A Case of Posterior Scleritis Presenting as Unilateral Secondary Angle-Closure Glaucoma

Gozde SAHIN VURAL[1], Eyyup KARAHAN[2], Cenap GULER[3]

ABSTRACT

Posterior scleritis is a rare ophthalmologic disease with inconstant manifestations, which occasionally leads to misdiagnoses and mistreatment. This paper reports a patient presented with secondary angle glaucoma after initially diagnosed and treated as primary angle closure glaucoma. Previous treatment with miotics worsens the closure of the angle and acute angle-closure attack was successfully resolved with immediate systemic corticosteroids plus cycloplegic treatment. Our aim in reporting this case is to remind the variable presentations of posterior scleritis and to emphasize diversity between treatment of primary and secondary angle closure glaucoma.

Keywords: Angle-closure, Secondary glaucoma, Posterior scleritis.

INTRODUCTION

Posterior scleritis is a rare inflammatory disease that causes changes in the retina, uvea and optic nerve. The diagnosis of the disease is made with the help of clinical examination, ultrasonography and computed tomography if necessary. The disease usually affects the posterior part of the eye, but in some patients, the anterior chamber angle becomes narrower due to the effusion and secondary angle-closure glaucoma occurs.[1,2] In these patients, differential diagnosis with primary angle-closure glaucoma is very important for appropriate treatment.

CASE REPORT

A fifty six year-old woman has been referred from a local ophthalmologist with complaints of hyperemia and minimal pain in left eye with a 1-week history. She was noted to have marked conjunctival injection, eyelid edema, shallow anterior chamber and increased intraocular pressure (IOP) of 32 mmHg in the left eye. Patient had been diagnosed as primary acute angle closure glaucoma, and she was treated with laser iridotomy (LI) after instillation of 0.5% timolol and 1% dorzolamide. The patient was referred to our department because the shallow anterior chamber and increased IOP persisted on the next day. She had only history of hypertension history and usage of Angiotensin converting enzyme inhibitor (ACE-inh) drugs for two-years.

On external ophthalmic examination right eye had normal view, where as in the left eye there was a mild subcutaneous edema on left zygomatic area and swelling of the left eyelid. The left eye had 1 mm of proptosis (Hertel: 22 mm), (Figure 1) painful eye movements and tenderness on globe palpation. The pupil was miotic (approximately 1.5-2 mm) due to previously dropped miotics. There was no reaction to the direct or indirect light. Her best-corrected visual acuity was 0.9 in the right eye and 0.2 in the left eye. The spherical equivalent of the refraction measured by autorefractometer was +0.25 diopters (refractive error of +0.50 -0.50*90) in the right eye and +2.00 diopters (refractive error of +2.25 -0.50*95) in the left eye. The IOP was 18 mmHg in the right eye and 42 mmHg in the left eye with Goldmann applanation tonometer. Slit-lamp examination of the left eye revealed minimal chemosis, venous dilatation and moderate vascular engorgement in the episclera and conjunctiva, a clear anterior surface of cornea with a few thin keratic precipitates, very shallow central anterior chamber, flat peripheral anterior chamber, 1+ cells

1- Assistant Prof. MD, Ophthalmogy Department of Balikesir University Medicine School, Balikesir, Turkey

2- Associate Prof. MD, Ophthalmogy Department of Balikesir University Medicine School, Balikesir, Turkey

3- Prof. MD, Ophthalmogy Department of Balikesir University Medicine School, Balikesir, Turkey

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Correspondence Adress:
Gozde SAHIN VURAL
Ophthalmogy Department of Balikesir University Medicine School, Balikesir, Turkey

