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SHORT COMMUNICATION

Bilateral ischemic maculopathy in a patient with AIDS

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PURPOSE. *To describe ischemic maculopathy as a cause of sudden bilateral decreased vision in a patient with human immunodeficiency virus (HIV) infection.*
METHODS. *A 44-year-old HIV-positive woman presented with bilateral decreased vision and normal examination, except for pale maculae and retinal vascular tortuosity. Fluorescein angiography showed bilateral enlargement of the foveal avascular zone with perifoveal dye leakage.*
CONCLUSIONS. *Ischemic maculopathy is a potential cause of decreased vision in patients with acquired immunodeficiency syndrome, even in patients with immune sustained recovery. This condition can be almost totally reversible, in the absence of other concomitant ocular pathologies. (Eur J Ophthalmol 2006; 16: 761-63)*

KEY WORDS. *AIDS retinopathy, immune sustained recovery, ischemic maculopathy*

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INTRODUCTION

One of the most common ocular manifestations associated with human immunodeficiency virus (HIV) infection is acquired immunodeficiency syndrome (AIDS) retinopathy (1). This retinal microvasculopathy consists of cotton-wool exudates with or without intraretinal hemorrhages initially localized at the posterior pole. These manifestations can resolve in a few weeks, remaining asymptomatic. However, cytomegalovirus (CMV) retinitis is still the most common opportunistic ocular infection in AIDS (2). Several causes of decreased vision have been reported in these patients (3). We present a case report of a patient with transient poor visual acuity due to bilateral ischemic maculopathy.

Case report

A 40-year-old woman presented to the hospital with bilateral sudden decreased vision. She had been diagnosed with HIV infection 6 years before, when she had Pneumocystis pneumonia and constitutional syndrome with severe weight loss. Since then she had been on highly active antiretroviral therapy (HAART) with an optimal response to the treatment. Blood tests showed almost normality in white and red cells count. Lymphocyte count was within normal limits with CD4 lymphocyte 41.3% (normal range: 40–50) and CD4/CD8 ratio 0.95. CMV serology showed IgG antibody titers indicative of previous CMV infection, with undetectable IgM titers. Visual acuity was 0.3 and 0.2 in the right and left eye, respectively. Anterior segment was normal, and ophthalmoscopic examination revealed normal optic disc, vascular tortuosity with caliber changes, and pathologic arteriovenous crossings. The appearance of the macula was slightly pale without hemorrhages or any other alteration. None of the common CMV retinitis features were present. Fluorescein angiography performed at this first stage showed bilateral irregular enlargement of the foveal avascular zone (FAZ) with moderate perifoveal vessels leakage in the late phase. Ischemic areas affected at least 1 disc diameter and nearly all the macular area in both eyes (Fig. 1). The

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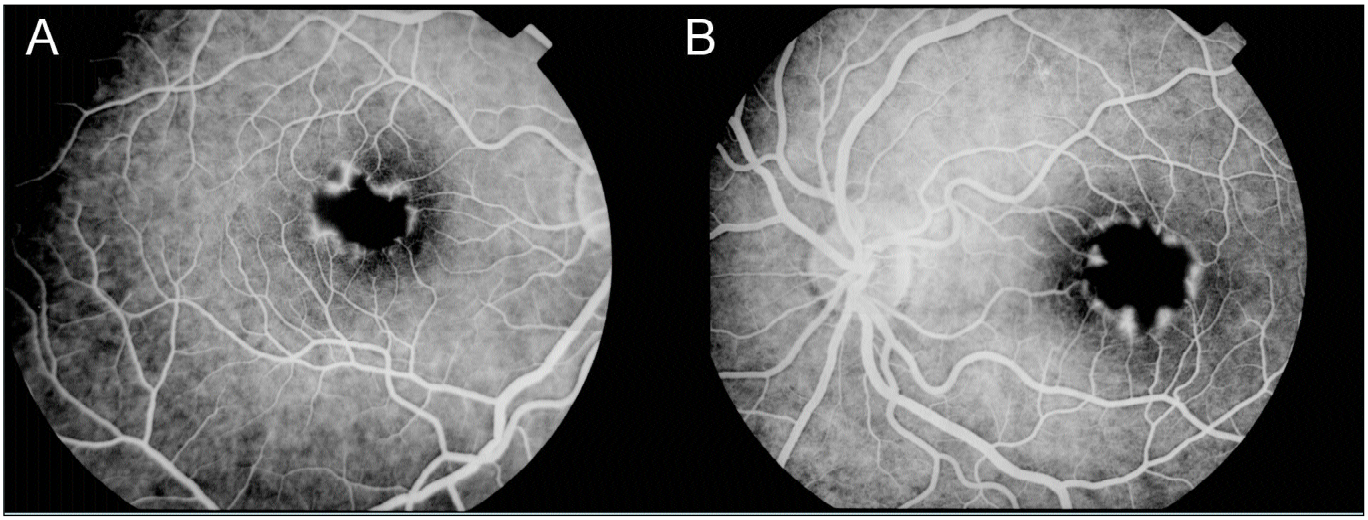

