiple recurrences with optic disc edema, macular edema and serous retinal detachment were observed despite combined immunosuppressive therapy. Inflammation could be controlled by tocilizumab and no recurrence was observed during follow-up.

Remission was achieved in all patients after treatment. The choroidal thickness was markedly decreased after treatment and the mean choroidal thickness was measured as 380.2 ±67.3 µm (187-500). In one case, cataract was developed during follow-up and the patient underwent cataract surgery (Case 4). In remaining cases, no additional surgery was required. The BCVA was 0.2 in one patient (16.7%; Case 4) whereas >0.8 in 5 patients (83.3%). The case with BCVA of 0.2 was the patient with comorbid RA and it was warranted to change therapeutic agent several times due to resistant to therapy; vision loss was due to optic atrophy in this patient. In remaining cases, no sequel other than minimal irregularity of retinal pigment epithelium (RPE) was observed.

Table 1 summarizes clinical and demographic characteristics, imaging findings and treatment modalities; one case (Case 1) was discussed below in detail.

CASE REPORT

Case 1

A 36-years old woman initially presented to neurology outpatient clinic with headache and pain at left eye over one week; the examination was assessed as normal and oral

Table 1: *Demographic and clinical characteristics, imaging findings and treatment of the patients.*

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6
Age/Gender	36/F	24/F	49/F	34/F	15/M	36/M
Follow-up	6 months	48 months	24 months	58 months	12 months	13 months
Etiology	Idiopathic	Idiopathic	Idiopathic	RA	FMF	FMF
Involvement	Left	Right	Left	Left	Bilateral	Left
Presenting complaint						
Periocular pain	+	+	+	+	+	+
Decreased vision	+	+	-	+	+	+
Red eye	-	+	+	-	-	-
Headache	+	-	-	-	-	-
Anterior segment	Normal	Dense congestion, Shallow anterior chamber	Conjunctival hyperemia, Chemosis	Normal	Normal	Normal
Fundus						
OD edema	+	-	-	+	+	-
Seröous RD	+	+	-	+	+	+
OCT	Serous RD	Serous RD	RPE changs	Serous RD	Serous RD	Serous RD
EDI-OCT Choroidal thickening	+	+	+	+	-	+
FFA	OD and perifoveal leakge, pooling	Pinpoint leakage at temporal to fovea, pooling	Leakage at inferior perifoveal area, pooling, Choroidal folds	OD leakage, Choroidal hyper-florescent lines	OD leakage, pooling	Leakage at inferior perifoveal area, choroidal folds, pooling,
Sonography T-sign	+	+	+	+	+	+
Treatment						
Topical corticosteroid	-	+	+	-	-	-
Systemic corticosteroid	+	+	+	+	+	+
Immunosuppressive	-	AZA	AZA	AZA+CSA	-	AZA
Biological agent	-	-	-	Rituximab Infliximab, Golimumab, Tocilizumab	-	-
Nüks	-	+	+	+	-	-
Pretreatment visual acuity	1.0/0.6	0.1/1.0	1.0/1.0	1.0/50 cmps	0.1/0.1	0.05/0.3
Post-treatment visual acuity	1.0/1.0	1.0/1.0	1.0/1.0	1.0/0.2	1.0/1.0	0.05/0.8

*F:Female, M:Male, RA: Rheumatoid arthritis, FMF: Familial Mediterranean Fever, OD: optic disc, RD: retinal detachment, RPE: retinal pigment epithelium AZA: azathioprine , CS-A:cyclosporine A

indomethacin therapy was started. However, the patient experienced decreased vision and presented to our clinic. In the examination, BCVA was 1.0 in the right eye and 0.6 in the left eye while IOP was 14 mmHg in right eye and 18 mmHg in the left eye. Bilateral anterior segment examination was normal; the patient had normal light reflex and no relative afferent pupil defect (RAPD). In fundus examination, right eye was found as normal while there was optic disc edema and serous swelling around macula in left eye (Figure 3a, 3b). On FFA, right eye was normal while hyperflorescence due to leakage at optic disc areas and perifoveal region was observed in left eye (Figure 3c, 3d). On OCT, right eye was normal while serous retinal detachment was detected at subfoveal and peripapillary areas in left eye. Undulation was marked at RPE. On EDI-OCT, choroidal thickness was measured as 305 µm in right eye while diffuse choroidal thickening was detected in left eye (Figure 3e, 3f). The patient was initially considered