Phone: +90 542 253 8910
E-mail: gozdejcgrl@hotmail.com

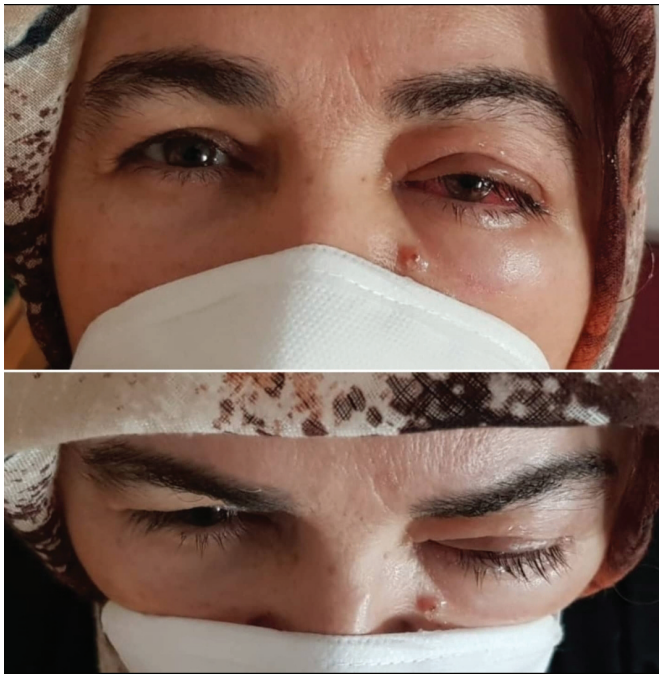

Figure 1: *Eye and ocular adnexes inspection at first visit.*

in the anterior chamber, and a patent iridotomy at 1 and 11 o'clock. (Figure 2) The depth of the anterior chamber of the right eye was shallow as well. Gonioscopy showed that the angle was grade 1 (Shaffer grading system) in the right eye and grade 0 in the left. B-scan ultrasonography of the left eye demonstrated a diffuse thickening of the posterior coats together with fluid in Tenon's capsule (Figure 3) and an annular choroidal detachment. (Figure 4) Computed tomography (CT) revealed a slight proptosis, thickening and anterior shift of the lens, choroidal detachment, and thickening of sclera in the left eye. (Figure 5) The

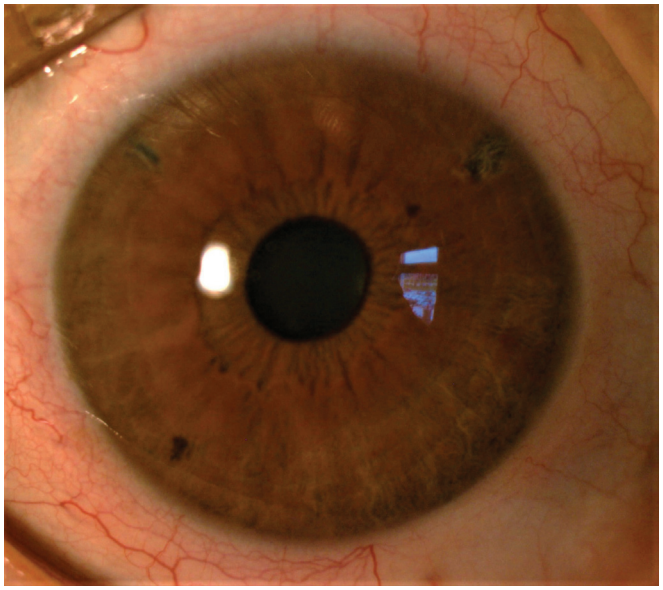

Figure 2: *Anterior segment photograph at first visit (Patent laser iridotomies at 1 and 11 o'clock).*

