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Case report

Vogt-Koyanagi-Harada disease like presentation in patients with chronic myeloid leukemia

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ABSTRACT

Purpose: To report two rare cases of chronic myeloid leukemia (CML) on tyrosine kinase inhibitors presenting as bilateral serous retinal detachment and ocular inflammation, simulating Vogt-Koyanagi-Harada (VKH) disease.
Methods: Case series and review of literature.
Result: Two young patients (one male and one female) with CML on treatment with tyrosine kinase inhibitors (imatinib and dasatanib) under remission presented with bilateral sudden vision loss. One patient had bilateral multiple pockets of serous retinal fluid while the other had panuveitis with exudative retinal detachment. There was neither prodromal symptoms nor systemic signs and symptoms suggestive of VKH in both cases. They responded well to systemic steroid therapy without recurrences with complete visual recovery.
Conclusion and importance: CML patients can have features similar to VKH even during stable hematological phase and may be possibly associated with the use of tyrosine kinase inhibitors. Hence it is important not to misdiagnose and treat such patients with long term immunomodulators.

1. Introduction

Patients with leukemia often have ocular manifestations. These occur either from direct infiltration of neoplastic cells or from indirect causes, including hematologic abnormalities, central nervous system involvement, opportunistic infections, or from drug toxicity. Awareness of the ophthalmic manifestations of leukemia is important as they may precede the diagnosis of leukemia or can occur during the course of the disease.[1]

Although nearly all ocular structures can be affected, leukemic retinopathy is often the most clinically apparent manifestation. Typically, manifestations of leukemic retinopathy are florid, with vascular changes such as retinal vein tortuosity or obstruction, flame-shaped hemorrhages, dot-and-blot hemorrhages, Roth spots, and even optic nerve edema.

Serous retinal detachment is not commonly seen in patients with chronic myeloid leukemia (CML), although anecdotal cases in lymphoblastic leukemia have been reported.[2–8] There are no reports of such ocular presentation associated with drugs used in the treatment of CML especially tyrosine kinase inhibitors. We report two rare cases of CML on hematological remission who presented with bilateral serous retinal detachment simulating Vogt-Koyanagi-Harada (VKH) disease. These patients were on tyrosine kinase inhibitors namely imatinib and dasatinib and their possible role as the cause of ocular inflammation was considered.

2. Case details

2.1. Case 1

A 32-year young female, a known case of CML since 8 years on hematological remission presented to us with complaints of sudden bilateral painless loss of vision since 10 days duration. Her recent blood counts were within normal limits. She was on treatment with imatinib mesylate since the past 6 years. Her best corrected visual acuity in the right eye was 6/36, N18 and in the left eye was 3/60, N36. Anterior chamber was quiet and fundus examination of both eyes revealed hyperemic discs and multiple pockets of subretinal fluid in posterior pole along with multiple confluent yellowish placoid lesions in choroid simulating VKH. The patient did not have any similar ocular history in the past nor had any prodromal symptoms or neurological, auditory or integumentary signs or symptoms. Fundus fluorescein angiography of

