LETTER TO THE EDITOR

Bilateral anterior ischaemic optic neuropathy in a patient with haemochromatosis

Neuropatía óptica isquémica anterior bilateral en paciente con hemocromatosis

Anterior ischaemic optic neuropathy (AION) is a frequent cause of painless sudden vision loss. There are 2 types of AION: arteritic and non-arteritic. Arteritic AION, which is mainly due to giant-cell arteritis (or temporal arteritis), requires early treatment with corticosteroids. Non-arteritic AION, on the other hand, is more frequent and has a multifactorial aetiology; no specific treatment is available for this type of AION. We present a case of bilateral non-arteritic AION in a patient with haemochromatosis.

Our patient was a 65-year-old man who visited the emergency department due to sudden vision loss, with onset of loss of part of the visual field in the left eye occurring 2 hours previously; he reported no other associated symptoms. The ophthalmologic examination evidenced a visual acuity of 0.6, vision loss in the lower hemifield, afferent papillary defect, and diffuse papilloedema with peripapillary haemorrhages in the left eye. The right eye displayed visual acuity of 0.9; the eye fundus examination revealed no alterations. After gathering the anamnesis (the patient reported no accompanying systemic symptoms), we conducted a thorough neurological examination and a complete analysis which revealed normal erythrocyte sedimentation rate and C-reactive protein levels. Our patient was diagnosed with non-arteritic anterior optic neuropathy and received no treatment. We scheduled a follow-up visit to conduct complementary tests. Six months later, our patient returned to the emergency department with the same symptoms in his right eye. An ophthalmologic examination revealed a visual acuity of 0.3 in the right eye and 0.5 in the left eye. There was partial vision loss in the lower hemifield of the right eye and complete vision loss in the lower hemifield of the left eye, afferent papillary defect and papilloedema involving the upper sector in association with haemorrhages in the right eye and papillary atrophy in the left eye. Subsequent examinations found no cardiovascular risk factors; a complete analysis ruled out coagulation disorders, congenital or acquired hypercoagulability, and presence of autoantibodies. A brain and spinal cord MRI study, an echocardiogram, and a Doppler ultrasound study of the supra-aortic trunks all revealed no alterations. CSF analysis yielded normal results. Additional, more thorough analyses revealed high transferrin saturation and elevated iron and ferritin levels in serum. These findings led to a diagnosis of haemochromatosis, which was subsequently confirmed by the presence of a homozygous mutation in C282Y. After more than 4 years of follow-up, our patient has experienced no further relapses and examinations reveal no changes.

Haemochromatosis is a systemic disorder caused by excessive absorption and accumulation of iron in body tissues. Although the most frequent form is hereditary, haemochromatosis can also be acquired. Since abnormal iron accumulation may interfere in oxygen radical generation and in the alteration of vascular endothelial cells, it could therefore increase the risk of infarction. In our case, vascular alterations may have affected the optic nerve and resulted in optic nerve infarction.

The literature has reported various ocular alterations secondary to iron accumulation, such as cataracts or Kayser-Fleischer rings in patients with keratoconus. To our knowledge, this is the first case of bilateral anterior ischaemic optic neuropathy in a patient with haemochromatosis as the sole risk factor.

Conflicts of interest

The authors have no conflicts of interest to declare.

References


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