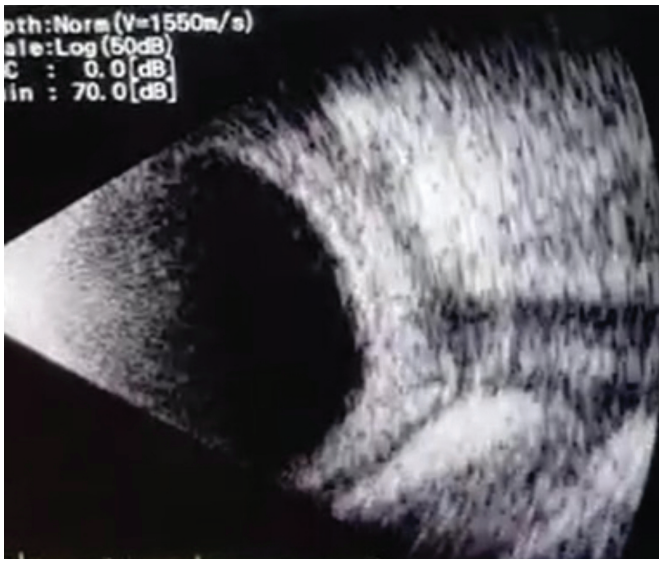

Figure 3: *T-sign in B-scan ultrasonography at first visit.*

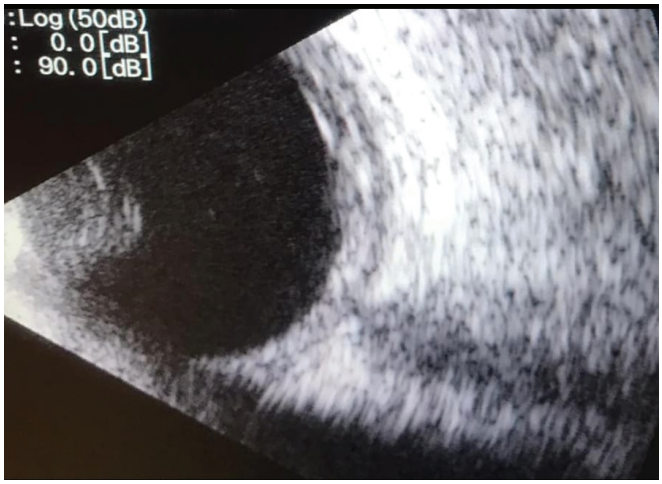

Figure 4: *Diffuse scleral thickening in B-scan ultrasonography at first visit.*

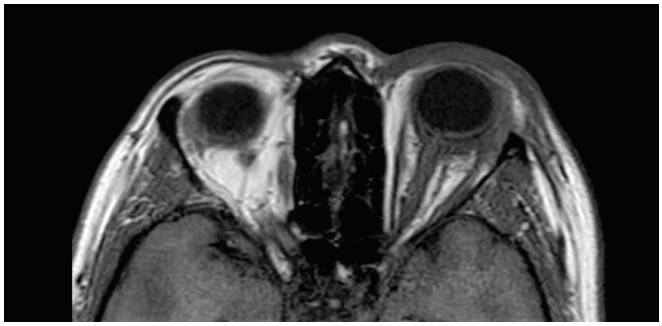

Figure 5: *Slightly proptosis, scleral thickening, choroidal detachment in computized tomography (CT).*

ultrasound and CT scan investigations confirmed the diagnosis of posterior scleritis.

Additionally, axial length was 20.40 mm and anterior chamber depths were 2.34 mm and 2.04 mm in right and left eyes. Chest X-ray did not show any abnormalities, and there

was no diagnosis after rheumatologic assessment [Routine hematological tests and CBC: Normal, Sedimentation: 9mm/h, ANA: (-), Anti CCP: (-), Anti Toxoplasmosis IGG-IGM: (-), Treponema pallidum IGG-IGM: (-), CMV IGG-IGM: (-), Rheumatoid factor:(-)]

The patient was diagnosed as having ciliochoroidal effusion secondary to posterior scleritis, which led to the acute angle-closure glaucoma. Cyclopentolate was instilled at 5-min intervals immediately together with intravenous infusion of 20% mannitol (300cc) and instillation of 0.5% timolol maleate and 1% dorzolamide fixed combination after the diagnosis. Steroid pulse therapy with 1000 mg of intravenous methylprednisolone sodium succinate was initiated with topical cyclopentolate and continued for 3 days and remained with 1mg/kg p.o methylprednisolone for one-week and then tapered by 20 mg daily.

