LETTERS TO THE EDITOR

Optic neuropathy in a pregnant patient with antiphospholipid syndrome

Neuropatía óptica en gestante con síndrome antifosfolípido

Dear Editor,

Antiphospholipid syndrome (APS) is characterised by the presence of antiphospholipid antibodies associated with arterial or venous thrombosis or history of repeated pregnancy loss. The first manifestation of APS can sometimes be neurological, presenting as optic neuropathy, for example.1–4

We present the case of a 27-year-old woman (29 weeks pregnant), who visited the emergency department due to a sudden-onset decrease in visual acuity (VA) of the right eye (RE) first noticed 10 days previously. She did not report retro-ocular pain or headache. Nothing in her general medical or obstetric history was relevant to this event (G0P0A0). Visual acuity was 0.1 in the RE and 1.2 in the left (LE). Pupil examination revealed anisocoria with mydriasis and relative afferent pupillary defect in the RE. Fundus examination showed oedematous papilla in the RE, while results for the LE were normal. No alterations in chromatic perception

![Image of campimetry](http://www.elsevier.es/neurologia)

**Figure 1** 30° campimetry (Octopus visual field analyser) showing a lower altitudinal defect in the right eye and a mild diffuse reduction in sensitivity of the left eye.

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were observed. The remaining results of the neurological examination were normal.

Campimetry (30°) using an Octopus visual field analyser revealed significantly reduced sensitivity along the lower hemi-field of the RE (Fig. 1). Blood test showed high titres of anticardiolipin antibodies (lgG 46 U/mL) with no other alterations. The patient was diagnosed with anterior ischaemic optic neuropathy and APS-IIb given that only anticardiolipin antibodies were high. APS was considered primary since no concurrent rheumatic diseases could be identified. The patient was subsequently treated with antiplatelet drugs and heparin as an anticoagulant.

On her last ophthalmological follow-up visit one year later, the patient was pregnant again and on double thromboprophylactic treatment with subcutaneous heparin and oral aspirin. Altitudinal scotoma persisted, although oedema had remitted. Papillary pallor was manifest in the RE (Fig. 2).

Diagnosis of APS is based on presence of deep vein thrombosis, cerebral venous thrombosis, repeated pregnancy loss and thrombocytopenia, along with analyses that detect certain antibodies, including anticardiolipin antibodies. Although ocular vascular thrombosis is not frequent, and cannot be included among the classic clinical manifestations of this syndrome, ocular symptoms can occasionally appear as the initial signs. The ophthalmologist therefore plays a key role in preventing the serious consequences that patients with a late diagnosis can suffer.

Regarding clinical management of APS, it is essential to minimise other risk factors of thromboembolic events, such as obesity, dyslipidaemia, and smoking. Furthermore, studies demonstrate the utility of intensive anticoagulant treatment for reducing relapses in cases of venous or arterial thrombosis. Treatment should also be started in pregnant women with APS, and treatment must be maintained during the immediate period after childbirth. Pregnancy increases risk of recurring thromboembolic events, so miscarriages are frequent and probably due to infarctions in the uteroplacental vessels.

Recommendations based on study results generally point to anticoagulation with heparin together with antiplatelet therapy with aspirin, both in women with high titres of antiphospholipid antibodies and repeated pregnancy loss, and in pregnant women with APS with no history of obstetric events. Regarding optic neuropathy induced by APS, researchers have described that, in exceptional cases, anticoagulant treatment can revert optic neuropathy, leading to resolution of the campimetric defect and recovery of visual acuity.

Many authors agree that routine ophthalmological examinations should be performed in asymptomatic patients with APS, since ocular manifestations can be painless. Furthermore, in young patients without thromboembolic risk factors who present symptoms and signs of ocular vaso-occlusive disease, doctors should consider running a full diagnostic panel of the prothrombotic state.

References


A. Asorey-García∗, E. Santos-Bueso, F. Sánchez-Francés, J. García-Feijoo

Unidad de Neurooftalmología, Servicio de Oftalmología, Hospital Clínico San Carlos, Instituto de Investigación Sanitaria del Hospital Clínico San Carlos (IdISSC), Madrid, Spain

∗Corresponding author.
E-mail address: Almudena.asorey@gmail.com
(A. Asorey-García).

Figure 2 Image of the patient’s posterior pole showing significant papillary pallor in the right eye and normal papilla in the left.