

Papillary oedema: True or false?

Papiledema: ¿verdadero o falso?

Dear Editor:

We read the work "Papilloedema: true or false?" by Muñoz et al¹ with interest and, as a neuroophthalmologist and neurosurgeon, we would like to express some considerations about it. Firstly, we commend the authors for their clarity in the exposition of their ideas on this highly controversial and necessary issue. It is very important to establish, with scientific truth, when a case is really a papilloedema, which confronts the patient with the possibility that an intracranial tumour may be present; the precocity of this diagnosis is essential in disease evolution.

Pseudopapilloedema is a non-pathological elevation of the papilla that may occur in some disorders, especially congenital. Other causes of pseudopapilloedema which we must keep in mind are: tilted disc syndrome and oblique implantation of the papilla, disc hypoplasia, double optic disc, disc staphyloma, melanocytoma, disc coloboma, morning glory anomaly and astrocytic hamartoma.²⁻⁶

Papilloedema can also be mistaken for malignant hypertensive retinopathy when there is a history of arterial hypertension and haemorrhage and the white, cotton-like foci extend to the peripheral retina. In the occlusion of the central retinal vein, the condition is usually unilateral and associated with sudden and painless loss of visual acuity.² In

anterior ischemic optic neuropathy, disc oedema is pale non-hyperaemic and accompanied by a decrease in visual acuity in the form of stroke.

Other entities to be excluded are infiltrative processes (leukaemia, lymphoma) where pupil impairment is predominant. Compressive optic neuropathy, which may be caused by a meningioma of the optic nerve sheath, has the optociliary shunt vessel as pathognomonic sign. Papillitis is usually unilateral, with decreased visual acuity and pupillary alterations and is usually associated with pain upon ocular movements.^{2,7,8} Foster Kennedy syndrome, secondary to an olfactory groove meningioma, occurs with papillary oedema in one eye and optic atrophy in the other.^{2,9}

In relation to table 1, which sets out the ophthalmoscopic differences, we propose adding that the papilla is not hyperaemic in pseudopapilloedema—unlike in papilloedema. In recent years, we have worked with optical coherence tomography (OCT), and although the authors believe that this study “has not proved effective in differentiating an incipient papilloedema from a pseudopapilloedema, since in both cases there is an increased thickness of the nerve fibre layer of the retina”,¹ we believe along with other researchers^{3,10} that, although these measurements in a first consultation have not been useful in establishing differences between both entities, the evolutionary repetitions of the protocol used indeed manage to observe differences.

Finally, we celebrate the quality of the photographs that illustrate the text and we reiterate our gratitude to the authors; such reviews, which clarify knowledge about controversial issues, are very necessary for the proper development of neuroophthalmology and neuroscience in general.

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