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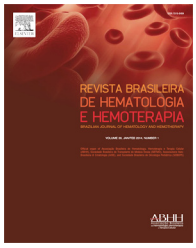

Case Report

Central retinal vein occlusion as first manifestation of relapse in acute lymphoblastic leukemia

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Introduction

Retinal vein occlusion is the second most common cause of visual loss due to retinal vascular disease after diabetic retinopathy.[1] Among the risk factors described, it is well known that the hypercoagulable state present in neoplasias can culminate in vein occlusion. It is described that any ocular structure may be involved in acute leukemia[2]; involvement of the choroid and retina is the most common. However, leukemic cells can infiltrate the conjunctiva and lacrimal glands, producing obvious masses. Karesh et al. published a two year prospective study of newly diagnosed adults with acute myeloid leukemia; 64% of patients had retinal or optic nerve abnormalities where hemorrhages and cotton wool spots (a consequence of nerve fiber ischemia) were the most frequent findings.[3] The association between central retinal vein occlusion and acute lymphoblastic leukemia (ALL) relapse has rarely been described though.[4–6] We hereby report a case of central retinal vein occlusion as the presenting manifestation of relapse in ALL.

Case report

A 59-year-old female was diagnosed with ALL in November 2013. At that time, she had the following laboratory results: hemoglobin: 8.7 g/dL; red blood cells: $2.95 \times 10^9/\mu L$; MCV: 89 fL; total leukocytes: $18.66 \times 10^9/L$; lymphocytes: $5.374 \times 10^9/L$; segmented neutrophils: $2.930 \times 10^9/L$; blasts: 9143cells/μL; uric acid: 5.2 mg/dL; lactic dehydrogenase: 1672 U/L; creatinine: 3.1 mg/dL; and urea: 83 mg/dL. She received volemic resuscitation for tumor lysis syndrome. Subsequently, she developed pneumonia caused by *Cryptococcus neoformans* and herpes zoster skin lesions. Treatment was made with meropenem, amphotericin and acyclovir. In January 2014, after completing treatment for the infections, she was submitted to chemotherapy induction using the hyperCVAD

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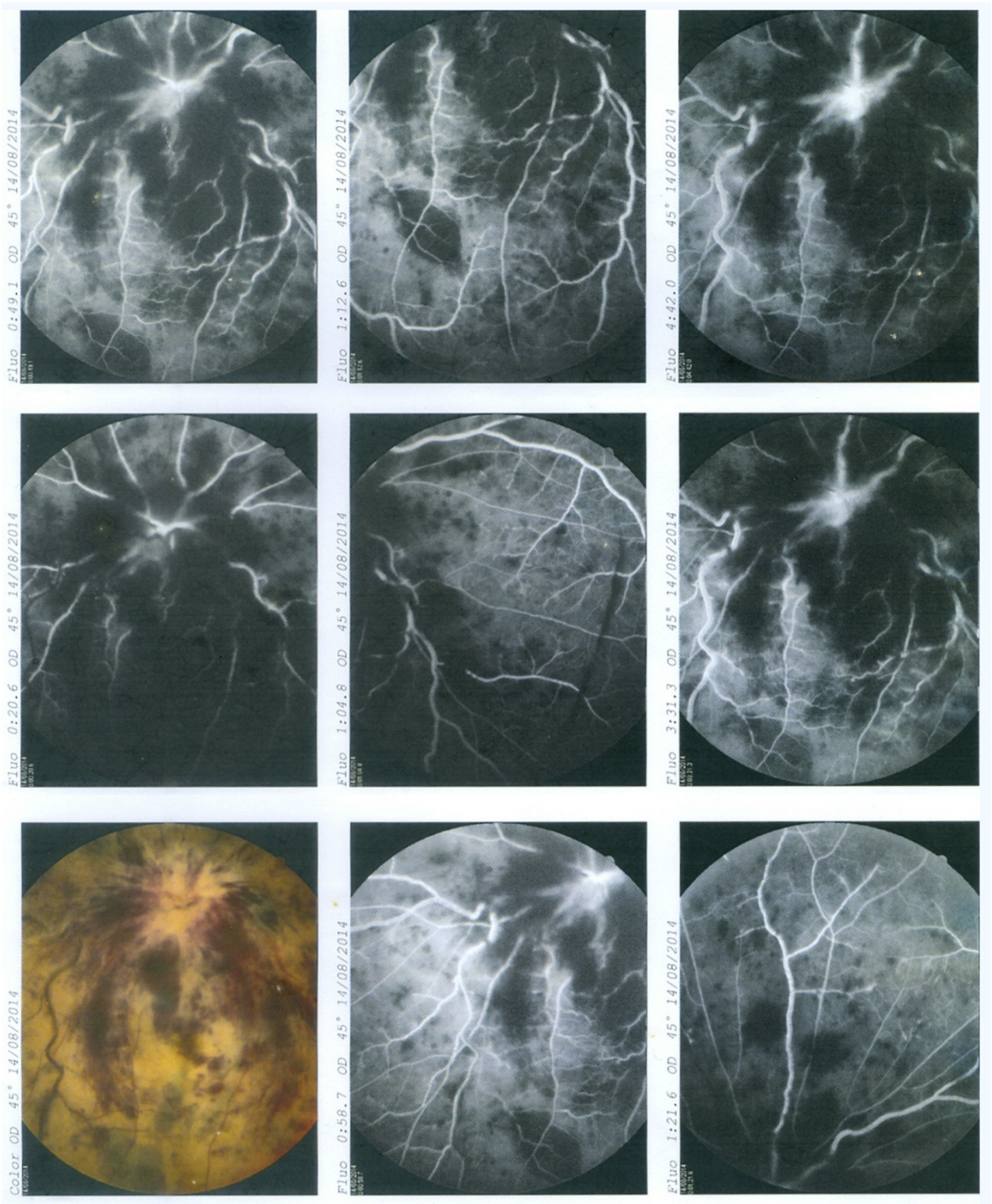

Figure 1 – Fluorescein angiography showing occlusion of central retinal vein.

regimen – course A (cyclophosphamide, vincristine, doxorubicin and dexamethasone). Immunophenotyping performed in February 2014 showed remission of the disease.

In March 2014 she presented febrile neutropenia and typhlitis. Empiric treatment with cefepime was started and the hyperCVAD regimen was interrupted. Blood cultures identified *Escherichia coli* and *Klebsiella pnemoniae*. Treatment was completed on April 20th 2014.

After hospital discharge, she was submitted to outpatient re-induction therapy with the adapted Berlin-Frankfurt-Munster (BFM) protocol (dexamethasone, vincristine, doxorubicin and L-asparaginase) in June 2014. In July she was unable