Reply to: Papillary oedema: True or false?

Respuesta a: Papilelde: ¿verdadero o falso?

Dear Editor:

We thank Drs Fuentes-Pelier and Hodellín-Tallada for their interest in the review “P apilloedema: true or false?”. W e want to clarify that the fundamental purpose of our work is to describe the diagnostic approach for a clinical suspicion of papilloedema and the role of new technologies in this context. That is why we consider it appropriate to present two distinct clinical scenarios like those appearing under the headings “P apilloedema versus pseudopapilloedema” and “Oedema versus papilloedema”.

In the first section, we discuss the changes of the optic disc that may pose reasonable diagnostic doubt, especially at the stage of incipient papilloedema, such as buried drusen, full disc in hyperopia, nasal elevation of the myopic disc and myelin fibre presence. Papillary tumours such as astrocytoma and melanocytoma present differential characteristics (very dark pigmentation in the optic disc that partially or completely obscures the papillary margins, or a round lesion that may indicate a blackberry-like spot superimposed on the disc with intralesional calcifications, respectively) which, according to our experience, we do not need to include in the differential diagnosis of pseudopapilloedema (figs. 1 and 2). Furthermore, we do not think it appropriate to consider abnormalities in disc development (papillary coloboma, morning glory anomaly and peripapillary staphyloma) in the differential diagnosis for the same reason.

We agree that papillary oedema can be produced by multiple causes and that the differential diagnosis should be carried out with optic neuropathies that eventually appear with papillary oedema at some point in their evolution (ischemic, infectious, infiltrative, tumour or compressive). Therefore, we propose the second clinical scenario oedema versus papilloedema; however, the appearance of papillary oedema in itself may be non-
Other anomalies present in the fundus may indicate a different aetiology of intracranial hypertension, so we emphasise the importance of a fundus examination under mydriasis. Still, the main indicator that the papillary oedema under study is being caused by an optic neuropathy is the visual function impairment from the early stages. The history of acute or chronic visual loss, colour vision impairment, afferent pupillary defect or atypical findings in the fundus all count for neuropathy and against papilloedema. We believe that the description of neuropathies and their differential diagnosis are beyond the scope of this publication and refer readers to the text of Arruga et al. We have attached our proposed diagnostic approach to papillary oedema suspected of papilloedema in adults and children (Figs. 3 and 4).

Finally, with reference to the usefulness of optical coherence tomography, its main limitation in the differential diagnosis of papilloedema is that the current resolution...
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Figure 4. Papilloedema in adulthood. Proposed approach guide. CSF: cerebrospinal fluid; CT scan: computed tomography scan; FA: fluorescein angiography; MRI: magnetic resonance imaging.

Malignant arterial hypertension
Diabetic papillopathy
Arterial pressure/glycaemia

Bilateral papillary oedema

True papilloedema

Pseudopapilloedema
Ocular ultrasound/FA/serial retinography

CT: intracranial mass
CT: normal

Spinal tap
Arterial and venous MRI

Register pressure
CSF study

Venous thrombosis, arteriovenous malformation
Normal

Polysomnography
Sleep apnoea

does not differentiate between a thickening of the retinal nerve fibre layer from an increase in their number (pseudopapilloedema by full papilla), or from an intracellular oedema (in papilloedema and other neuropathies). Even in papilloedema monitoring, it should be interpreted with caution for the same reason, given that thinning or normalisation of the fibre layer thickness can occur either by resolution or by axonal loss.5

References


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