Vogt–Koyanagi–Harada disease in a patient of chronic myeloid leukemia

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This case report describes the concurrent development of Vogt–Koyanagi–Harada (VKH) disease in a 39 year old male patient of chronic myeloid leukemia (CML). The patient being reported was a known case of CML in remission with history of painless sudden loss of vision in both eyes. Cases of leukemia can present with visual loss due to multiple ocular manifestations of leukemia itself or side effects of modern drugs used for its treatment. Clinical examination and multimodal imaging of our patient were suggestive of concurrent development of VKH. The patient was started on oral steroids, to which he showed a good response. Thus, the cases of CML may rarely develop concurrent ocular disorders not related to leukemia. These associated ocular disorders need to be distinguished from the ocular manifestations of leukemia itself. Our case highlights the concurrent development of VKH as the etiology of visual loss in a case of CML.

Key words: Chronic myeloid leukemia, imatinib, Vogt–Koyanagi–Harada disease

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A significant number of cases of leukemia have changes in the fundus. The presence of serous retinal detachments is rare in association with leukemia. Serous retinal detachments have been described only in a few cases of acute leukemia.[1-5] To the best of our knowledge, there is just one report of myelogenous leukemia with bilateral exudative detachments.[6] We report a 39 year old male patient of chronic myeloid leukemia (CML) who presented with bilateral serous retinal detachments associated with prodromal neurologic and auditory symptoms suggestive of acute Vogt–Koyanagi–Harada (VKH) disease.

Case Report

A 39-year-old male, who was a known case of CML, presented with sudden painless visual loss in both eyes. There was an associated history of intermittent headache, fever, and tinnitus. There was no history of dysacusia. The patient was on treatment with imatinib mesylate since 2012. At present, he was in remission as his last breakpoint cluster region-abelson assay done a month back was 0%. His recent blood counts were also within normal limits (Hb = 14.2 g% [13.5–18 g%], TLC = 7900/cu mm [4000–10,000/cu mm], and platelets 229,000/cu mm [150,000–400,000/cu mm]). Audiometry was not performed.

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Best-corrected visual acuity was finger counting at 1 m in the right eye (oculus dextrus [OD]) and 20/30 in the left eye (oculus sinister [OS]). Fundus examination revealed multiple areas of neurosensory detachments and focal areas of multiple confluent yellow-colored placoid choroidal lesions in both eyes [Fig. 1]. A swept source optical coherence tomography (OCT) scan revealed neurosensory detachments with reflective subretinal fluid and subretinal septae with a few vitreous cells. Internal limiting membrane (ILM) irregularity/fluctuations were noted [Fig. 2]. The choroidal thickness was 533 μ OD and 458 μ OS. Fluorescein angiography (FA) in the early phase showed delayed choroidal filling and hypofluorescence of the placoid lesions at the macula, followed by multiple hyperfluorescent pinpoint leaks in the mid-phase which gradually enlarged with pooling of the dye in the subretinal space [Fig. 3]. The optic discs of both eyes showed mild hyperemia of the nasal halves with corresponding mild hyperfluorescence on FA.

A provisional diagnosis of incomplete VKH disease in a case of CML was considered, and the patient was started on oral steroids (prednisolone at a dose of 1.5 mg/kg body weight). After 2 weeks, the patient's vision improved to 20/20 in both eyes. The serous detachments resolved bilaterally, and the choroidal thickness also reduced (378 μ OD and 298 μ OS) [Fig. 4]. The steroids were tapered slowly over 6 months. At 6-month follow-up, the patient is stable with a vision of 20/20 in both eyes.

Discussion

The ophthalmic manifestations of CML are quite variable and include intraretinal hemorrhages, Roth spots, nerve fiber layer infarcts, subhyaloid and vitreous hemorrhages, and papilledema secondary to raised intracranial pressure. The cases of leukemia can also loose vision due to ocular side effects of imatinib. The various reported ocular side effects of imatinib therapy are periorbital edema, epiphora, recurrent subconjunctival hemorrhage, glaucoma, and optic neuritis.[7,8] Apart from one case of serous detachments in a patient of myelogenous leukemia,[6] there are no reports of

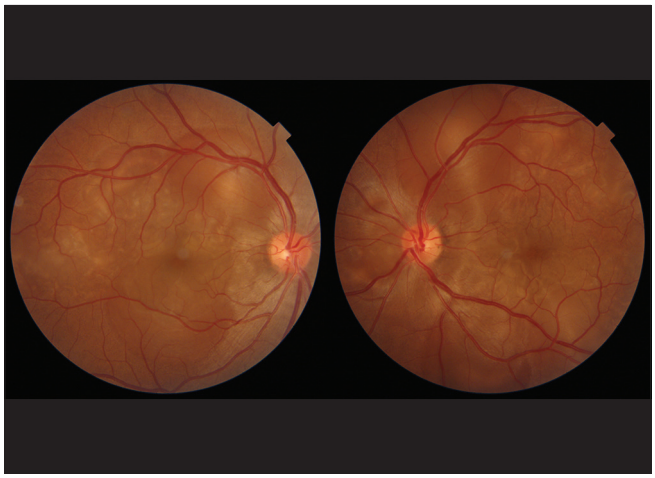

Figure 1: Fundus photographs showing multiple areas of neurosensory detachments and focal areas of multiple confluent yellow-colored placoid choroidal lesions in both eyes

