MIGRAINEOUS ANTERIOR OPTIC ISCHEMIC NEUROPATHY

NEUROPATÍA ÓPTICA ISQUÉMICA ANTERIOR MIGRAÑOSA

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ABSTRACT

Case report: We report the case of a 22-year-old man who presented with headache and blurred vision which had started four days previous. A periventricular lesion was found in the magnetic resonance imaging. The patient was diagnosed with demyelinating neuritis and treated with intravenous methylprednisolone.

Discussion: After six months visual function had not improved, so the initial diagnosis was probably erroneous. It is likely that the patient suffered from migraineous optic ischemic neuropathy. In this paper we review the scarce literature about this topic, and the role of migraine as a cardiovascular risk factor (Arch Soc Esp Oftalmol 2009; 84: 473-476).

Key words: Anterior ischemic optic neuropathy, demyelinating neuropathy, migraine with aura, migraineous infarction, cerebro-vascular risk factors.

RESUMEN

Caso clínico: Se presenta el caso de un paciente varón de 22 años de edad que acudió refiriendo visión borrosa y cefalea de cuatro días de evolución. Fue diagnosticado inicialmente de neuritis óptica desmielinizante ante el hallazgo en la resonancia magnética de una lesión periventricular, y tratado con metilprednisolona intravenosa.

Discusión: Ante la ausencia de mejoría de la función visual tras seis meses de evolución, es probable que el diagnóstico inicial fuera erróneo y que el paciente sufriera un cuadro de neuropatía óptica isquémica anterior migrañosa. Se revisa la escasa literatura publicada en relación con este tema y el papel de la migraña como factor de riesgo cardiovascular.

Palabras clave: Neuropatía óptica isquémica anterior, neuropatía desmielinizante, migraña con aura, infarto migrañoso, factores de riesgo cerebro-vascular.

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SHORT COMMUNICATION

CLINICAL CASE

A 22-year-old man visited the emergency section referring headache and blurred vision which had started four days before. As relevant personal history, the patient exhibited a clear family and personal history of migraine with aura. He practiced sport regularly and led a special sports diet including carbohydrates and protein supplements. His visual acuity (VA) was of 20/20 in
both eyes. Color vision was normal (Ishihara plates 9/9). He exhibited a slight relative afferent pupilar defect (1+/4+) in the right eye. The ocular fundus exploration revealed the presence of edema in the interior pole of the papilla and the corresponding nervous fiber layer in the right eye which was quantified with Optic Coherence Tomography (OCT). In the left eye, the papilla exhibited clear edges and only slight excavation. Computerized perimetry revealed the existence of a deep superior arch-shaped defect with very good correlation with the papillary exploration. All the lab parameters were normal except a lower level of HDL cholesterol and consequently a high rate show of total cholesterol to HDL cholesterol (lipid profile: VLDL 20 mg/dl, LDL 118 mg/dl, HDL 10 md/dl, total cholesterol/ HDL

![Fig. 1: Right eye retinography at diagnostic time, showing intense papilla edema circumscribed to the inferior pole (top). With ameritra light, the nervous fiber layer edema becomes more evident (bottom image).](image1)

![Fig. 2: The magnetic resonance shows a lesion which, considering the age of the patient, was initially labeled as probably demyelinizing.](image2)

![Fig. 3: superior arch-shaped defect in RE at diagnostic time and after three months, showing an extraordinary correlation between the papillary exploration and the Optic Coherence Tomography.](image3)
cholesterol 14,8). The rest of the neurological exploration was normal.

The patient was admitted to the neurology service and submitted to a complete body check-up including lumbar puncture. All parameters were normal. A cranial computerized tomography and brain magnetic resonance were performed which evidenced the presence of a non-specific peri-ventricular lesion in the frontal region. Considering the age of the patient, the condition was labeled as probably demyelinizing and treatment was established with intravenous methylprednisolone (1 gram/day) for three days.

In the subsequent exploration, the patient remained stable without evidence of significant changes in visual field or acuity. After three months the papilla edema was totally resolved with a loss of thickness located in the nervous fiber layer of the corresponding area. This evolution is typical of an anterior ischemic optic neuropathy, and accordingly the thrombophyllia study was increased (homocystein, functional antithrombine 2I, prothrombin 20210, lupic anticoagulant, V2 factor, Leiden Factor V, C protein and S protein), all producing negative results. The front lesion did not modify its appearance in subsequent neuroimaging studies.

**DISCUSSION**

This paper presents the case of a patient who visited the emergency section due to visual loss in the context of a migraine with aura episode. He was initially diagnosed with demyelinizing optic neuritis. The natural evolution of these processes is the spontaneous recovery of the majority of the visual function. This recovery is faster if the patient is treated with intravenous corticoids. However, the chronological relationship with the migrainous episode, the absence of pain with ocular movement at diagnostic time and above all the posterior evolution of the patient makes it much more likely that the patient had an ischemic condition (1).

Non-arteritic anterior ischemic optic neuropathy (NAION) is an entity which appears mainly in old patients, with the papillary morphology constituting the main risk factor and classic cardiovascular risk factors contributing in a proportion which has been the object of discussion in different studies. Recent papers show that ischemic optic neuropathy is not so rare in patients under 50 (2). However, most of the cases described in younger patients occur after surgical interventions involving an important loss of blood volume (mainly spine surgery) (3).

Migraine increases only slightly the risk of infarction in the general population because the weight of the remaining cerebral vascular risk factors is much greater. This low specific weight explains why, even though migraine is a highly prevalent disease, there are very few published cases of ischemia events of migrainous origin (4). However, excluding the conventional risk factors, in the young population the relative weight of migraine increases significantly. Some studies estimate 3 to 5 times more risk. Even so, nearly all published cases include other associated risk factors (5). Usually, said infarctions affect posterior circulation (the posterior cerebral artery) but the literature also describes ischemic events with the involvement of the retinal circulation as well as some ischemic optic neuropathy cases such as the one referred herein. The majority of the described ischemic neuropathy cases referred to anterior forms, but the literature also describes two cases of posterior ischemic neuropathy cases of migrainous origin (1).

We believe that in our patient the anatomy of the optic disc and high cholesterol values have behaved as risk factors, with the migraine episode becoming the final trigger of the ischemic event. Even though the sports diet the
patient followed did not comprise androgens, considering that the composition of dietary supplements was not very clear and the possibility of causal association, the patient has been recommended to discontinue the use of said supplements and hypolypemia and anti-aggregating treatment has been established. The front lesion did not change in subsequent neuroimaging studies and for this reason we consider that the lesion is probably ischemic in nature and related to the migraineous condition.

REFERENCES