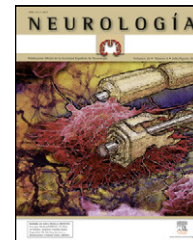




NEUROLOGÍA

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LETTER TO THE EDITOR

Paralysis of VI cranial pair after epidural anaesthesia. Reply[☆]

Paresia del VI par tras anestesia epidural. Réplica

Dear Editor:

It was with great interest that we read the study recently published by Durán-Ferreras¹ describing VI cranial nerve dysfunction secondary to intracranial hypotension (IH) syndrome. While the author clearly explains the physiopathological mechanism underlying VI cranial nerve dysfunction and other clinical manifestations typical of intracranial hypotension syndrome, he barely mentions the syndrome's characteristic radiological signs even though the clinical-radiological correlation is evident.²

Intracranial hypotension syndrome is a clinical-radiological entity resulting from cerebrospinal fluid volume depletion secondary to dural laceration.³ It is easy to assign a suspected diagnosis in the presence of clear clinical symptoms (especially orthostatic headache) and an obvious cause. However, cases exist in which doctors cannot identify the precipitating factor (idiopathic or essential intracranial hypotension syndrome).³

The case in question illustrates one of the radiological signs associated with intracranial hypotension: the presence of subdural collections.² Nevertheless, we should note that the key radiological finding in intracranial hypotension syndrome is diffuse dural thickening with enhanced gadolinium uptake.³ This may be the earliest sign of onset, and its persistence is directly related to headache duration.^{4–6} Other radiological signs associated with intracranial hypotension syndrome and already mentioned by the author¹ are rostrocaudal gradient and decreased size of subarachnoid cisterns.²

However, the author does not mention pituitary enlargement, which is another radiological finding consistently associated with intracranial hypotension syndrome.^{7–9} This less familiar radiological finding appears alongside dural thickening, and both processes share a common pathogenic

mechanism.⁸ Our group analysed pituitary enlargement in 11 consecutive patients with intracranial hypotension syndrome. We observed pituitary enlargement in all patients; following clinical improvement, the pituitary returned to its normal size.⁸ We should highlight that pituitary reverted to its normal size before dural thickening had resolved.⁸ The physiopathological cause underlying both radiological signs is alteration of cerebrospinal fluid secondary to intracranial hypotension. According to the Monroe-Kellie doctrine, cerebrospinal fluid hypovolaemia would result in a compensatory increase in blood volume, that of venous blood. Vasodilation in epidural venous sinuses increases pressure in the cavernous sinus and, in turn, the size of the pituitary. The same venous hyperaemia also causes diffuse dural thickening with gadolinium-contrast enhancement. Lastly, it is important to distinguish this sign from other pituitary anomalies such as adenomas or even physiological changes (such as pituitary enlargement occurring during puberty).^{7–9}

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