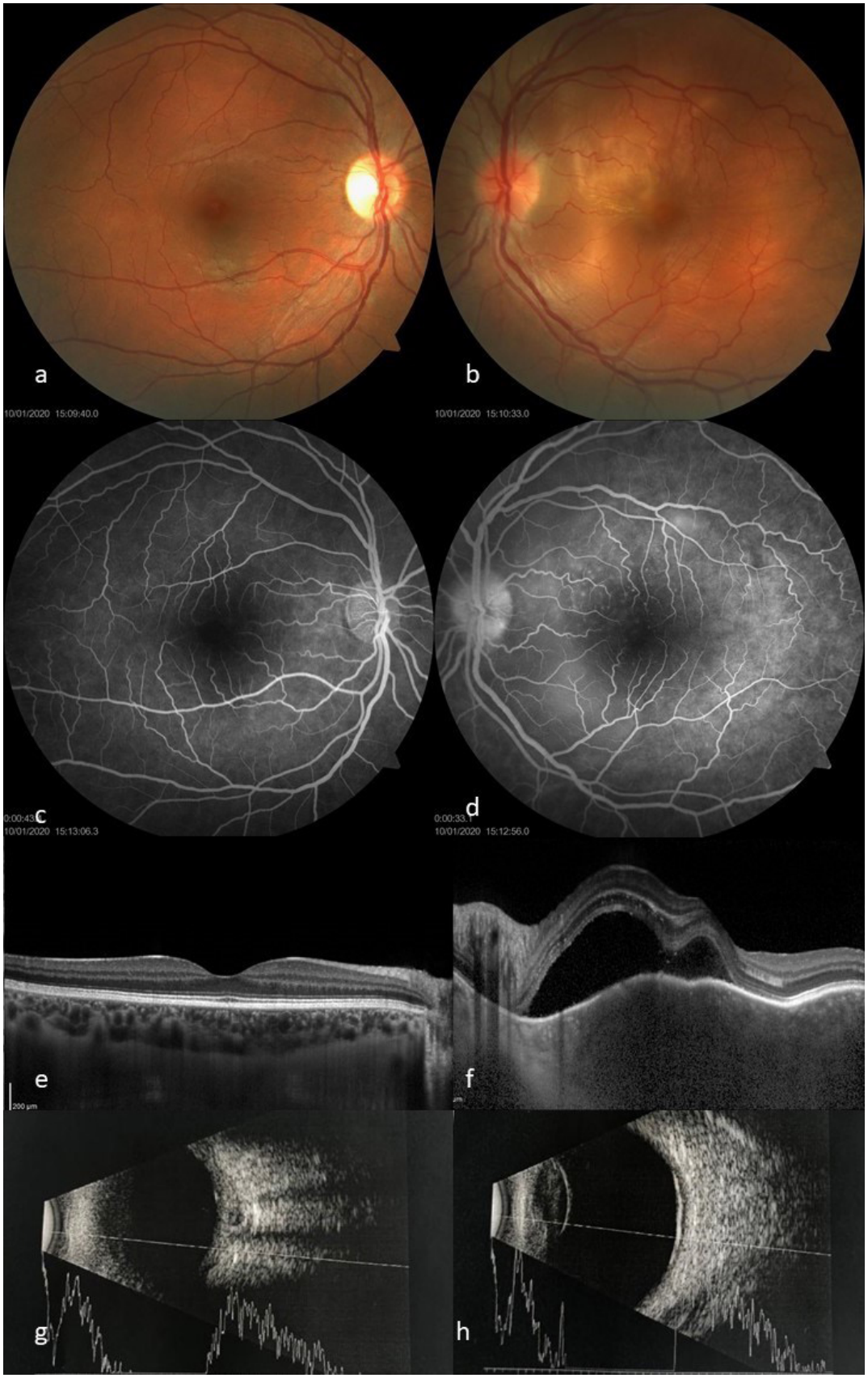

Figure3 a-h: *Case 1. Normal fundus findings in the right eye (a) Optic disc edema and hyperemia in the left eye, serous retinal elevation extending from optic disc to fovea (b) Normal FFA findings in the right eye (c)Optic disc hyperfluoresence in the left eye and pinpoint leakage in the macula. (d) Normal choroidal thickness in the right eye on EDI-OCT (e) Serous retinal detachment and RPE undulation with diffuse increase in choroidal thickness in the left eye on EDI-OCT (f) Scleral thickening and T sign (g), and serous retinal detachment on B-scan ultrasonography (h)*

as Vogt-Koyanagi-Harada (VKH) disease with asymmetric involvement; thus, B-scan ultrasonography was performed which revealed scleral thickening and T-sign (Figure 3g, 3h). The patient was diagnosed as posterior scleritis based on these findings. Due to lack of any abnormal finding in systemic, rheumatologic or laboratory workup, the patient was considered as idiopathic posterior scleritis. The patient was given pulse methylprednisolone (1 g/day for 3 days); followed by oral methylprednisolone (80 mg/day). The serous retinal detachment was regressed after one week and BCVA was improved up to 0.8. The clinical and imaging findings were normal on day 45 and final BCVA was 1.0. Oral corticosteroid therapy was gradually tapered and no recurrence was detected.