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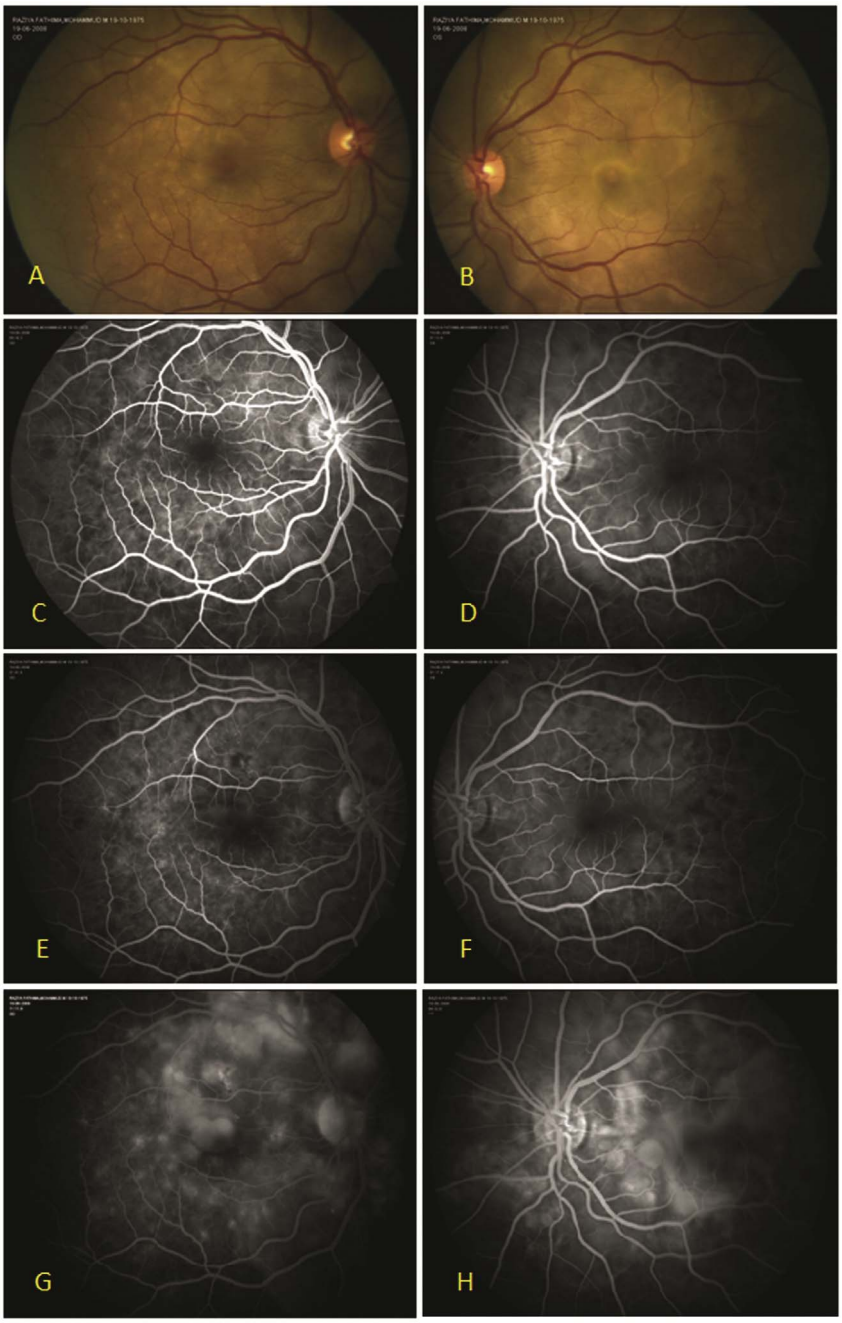

Fig. 1. Case 1 findings at presentation.
A & B) Fundus examination of both eyes shows hyperemic discs and multiple pockets of subretinal fluid in posterior pole along with multiple confluent yellowish placoid lesions in choroid; C & D) Fluorescein angiography of both eyes in early phase revealed hypofluorescent spots corresponding to the placoid lesions in choroid; E & F) followed by multiple hyperfluorescent pinpoint leaks in the mid phase; G & H) which showed pooling of the dye in the subretinal space during the late phase.

both eyes in early phase revealed hypofluorescent spots corresponding to the placoid lesions in choroid, followed by multiple hyperfluorescent pinpoint leaks in the mid phase which showed pooling of the dye in the subretinal space during the late phase. [Fig. 1]. Ultrasonography (USG) B scan of both eyes showed increased choroidal thickness and did not reveal any choroidal mass. Investigations revealed a normal chest X-ray and a negative Quantiferon-TB Gold (QFT-G) and Mantoux tests. Patient underwent bone marrow trephine biopsy which showed markedly hypocellular bone marrow. Although CSF analysis was not done during the ocular presentation, she was re-evaluated by oncologist and was found to be haematologically stable.

Patient was treated with pulse therapy of intravenous methyl prednisolone (IVMP) 1G for 3 days followed by tapering course of systemic steroids at 1 mg/kg body weight after hematologist and physician's clearance. BCVA improved to 6/6 with complete resolution of subretinal fluid in both eyes at 6 weeks. Oral steroids were tapered over a period of 3 months with complete resolution of inflammation. There was no reactivation of ocular inflammation and the patient continued to remain in hematological remission till the last follow-up period of 20 months.

2.2. Case 2

A 41-year old male, on treatment for CML since 10 years and in hematological remission, presented with complaints of sudden painful loss of vision in both eyes since 5 days. The patient did not have any