Retinal thrombosis secondary to the combination therapy of pegylated interferon and ribavirin for chronic hepatitis C virus infection. A rare complication

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INTRODUCTION

Despite the fact that there are new therapies for treating chronic hepatitis C virus (HCV) infection (boceprevir, telaprevir), the combination of pegylated interferon and ribavirin continues to be the standard therapy. However, this treatment is not exempt from adverse effects (1-4).

CASE REPORT

A 52-year-old man with chronic HCV genotype 2b infection was treated with 100 μg of pegylated interferon alpha-2b per week and 800 mg of ribavirin per day. He was asymptomatic before treatment. At 24 weeks, the patient was classified as a responder and 4 weeks after treatment he presented with left hemicranial headache and deteriorating vision on the left side. Fundus examination detected a generalized thinning of the arteriole and flame hemorrhages in the superior hemiretina with macular involvement (Fig. 1). Bilateral fluorescein angiography detected a delay in the inferior venous filling of the left eye. Abundant early phase hypofluorescent lesions due to blockage that were consistent with intraretinal blood, and microaneurysms in the macula were found (Fig. 2). Protein C and protein S levels were normal and the patient did not have Factor V Leiden. Ischemic occlusion of the superior venous branch of the central vein of the retina was diagnosed. Antiangiogenic therapy was prescribed (bevacizumab, a monoclonal antibody directed against vascular endothelial growth factor [VEGF]) every 6 weeks that resulted in a 70 % recovery of visual acuity.

There are fewer than 10 reports in the international literature on retinal thrombosis associated with interferon and ribavirin combination therapy (2-4). Previous studies suggest that interferon may cause immune complex deposition in the retinal vasculature that leads to retinal ischemia, congestion, and hemorrhage (4). The use of an anti-VEGF therapy is based on...
evidence of a relationship between the ophthalmologic vascular alterations during antiviral treatment and the circulating VEGF levels (5).

REFERENCES