One day later after initiation of the treatment, IOP decreased to 24 mmHg with Goldmann applanation tonometer and gonioscopy showed that the angle was now grade 1 (Shaffer grading system) and keratic precipitates on the endothelium and cells in anterior chamber were disappeared totally, ophthalmoscopic examination of the left eye demonstrated residue serous retinal detachment and choroidal thickening.

After one week, BCVA improved to 0.9 in the left eye. The spherical equivalent of the refraction measured by autorefractometer was +1.00 diopters (refractive error of +1.25 -0.50*95) after the treatment, the depth of the anterior chamber in the left eye was almost same as in the right eye, (Figure 6) IOP in the left eye decreased to 18 mmHg via Goldmann applanation tonometer without any antiglaucoma medication and complaints of hyperemia, pain and lid edema were resolved, (Figure 7) and also extraocular movements were normal. The scleral thickening decreased but still not in the normal diameters. (Figure 8) There has been no recurrence during the follow-up period of 3-months.

DISCUSSION

The angle closure glaucoma is described as one of the most hazardous complication of posterior scleritis.[3,4] The definition of angle-closure glaucoma secondary to posterior scleritis is important because the therapeutic strategy for ciliochoroidal effusion syndrome differs from conventional angle-closure glaucoma with a pupillary block. Interestingly, in our case fellow eye had also narrow anterior chamber and angle. It might complicate to differentiate primary and secondary angle closure glaucoma. Nevertheless, worsening of the angle closure after miotics and patent laser iridotomy (LI) should be a

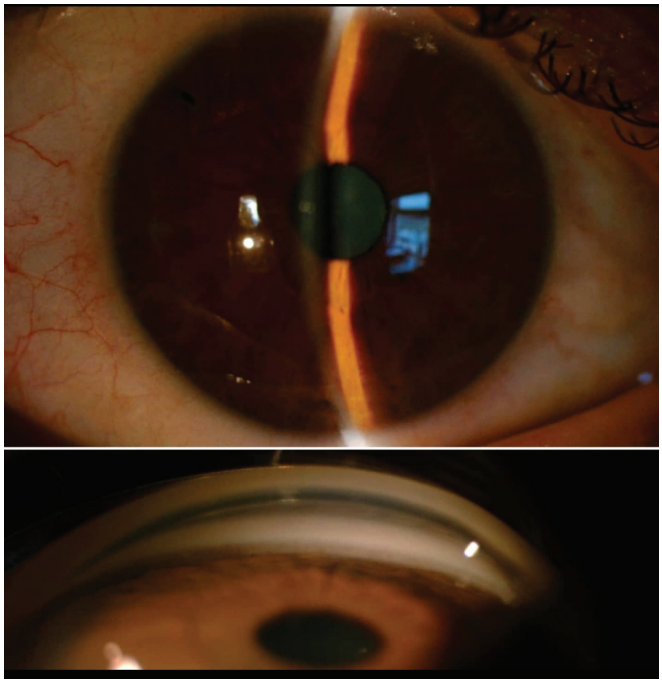

Figure 6: *Schaffer Grade 1 iridocorneal angle in gonioscopy at first week control.*