DISCUSSION

Posterior scleritis is a rare form of scleritis which may threaten vision and can be often overlooked due to non-specific clinical characteristics. Thus, clinical characteristics are reported at varying rates in case series.[4-6] Although posterior scleritis can be seen at any age, it is more commonly seen at decades 4 or 5, in young adults and women. Unilateral involvement is more common; however, bilateral involvement has been rarely reported.[1, 10] In a study on 114 patients, Lavric et al. reported that 71.1% of the cases were women and posterior scleritis was bilateral in 15.8% of patients.4 In our series, posterior scleritis was more common in women and bilateral involvement was rare (16.7%) in agreement with literature. Initially, all patients showed unilateral involvement; however, one patient presented with posterior scleritis in contralateral eye during follow-up. This patient had diagnosis of RA. It was thought that comorbid autoimmune systemic disease led tendency to bilateral involvement.

In previous studies, it has been reported that periocular pain and impaired vision are most common symptoms in posterior scleritis.[4, 9, 10] In a case series including18 patients, Kumar et al. reported decreased vision in 16 (88.8%) and periocular pain in 11 (61.1%) of the patients.6 In our study, the most common presenting symptom was periocular pain which was observed in all patients. There was impaired vision in 83.3% of cases. Pain is an important symptom in posterior scleritis; it occurs due to strain and swelling of scleral nerves. However, it should be kept in mind that posterior scleritis can develop without ocular pain.[6] The pain can be limited to periocular region or may project to head; it typically aggravates by ocular movements and during sleep.[1] The questioning characteristics of pain and detailed history is highly helpful when assessing patients.

In the posterior scleritis, findings of anterior segment inflammation such as conjunctival hyperemia, chemosis or anterior chamber reaction can be observed; however, it may manifest without red eye.5 Kumar et al. reported anterior chamber reaction in 50% of cases with posterior scleritis while Dong et al. reported anterior scleritis in 52% of the cases.6, 10 In our series, anterior scleritis was observed in 33.3% of the patients while anterior segment examination was normal in 66.7%. In recent years, association of anterior and posterior scleritis has been emphasized, proposing the term “panscleritis” to define the diffuse scleritis.11 In posterior scleritis, several findings including serous retinal detachment, optic disc edema, choroidal folds, macular edema, vitritis and subretinal granuloma can be seen at posterior segment.4-6 In a study by McCluskey et al., it was reported that serous retinal detachment was the most common posterior segment finding while there was no posterior segment finding in 17% of the cases.5 In our series, the most common posterior segment findings were serous retinal detachment (83.3%) and optic disc edema (50%).

Posterior scleritis may manifest with highly varying clinical presentations. The inflammation of sclera and choroid leads serous macular detachment through vasodilatation in choroidal vessels and subretinal fluid accumulation.3 In these patients, differential diagnosis should include CCSR and VKH disease.3, 6 The inflammation of peripheral sclera may lead uveal effusion and secondary angle closure; in such case, uveal effusion syndrome, rhegmatogenous retinal detachment and causes of annular ciliochoroidal detachment such as intraocular surgery should be considered in differential diagnosis.5, 6 In cases with optic disc edema, the conditions which may lead elevation in intracranial pressure such as dural sinus thrombosis, intracranial mass lesion and pseudotumor cerebri should be kept in mind in differential diagnosis; optic disc edema is generally bilateral in intracranial pressure elevation; however, it can be asymmetrical in some cases.5 Nodular inflammation of sclera can lead appearance of subretinal mass lesion, leading confusion with different diagnoses such as choroidal malignant melanoma, metastatic malignancies of uvea, choroidal hemangioma or benign reactive uveal lymphoid hyperplasia.12, 13 Magnetic resonance imaging and sonography are helpful in diagnosis in such cases.5, 6, 12, 13 As seen, clinical findings vary according to localization and degree of inflammation in posterior scleritis. Thus, the diagnosis is made by excluding other diagnosis in posterior scleritis; in addition, multimodal imaging studies have an important role in diagnosis.

B-scan ultrasonography is a non-invasive, simple method and should be first diagnostic tool when posterior scleritis is suspected. On ultrasonography, scleral and choroidal thickening, edema at optic disc head and sheath and

retinal detachment can be seen.1 A specific sign, termed as "T-sign" is observed in posterior scleritis which is caused by increased thickness of scleral wall and encircled optic nerve by subtenon fluid.[1, 5] The T-sign was observed in all patients in our case series. T-sign may not always present in posterior scleritis; however, it should be kept in mind that the lack of T-sign isn't necessarily exclude posterior scleritis. In a study, T-sign was observed in only 41% of 114 patients.[4] MR imaging can be used to establish diagnosis in posterior scleritis cases with non- specific signs on ultrasonography or in those patients found to have subretinal or choroidal mass appearance.[12, 13] In our study, in a patient who presented with pain during follow- up and normal ultrasonography, inflammatory changes were detected around optic nerve on MR imaging; thus, the patient was considered as recurrence and treatment was re-arranged. The MR imaging can be used in both differential diagnosis and detailed assessment of posterior scleral structures during follow-up when clinical examination and ultrasonography are insufficient.

