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Key words: Acute angle-closure, AION, optic atrophy, panuveitis, VKH

A 27-year-old woman presented with pain, redness, and diminution of vision in the left eye (LE) for two days. On presentation, the best-corrected visual acuity (BCVA) was 20/20 in the right eye (RE) and 20/125 in LE. Intraocular pressure (IOP) was 14 mmHg and 26 mmHg in RE and LE (on anti-glaucoma medications), respectively. On examination, RE was unremarkable, and LE showed ciliary congestion, shallow anterior chamber, keratic precipitates, grade-2 cells, and a mid-dilated pupil. Gonioscopy revealed open angles in RE and grade-1 narrow angles in LE. The fundus examination of LE showed a cup-disc ratio of 0.9:1 with multiple serous retinal detachments in the posterior pole [Fig. 1]. Fluorescein angiography, ultrasonography, and optical coherence tomography findings [Fig. 2] were suggestive of probable



Figure 1: Fundus photographs of (a) uninvolved right eye and (b) left eye demonstrating a clear vitreous, a large and deep optic cup (cup disc ratio of 0.9:1) with multiple serous retinal detachments in the posterior pole

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Received: 03-Apr-2020 Accepted: 07-Jun-2020 Revision: 28-May-2020 Published: 20-Aug-2020 20/32, IOP was 10 mmHg, the anterior chamber was deep and quiet, and fundus showed complete resolution of serous detachments [Fig. 3]. A visual field test revealed a reproducible superior arcuate scotoma in LE suggestive of nonarteritic anterior ischemic optic neuropathy (AION). On completion of the course of steroids, immunosuppression therapy was initiated. At one and half year follow-up, LE BCVA was 20/32 and the visual field defect remained stable. Her right eye remained uninvolved over the entire follow-up period.

Discussion

VKH can rarely mimic acute angle-closure glaucoma (AACG) due to ciliary body swelling and suprachoroidal fluid accumulation.^[1,2] The visual field defect could not be attributed to glaucomatous damage, as she had presented after a day of developing angle-closure, suggesting possible previous episodes of sub-clinical inflammation of panuveitis. It could be attributed to AION, which though rare, has been reported



Figure 2: (a) Fundus fluorescein angiography of the left eye showing multifocal points of pinpoint leakage in the early venous phase and (b) pooling within the subretinal fluid in the late phase. (c) B-scan ultrasonography demonstrating thickening of the posterior choroid (d) Optical coherence tomography showing multiple serous retinal detachments

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Figure 3: Sequential fundus photographs and optical coherence tomography (OCT) images of the left eye taken at presentation and after initiating corticosteroids. (a) Fundus photograph and OCT at presentation showing multiple serous retinal detachments, (b) 3 days, (c) 2 weeks, demonstrating a complete resolution of serous retinal detachments, and (d) 2 months follow-up

in patients less than 40 years of age with no vasculopathic risk factors.^[3] Eyes with AION may not always show altitudinal visual field defects but also a variety of visual field defects depending upon the involvement of the branches of the posterior ciliary artery. Association of VKH with AION is

known but rare, all of which manifested in the fifth and sixth decades of life.^[4,5] Our case shows a unique presentation of VKH associated with AION as AACG in a young woman.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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