Fig. 1 - *Fluorescein angiography of the right* (**A**) *and left* (**B**) *eye of the patient, during the acute episode, showing enlargement of the foveal avascular zone and late staining of the perifoveal capillaries.*

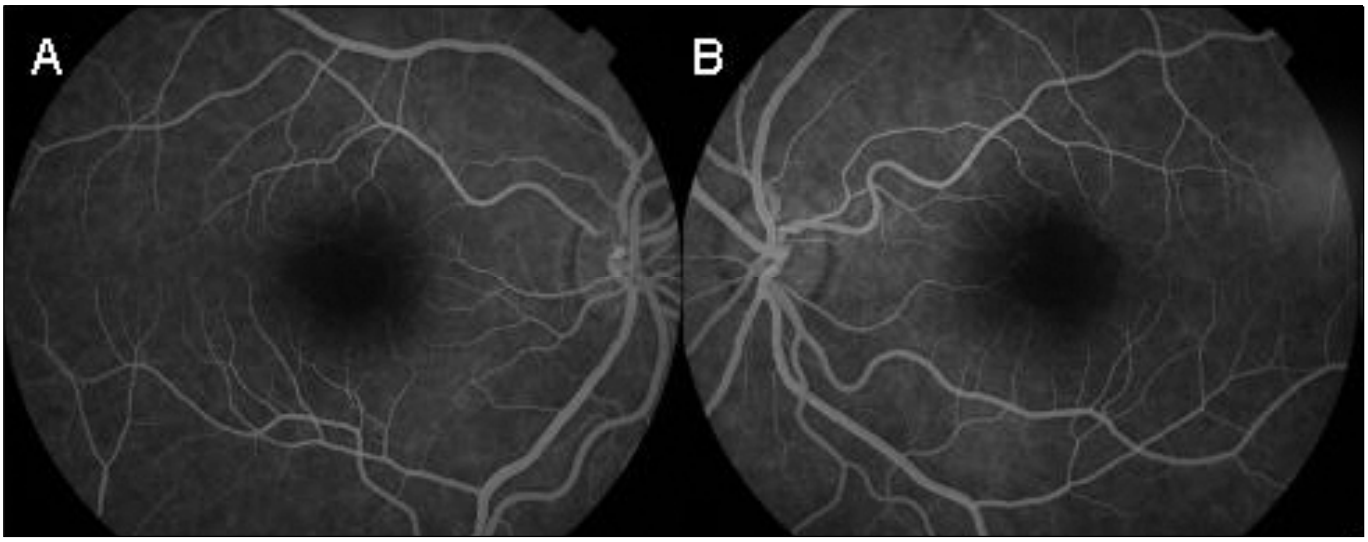

Fig. 2 - *Three months later, fluorescein angiography shows almost normality of the foveal area, with slight enlargement of the foveal avascular zone but no leakage from the capillaries.* (**A**) *Right eye,* (**B**) *left eye.*

patient was not given any treatment other than HAART and was reviewed 3 months later. On this later examination, visual acuity improved to 0.9 in the right eye and 0.8 in the left. Funduscopy returned to normal, and angiographically only a slight FAZ enlargement was noticed with disappearance of the dye leakage (Fig. 2).

DISCUSSION

Ischaemic maculopathy is a potential cause of sudden bilateral decreased vision in HIV-infected patients with immune sustained recovery. The pathogenesis is not known, though several theories have been proposed. The deposit of immunoglobulins on the retinal capillaries may play a key role in its development (4). Also, retinal vascular endothelial cells infection by the HIV with altered plasmatic fibrinogen levels, increased erythrocyte aggregation, and slower hematic flow with consequent macular hypoxia may contribute to the establishment of the microvasculopathy and subsequent focal macular ischemia. Cunningham et al (3) reported similar findings in five patients. Of the nine eyes described by them, 55% remained stable, 23% worsened, and the rest improved, with a final vi-

sual acuity ranging from 0.8 to counting fingers. It is interesting that most of their patients had concurrent CMV retinitis and poor systemic control before the HAART era. With no specific therapy other than the patient's current AIDS treatment, this condition can be almost totally reversible. This can happen in a short period, 3 months as in our report, if it is not accompanied by other retinal complications and the patient has immune reconstitution.

None of the authors has any proprietary interest in anything presented in this study.

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