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Challenges in dual etiology diagnosis: probably idiopathic intracranial hypertension and systemic arterial hypertension in a case of headache with facial palsy

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Introduction
The facial palsy secondary to Idiopathic Intracranial Hypertension (IIH) is an uncommon clinical condition, with relatively few studies, mostly case reports - some pointing to a probable traction of the facial nerve or simply its edema due to intracranial hypertension. The Systemic Arterial Hypertension (SAH) is a known cause of cranial neuropathies, but it most often affects cranial nerves 3, 4, and 6 – furthermore, the facial palsy due to SAH is more common in children.

Objectives
The objective is to describe the challenges of the etiological diagnosis of facial paralysis with two possible pathophysiologies, whose treatments overlapped in time.

Case Report
JOS, female, 37 years old, hypertensive, taking valsartan 160 mg/day, reported waking up due to a new pattern of headache (she had previous migraine symptoms, with no red flags until then). She was admitted with BP 280x180 mmHg, non-specific visual clouding and peripheral facial palsy on the left, with no other alterations on neurological examination. Intravenous sodium nitroprusside was started under multiparameter monitoring. Brain CT was normal (hypoplasia of the left transverse sinus on angiotomography), and brain MRI showed a partially empty sella. Lumbar puncture had an opening pressure of 22 cmH2O, but with total and immediate improvement of the headache and partial improvement of PFP after the puncture. Cerebrospinal fluid, serologies, inflammatory and rheumatological tests were normal. After six days, her blood pressure and palsy improved, with prescription for acetazolamide and adjustments to her antihypertensive medication.

Conclusion
This case illustrates the challenge in encountering a headache with neurological deficit with two possible etiologies of fluctuating nature and with less common clinical manifestations. Diagnosis hinges on understanding the pathophysiology, the relationship with clinical signs, and the temporal connection, which can suggest two hypotheses that are not mutually exclusive and require simultaneous management due to their associated risks. Moreover, in this instance, intracranial hypertension may be "incipient", exacerbated by SAH, as blood pressure impacts intracranial pressure under both normal and pathological conditions.

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