Dear Editor,

We present a case of bilateral choroidal detachment (CD) and exudative retinal detachment (ERD) following laser peripheral iridotomy (LPI) in a patient of ocular Vogt-Koyanagi-Harada’s disease (VKHD). A 42-year-old lady presented to us with the complaint of sudden onset of pain, redness, and dimness of vision in her right eye for past two weeks. Her best-corrected visual acuity (BCVA) was 20/40 and 20/30 in right and left eye respectively. There were few large granulomatous, mutton fat type keratic precipitates (KP) in both eyes. Anterior chamber had grade 1+flare in both eyes and occasional cells in left eye and grade 1+cells in right eye. Pupil in left eye was 3 mm in diameter with normal reaction. Right eye pupil was 4.5 mm in diameter, irregular, mid-dilated, not reacting to light with posterior synechiae. Intraocular pressures (IOPs) were 42 and 14 mm Hg in right and left eyes respectively. Gonioscopy showed occludable angles with peripheral anterior synechiae (PAS) in both eyes.

Approval was taken from the Ethics Committee of our hospital and the study was conducted in accordance with the principles of the Declaration of Helsinki. Informed consent was taken from the patient.

Fundus examination showed depigmentation of fundus with an orange-red discoloration with presence of retinal pigment epithelial clumps on the posterior pole in both eyes (Figure 1A-1B). The cup-disc ratio (CDR) was 0.3 in both eyes. There was no history of penetrating ocular trauma or surgery. These features were suggestive of probable, ocular (incomplete) VKHD since there were no associated cutaneous or neurological findings. A diagnosis of acute attack of angle closure in right eye in a patient of probable ocular (incomplete) VKHD in both eyes was made. Yttrium-Aluminium-Garnett (YAG) LPI in both eyes was done. Two LPIs were done in right eye at 2 o’clock (5.2 mJ, 3 shots) and 10 o’clock (5.2 mJ, 4 shots) positions and one LPI was done at 3 o’clock position (5.0 mJ, 4 shots) in left eye. Patient was prescribed prednisolone acetate ophthalmic suspension, United States Pharmacopeia (USP) (1%), a fixed-dose combination of brimonidine tartrate 0.2% w/v and timolol maleate 0.5% w/v eye drop in both eyes, oral acetazolamide tablets (250 mg), one tablet twice daily for 5d and tablet prednisolone, 40 mg/d for one week.

At the follow-up visit after five days, the patient complained of further dimness of vision. On examination her BCVA was 20/120 in right eye and 20/80 in left eye. IOPs were 16 and 14 mm Hg in right and left eyes respectively. Anterior chamber reaction had subsided in both eyes. All LPIs were patent. Fundus examination showed CD inferiorly along with exudative-non-rhegmatogenous retinal detachment in both eyes. Submacular fluid was noted on examination of the posterior pole. Optomap retinal examination (OPTOS, wide-field retinal imaging) confirmed the clinical findings (Figure 1C-1F). Spectral domain optical coherence tomography (SD-OCT) showed submacular fluid along with choroidal undulation in both eyes. Central foveal thickness (CFT) was 852 µm in right eye and 458 µm in left eye (Figure 1G and 1H). Axial lengths were 22.56 and 22.71 mm in right and left eye respectively. Gonioscopy showed occludable angles with peripheral anterior synechiae (PAS) in both eyes.

After one week, her BCVA had improved to 20/30 in right eye and 20/40 in left eye. IOP was 14 mm Hg in both eyes with patent LPIs (Figure 2A and 2B). OPTOS imaging showed almost complete resolution of the CD and ERD (Figure 2C-2F).
SD-OCT images showed marked resolution of submacular fluid and choroidal undulation in both eyes (Figure 2G and 2H). CFT had reduced in both eyes. Topical and oral steroids, atropine eye drops, and anti-glaucoma medication were continued. At last follow up her BCVA was 20/20 in both eyes. The submacular fluid and the CD and ERD had completely resolved. She was advised regular follow-ups at Retina and Glaucoma Departments.

We know that VKHD is a systemic condition which is associated with a non infectious granulomatous panuveitis. In 2001, the first International Workshop on VKHD proposed diagnostic criteria that rely on specific ocular and systemic criteria. In its complete form, VKHD is defined as a nontraumatic bilateral panuveitis and is associated with integumentary, neurological/auditory signs. Probable (ocular) VKHD is characterized as matching ocular manifestations but in the absence of extraocular manifestations.

Incidence of glaucoma noted in cases of VKHD is between 6%-45%. In VKHD, ocular hypertension, open angle glaucoma, angle-closure glaucoma, glaucoma due to combined mechanisms and secondary glaucoma have been described. Potential mechanisms of glaucoma secondary to uveitis include blockage of the trabecular meshwork with
inflammatory cells, prostaglandin-induced IOP elevation, swelling of the trabecular meshwork (trabeculitis), peripheral anterior synchiae formation, pupillary block with secondary angle closure and corticosteroid induced IOP elevation[4-5]. In our case the chronic anterior-chamber inflammation was probably the cause for anterior rotation of the ciliary body and subsequent pupillary block and angle closure. Acute angle-closure has been noted to be the presenting manifestation of VKHD in a few reported cases as was seen in our case[6].

If a patient of VKHD presents with a shallow chamber with normal or moderately raised IOP with no PAS then treatment with topical and oral steroids and mydriatic-cycloplegic eye drops results in resolution of the episode[4]. If however the presentation is with pupillary block, angle closure, raised IOP and presence of PAS, the treatment is on the lines of management of an acute attack of angle closure and intervention in the form of LPI, surgical peripheral iridotomy or trabeculectomy may be required[46]. Cases of VKHD with angle closure may have CD with ERD preintervention, that is, before an LPI is done[6]. Our patient had no evidence of ERD or CD at presentation. There is no evidence in literature of the development of bilateral CD and ERD post YAG laser iridotomy in a case of VKHD as was seen in our case.

The age of the patient, axial length, refractive correction, amount of laser energy used, IOP at the time of the procedure, medications used pre-procedure and post-procedure were nothing out of the ordinary and could not shed any light as to the probable cause. The close temporal relationship between the procedure and the appearance of subjective symptoms suggests that probably Nd:YAG LPI had some role in the development of the CD and ERD in this VKHD patient. Uveal effusion or ERD can and does occur fairly commonly in nanophthalmic eyes spontaneously or following laser treatment. Acute angle closure has been noted to be the presenting manifestation of VKHD in a few reported cases as was seen in our case[6].

This case brings to light a lot of important issues. It shows that CD and ERD may occur in a case of VKHD presenting with pupillary block after an LPI has been performed. Thus even though at presentation there may be no evidence of an ERD or CD, the possibility of such an event occurring after the laser procedure should be kept in mind. The possibility of subclinical effusion being present in untreated eyes with primary angle-closure disease, nanophthalmos and probably in eyes with VKHD should also be kept in mind. Thus the possibility of an uveal effusion developing in these eyes post laser iridotomy may not be as uncommon as it is thought to be. Even though most of these effusions are subclinical there is the chance that a clinically significant effusion may develop in a small percentage of these patients.

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