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LETTERS 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.[1] 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.[2] 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 pupillary 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 pupillary 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.[3] 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.[4] To our knowledge, this is the first case of bilateral anterior ischaemic optic neuropathy in a patient with haemocromatosis as the sole risk factor.

Conflicts of interest

The authors have no conflicts of interest to declare.

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Comments on ''Role of intestinal microbiota in the development of multiple sclerosis''☆

Comentario del artículo «Papel de la microbiota intestinal en el desarrollo de la esclerosis múltiple»

Dear Editor,

It was with great interest that we read the review article by Drs Castillo-Álvarez and Marzo-Sola on the role of intestinal microbiota in the development of multiple sclerosis.[1] Their study constitutes a valuable contribution to the literature addressing the role of gut microbiota in multiple functions of the human body, especially immune response. However, we feel that the authors have not mentioned some of the topics addressed in other studies conducted by Spanish researchers and published in international journals. With a view to enriching the discussion and reflections on the role of microbiota in multiple sclerosis, and more specifically to complementing, if possible, the outstanding article by Castillo-Álvarez and Marzo-Sola,[1] we will address the omitted subjects, particularly the association between *Candida* species and multiple sclerosis.

The human microbiota has been reported to host some yeast species of the *Candida* genus.[2] Furthermore, several studies have suggested a link between cow milk and multiple sclerosis[3,4] and it is a well-known fact that yeasts of the *Candida* genus can be isolated from some dairy products.[5] Likewise, some rare cases of acute zonal occult outer retinopathy associated with multiple sclerosis and infections due to *Candida* species have been described in the literature.[6—8] These observations led us to hypothesise that *Candida* species may be associated with multiple sclerosis.[9] In that study, we obtained blood samples from 80 patients with multiple sclerosis and 240 age- and sex-matched controls.[9] According to our results, presence of *Candida* antigens was significantly associated with multiple sclerosis. More specifically, odds ratios (95% CI) were as follows: 2.8 (0.3-23.1; $P = .337$) for *Candida famata*, 1.5 (0.7-3.4; $P = .290$) for *Candida albicans*, 7.3 (3.2-16.6; $P < .001$) for *Candida parapsilosis*, and 3.0 (1.5-6.1; $P = .002$) for *Candida glabrata*. Results were similar after excluding those patients receiving immunosuppressants.[9] These findings led us to conclude that presence of *Candida* antigens was associated with increased risk of multiple sclerosis. Although the significance and scope of our results is difficult to determine, they lay the foundations for further research into this topic.

In any case, we wish to congratulate Castillo-Álvarez and Marzo-Sola on their excellent study and look forward to meeting them at a medical conference to further discuss these topics in person.

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