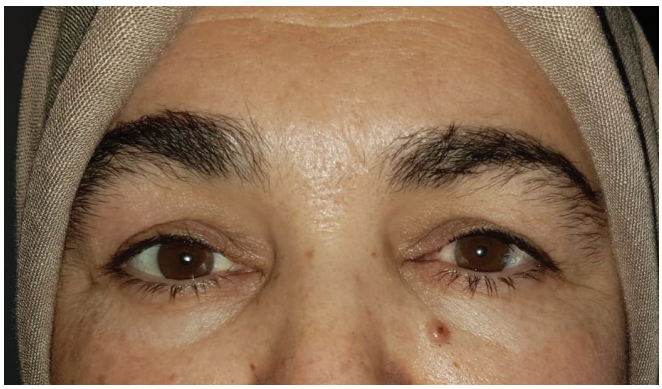

Figure 7: *Eye and ocular adnexes inspection at first week after treatment.*

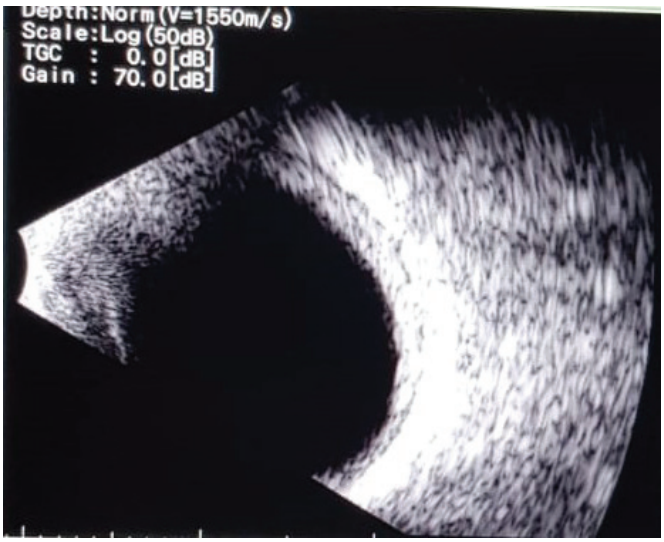

Figure 8: *Decreased scleral thickenning after treatment at first week control.*

warning sign. Unnecessary LI treatment is not rare in this patients similar to our case.[5,6] In our case, both anterior chambers were shallow but asymmetric and gonioscopy showed grade 1 narrow-angle in the fellow eye. Therefore, we could differentiate whether the primary angle-closure attack had occurred due to shallow anterior chamber depth and narrow angle or secondary angle-closure had appeared after ciliochoroidal effusion related with scleral inflammation. We investigated the patient for the first time during first attack and we could not reach any additional data about previous ocular condition. There were no histories of a red eye, ocular pain or visual loss those suspected us about prior attack on patient's and family history. Although the angle was asymmetrically narrow in both eyes, iridocorneal contact (up to three-quadrants) was not observed. Thus, this patient has not been defined as primary angle closure suspect and the beneficiary effect of cycloplegics rather than antiglaucomatous therapy alone also supported the secondary angle-closure glaucoma.

Increased IOP is found in 12-46% of cases and is associated with a variety of mechanisms such as increased viscosity of the aqueous humor, inflammation of the outflow pathways, trabecular meshwork obstruction by inflammatory cells and debris, elevated episcleral venous pressure, peripheric anterior synechia (PAS) formation, neovascular glaucoma and angle-closure glaucoma caused by choroidal effusion.[1,7,8] The mechanism of the development of secondary angle-closure glaucoma has been suggested to be initiated by posterior scleritis which can cause accumulation of suprachoroidal and supraciliary fluid due to inflammation and vascular congestion. Indocyanine green angiography (ICG) findings in eyes with posterior scleritis show diffuse zonal choroidal hyperfluorescence due to pinpoint leakage in approximately 50% of such cases and an enlargement of the draining choroidal veins. ICG results showed that vascular hyperpermeability may be induced by congestion of choroidal veins by the thickening of the sclera.[9] As a result, the ciliary ring narrows, the ciliary body and iris root rotates anteriorly into the angle, and lens-iris diaphragm displaces anteriorly.[3] Therefore, sympathomimetics may aggravate the anterior displacement of lens-iris diaphragm. Ugurbas et al presented a case misdiagnosed as angle closure glaucoma, the patient had been used pilocarpin HCL 2% for 3 days.[6] In the presented case, the shallow anterior chamber, angle closure and increased IOP is declared similar to mentioned mechanisms. Cycloplegic drop was immediately appiled to widen ciliary ring. Because of the pathophysiology of disease is related with vascular congestion and hyperpermeability, the recovery of anatomical changes takes time until 13 days [10] even if IOP is in normal range.

In conclusion, posterior scleritis may recur if diagnosis is delayed and may lead to sight-threatening complications if appropriate treatment is not provided. Early diagnosis is significant because appropiate treatment is successful in bringing about a rapid recovery of the condition and preventing severe ocular morbidities.

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