ejpmr, 2022, 9(8), 453-454

SJIF Impact Factor 6.222

EUROPEAN JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.ejpmr.com

Case Study

ISSN 2394-3211

EJPMR

ACUTE LYMPHOBLASTIC LEUKEMIA PRESENTING AS ACUTE VOGT-KOYANAGI-HARADA SYNDROME

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Article Received on 16/06/2022 **Article Revised on 06/07/2022** **Article Accepted on 27/07/2022**

ABSTRACT

We aim to describe a case of acute lymphoblastic leukemia (ALL) that presented as Vogt-Koyanagi-Harada syndrome (VKH). A 70-year-old man presented with vision loss starting 1 week ago. There was no prior systemic or ocular history. Visual acuity was 20/200 in both eyes. Anterior segment exam was also normal in both eyes. On funduscopy, foveal reflex was significantly reduced. On optical coherence tomography, subretinal fluid was evident in both eyes. Fluorescein angiography showed a hypofluorescent area compatible with subretinal fluid and multiple pinpoint hyper- and hypofluorescent dots surrounding the detached retina. After 4 days, patient was admitted to a general hospital due to spontaneous ecchymosis and melena. On complete blood count, there was a high white cell count, thrombocytopenia, and low hemoglobin concentration with a probable diagnosis of leukemia. On bone marrow biopsy, ALL with B-cell precursor was confirmed.

KEYWORDS: funduscopy, foveal reflex.

INTRODUCTION

Retinal manifestations are the most common ocular signs in leukemia, and they are divided into primary or secondary. The first category is due to malignant cell invasion, and the second category is due to complications of the treatment, infections, or blood alterations. Some of the secondary retinal manifestations of acute lymphoblastic leukemia (ALL) include retinal hemorrhages, Roth spots, and retinal ischemic manifestations, such as vascular occlusions and scattered cotton wool spots. Exudative retinal detachment is a rare manifestation in ALL.[1]

CASE REPORT

The patient was a 70-year-old man referred to our clinic with the chief complaint of decreased visual acuity starting 1 week ago. There was no prior systemic or ocular history. Visual acuity was 20/200 in both eyes. Intraocular pressure was 15 mm Hg in his right eye and 17 mm Hg in his left eye. On slit lamp examination, there was no conjunctival infection, the anterior chamber was normal, and there was mild cataract without significant refractive error in both eyes. There were trace cells visible in the anterior vitreous of both eyes. Optic disc was pink bilaterally with no atrophic changes. Foveal reflex was significantly reduced bilaterally. On optical coherence tomography (OCT), multi-lobulated retinal detachments were evident in both eyes. Fluorescein angiography (FA) showed a hypofluorescent area compatible with subretinal fluid and multiple pinpoint hyper- and hypofluorescent dots within the detached retina.There were no clear auditory or integumentary symptoms. First differential diagnosis were VKH and CSR. There also was no history of recent headache or neck stiffness. Other differential diagnoses were lymphoma and choroidal metastasis due to the patient's age. Our patient's old age (more than 60 years) was not typical for VKH or CSCR. As VKH is a diagnosis of exclusion, we decided to do a systemic workup and patient was empirically started on oral prednisolone 50 mg daily. After 4 days, the patient had been admitted to a general hospital due to spontaneous ecchymosis and melena. On complete blood count, there was a high white cell count, thrombocytopenia, and low hemoglobin concentration with a probable diagnosis of leukemia. On bone marrow biopsy, ALL with B-cell precursor was confirmed. Patient was started on chemotherapy (ALL induction regimen: cyclophosphamide, v L-asparaginase, and prednisolone), the patient's vision was improving, and 2 months later, the patient was on remission. His visual acuity improved to 160/200, and all subretinal fluid was absorbed in both eyes clinically. Patient examination during and after treatment was done in a general hospital by indirect fundoscopy without any other imaging, such as FA and OCT. Unfortunately, the patient expired 10 months after initial presentation.

DISCUSSION

Based on our literature review, there are few reports of leukemia with an initial presentation as VKH. In a similar case report, a 42-year-old man with bilateral

exudative retinal detachment presented with a first diagnosis of VKH, and, later, ALL was confirmed. One week after induction of systemic chemotherapy, visual acuity was recovered.[2] The choroid with its high blood supply can be a suitable tissue for circulating blast cells that can lead to choroidal infiltration with malignant cells. Choriocapillaris occlusion and consequent ischemia can cause retinal pigment epithelial dysfunction. On the other hand, leukemic cells secrete different factors that can cause increased exudation. This increased fluid exudation due to the tumoral nature of these cells and reduced resorption of subretinal fluid due to dysfunction of the retinal pigment epithelium can lead to exudative retinal detachment.[2,3] In this case, the first differential diagnoses were VKH and CSCR. Nevertheless, due to multiple pinpoint leakages on FA, bilateral involvement, and patient age, the VKH diagnosis was more probable. An important point that should be noticed by clinicians is that infiltration of leukemic cells in the choroid can lead to an undulating choroidal surface or sick-sea appearance in the choroid that can mimic retinal pigment epithelium undulation, which is an important feature of VKH especially in differentiating it from CSCR.[4] Another important point in leukemia-induced subretinal fluid is that none of the reported cases showed "subretinal septa" on OCT, which is generally evident in n typical VKH cases. VKH-like presentation can also happen in chronic myeloid leukemia as its first manifestation or even after disease remission.[5] An important criterion for the diagnosis of VKH is the absence of other systemic diseases, and VKH is a diagnosis of exclusion.[5] It is important to keep this in mind – especially in atypical presentations and older age – in order not to miss important life-threating underlying diseases. Leukemia and lymphoma should be considered in the differential diagnosis of VKH in atypical cases, as treatment with systemic corticosteroids and other immunosuppressive agents can change the disease presprompt lifesaving treatments.[6]

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