Optic disc leakage and pinpoint leakage around optic disc and perifoveal area have been reported in patients with posterior scleritis on FFA.[10] Similar FFA findings were observed in our patients; in addition, choroidal hypoflorescence lines together with optic disc leakage were observed in one patient on FFA. In acute period, serous retinal detachment as well as RPE folds can be seen on OCT images while choroidal thickness can be observed on EDI-OCT.[10, 14] In all 5 patients with serous retinal detachment, concurrent RPE folds on OCT and choroidal thickening on EDI-OCT were observed. Posterior scleritis can often be confused with VKH disease due to similar clinical and imaging findings.[15] Unlike posterior scleritis, VKH disease is a panuveitis with bilateral involvement. Subclinical disease can be detected in contralateral eye by indocyanine angiography in VKH patients presenting with unilateral involvement.[16] In addition, in VKH disease, serous retinal detachment with septation in subretinal area was observed on OCT while choroidal thickening was more marked on EDI-OCT.15 Initially, asymmetric VKH disease was considered in two patients based on OCT, EDI-OCT and FFA findings; however, VKH was excluded by presence of T-sign on ultrasonography in addition to pain. The studies in the literature have been reported that choroidal thickness measurements by EDI-OCT is a good biomarker for monitoring both disease activity and treatment response.[10, 14] Ando et al. evaluated changes in choroidal thickness following corticosteroid therapy during 2-years follow-up in patients with posterior scleritis and reported that there was significant reduction upon month 1 after treatment when compared to baseline.[14] In our series, in all cases with available choroidal thickness measurement, it was found that choroidal thickness as measured by EDI-OCT was increased at acute period and markedly decreased by treatment.

In the literature, association with systemic rheumatoid disease was reported in 30-40% of patients with posterior scleritis.[4, 5, 14] RA, systemic lupus erythematosus, poliangitis and granulomatous poliangitis are most common disorders in patients with posterior scleritis.[4, 5] In our series, there was systemic disorder in 50% of patients. There was association with RA in one patient and FMF in 2 patients. Three patients (50%) were considered as idiopathic based on laboratory evaluations and detailed clinic examination.

High-dose corticosteroids is mainstay of the treatment regarding rapid control of inflammation in posterior scleritis.[17, 18] It is recommended to use immunosuppressive agents (e.g. methotrexate, AZA, mycophenolate, CSA) in order to avoid potential side effects of long-term corticosteroid use in patients with recurrent disease or comorbid autoimmune disease.[18, 19] Biological agents are the next step in the treatment of cases who are unresponsive to conventional therapy.[20, 23] In our series, all patients received high-dose corticosteroid therapy at acute phase. Recuurence was observed in 2 patients with incompliance to treatment and 1 patient with comorbid RA. Systemic immunosuppressive therapy was added to corticosteroids in 3 patients with recurrence and in one patient with comorbid FMF and corticosteroid-related CCSR in contralateral eye. In the patient with RA, due to recurrent, refractory attacks, biological agents including infliximab, rituximab, golimumab and tocilizumab were added to combined immunosuppressive therapy as recommended by rheumatology department; inflammation control was eventually achieved by tocilizumab. It is known that scleritis has more severe course in patients with underlying systemic diseases; thus, cooperation and aggressive treatment approach are required to prevent complications and recurrent attacks. After treatment, remission could be achieved in all patients in our series and final BCVA was found to be >0.8 in 5 (83.3%) of 6 patients. The only case with low BCVA was the patient with comorbid RA who was refractory to the treatment; vision loss was due to optic atrophy in this patient.

In our study, it was aimed to discuss clinical characteristics, imaging findings and treatment approaches in patients with posterior scleritis accompanied by the current literature and to increase awareness on this rare disease which is often misdiagnosed.

In conclusion, posterior scleritis is a rare disorder which is generally unilateral and manifests with pain and decreased vision. As clinical and imaging findings can

resemble other disorders, mainly VKH disease, diagnosis can be readily overlooked. It is possible to achieve good visual and clinical outcomes with early diagnosis and appropriate treatment. In unilateral cases with periocular pain exacerbating by ocular movements and serous retinal detachment, posterior scleritis diagnosis can be made by EDI-OCT, ultrasonography and orbital MR imaging if needed. Systemic corticosteroids and immunosuppressive agents are effective in the treatment; the screening of systemic diseases which may accompany posterior scleritis and appropriate treatment are important factors for prognosis.

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