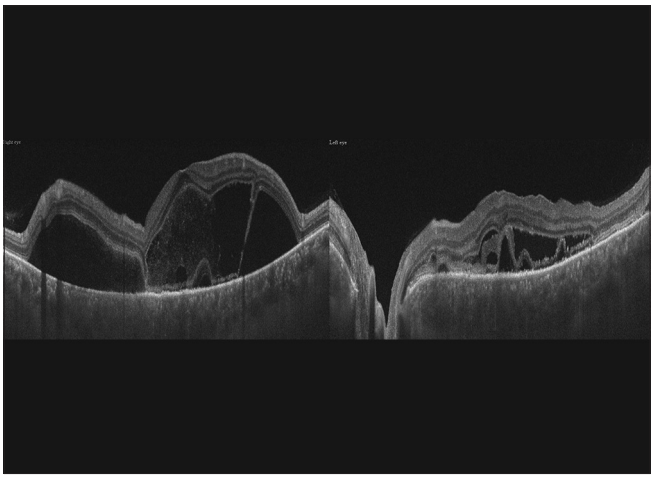

Figure 2: Optical coherence tomography scan showing the neurosensory detachments with reflective subretinal fluid and subretinal septae in both eyes. Internal limiting membrane irregularity/fluctuations were also noted

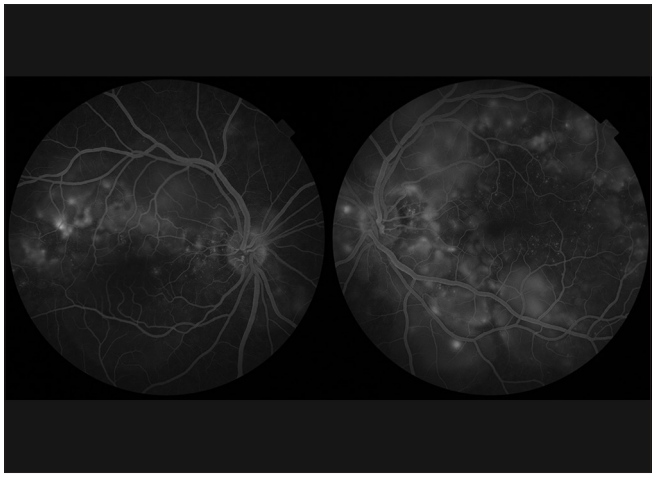

Figure 3: Late phase of fluorescein angiography of both eyes showing multiple hyperfluorescent leaks with associated pooling of the dye in the subretinal space

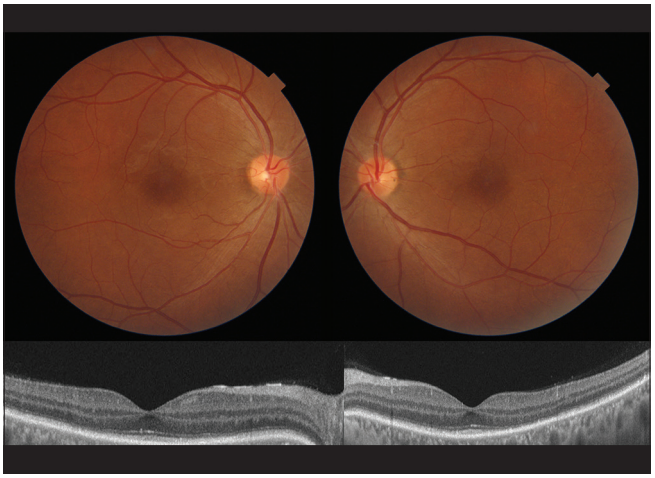

Figure 4: (Top) Follow-up fundus images showing resolution of the choroidal lesions and the neurosensory detachments. (Bottom) Follow-up optical coherence tomography scans showing normalization of the foveal contour with resolution of the subretinal fluid in both eyes

serous detachments in cases of CML or patients on imatinib therapy.

Our patient, who was a known case of CML in remission on treatment with imatinib, developed bilateral sudden painless loss of vision due to the development of bilateral serous detachments.

VKH disease presents as a bilateral panuveitis associated with exudative retinal detachments. The prodromal features consist of a viral fever like illness, with features of meningismus, neck stiffness, and headache associated with auditory symptoms such as tinnitus and dysacusia. The acute phase is characterized by choroidal infiltration and development of neurosensory detachments with granulomatous anterior uveitis. FA demonstrates characteristic features of focal areas of delay in choroidal perfusion, multifocal areas of pinpoint leakage, large placoid areas of hyperfluorescence, and pooling within subretinal fluid. Typical OCT features of VKH include neurosensory detachments with subretinal septae and ILM fluctuations.[9,10] Considering the presence of typical prodromal symptoms with bilateral serous detachments and typical FA and OCT features of VKH, we made a diagnosis of concurrent onset of incomplete VKH disease[11] in a case of CML. Since our patient was in remission, we do not attribute the development of the serous detachments to leukemia itself. The patient also responded well to immunosuppressive therapy with steroids alone and there has been no other systemic evidence of relapse of CML.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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