



Seventh nerve palsy as a false localizing sign in benign intracranial hypertension

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DECLARATIONS

This case report identifies a rare clinical sign to a common condition and some hypotheses to the potential causes.

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Introduction

Rare clinical signs can pose a diagnostic conundrum to even the most experienced clinician. Most doctors feel safe in the knowledge that the myriad of clinical signs taught at medical school are key investigative tools in day-to-day practice. Clinical signs should be signposts to potentially altered anatomy or physiology, although in some cases, can lead even the good diagnostician down the wrong path.¹ Even in the simplest cases, the diagnostic jigsaw puzzle can be complicated by rarely-seen (or taught) false localizing signs. The term 'false localizing sign' may be self-explanatory, but what is not always clear is the level of potential deviation from 'normal' pathology that can occur. False localizing signs can potentially waste valuable time and resources directing investigation at a disease that the patient does not have. Furthermore, patients can be labelled with an inappropriate diagnosis which can have serious psychological or social connotations for the rest of their lives. The differential diagnoses entertained in this case could have been significantly life-limiting and potentially life-threatening for a patient of such a young age. It is, therefore, important to highlight unusual false localizing signs in order to inform clinicians that certain conditions may present with rare but clinically vital false clinical signs. It is understood that sixth nerve palsies are a reasonably common occurrence in benign intracranial hypertension (BIH) (10–30% of cases)² but there have been far less reported cases of sev-

enth nerve palsy^{3–5} and only one case of facial diplegia documented.⁶

A 19-year-old woman was admitted with a 10-day history of left-sided headache and vomiting which worsened over five days and developed into diplopia on extreme left gaze (denoting lateral rectus palsy even on admission). The headache was associated with photophobia and general malaise although there was no rash, neck stiffness or fever. Her menstrual history was unremarkable with no association to the headache and she had no significant past medical history.

Her BMI was >30 kg/m². Ophthalmoscopy revealed bilateral papilloedema with normal visual acuity and visual fields. The rest of the neurological examination was normal on admission. The following day, she developed bilateral lateral rectus weakness that was more pronounced on the left with concurrent lower motor facial nerve palsy on the left side (Figure 1).

A diagnosis of BIH was made and an urgent CT brain/venogram was normal. Baseline biochemical, haematological and immunological investigations were within normal limits, and a lumbar puncture yielded a clear and colourless CSF with 3 WBC and 1 RBC. Microscopy and culture of the fluid showed no evidence of infection. The opening pressure was measured at 35 cmH₂O and a closing pressure of 20 cmH₂O.

She was started on acetazolamide and within the next two days her signs were found to be completely resolved. Rapid resolution of her seventh nerve palsy within two days indicates the readily reversible nature of this lesion and is against concomitant Bell's palsy or simple arterial or venous brainstem infarct involving the sixth and seventh nerve nuclei residing in the Pons.

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Reviewer

Simon Green

Discussion

It is a well-established fact that due to the long course of the sixth cranial nerve it is particularly susceptible to raised intracranial pressure resulting in reversible dysfunction of the nerve.⁷ The anatomy and course of the seventh nerve, however, is less likely to manifest cranial nerve dysfunction in raised intracranial pressure. The intracranial portions of the motor fibres of the seventh nerve run a reasonably short course before they enter the petrous temporal bone of the skull. While localized there, it is relatively protected from the effects of pressure in the vault. There are far more common causes of seventh nerve palsy such as infection (viral and acute/chronic otitis media), Bell's palsy, congenital, neurosarcoid or trauma, but in this case the young woman demonstrated no clinical evidence of these causes so dysfunction of the nerve could indicate localized pressure effects in or around the seventh nerve nucleus. From the origin in the Pons, fibres of the seventh nerve wrap

around the nucleus of the sixth nerve. Any damage at the sixth nerve nucleus, could involve the lower motor fibres of the seventh nerve. Perhaps pressure effects of BIH are felt mostly in this area rather than over the petrous temporal bone tip which has been shown in the traditional teachings. As the seventh nerve fibres run in close proximity to the third cranial nerve nucleus, the lack of third nerve involvement can be explained by the diffuse nature of the intracranial pressure in BIH and distortion of the brainstem and facial colliculus.⁸

This young woman had moderate acne vulgaris over her face which had become a psychological and aesthetic problem for her. She had purchased over-the-counter spot preparations all containing multivitamin complexes before being prescribed two compounds by her GP (tretinoin and isotretinoin). Since she had never been on the contraceptive pill, two other possible aetiologies were an idiopathic nature to the condition, or hypothetically, the use of vitamin A containing face creams for her skin condition. Since it cannot be ascertained how much vitamin A the woman absorbed and the only link is excessive chronic vitamin A ingestion, this aetiology for her signs cannot be proved as causal but remains an interesting hypothesis in the development of BIH in this case.⁹ It should not be forgotten that obesity is also a known association with BIH and this lady had a high BMI. During further questioning it was revealed that she had used many branded face creams but had only used each one for approximately a week before changing to another product. The reason for her swapping topical application was a subjective poor efficacy of the non-prescription strength compounds. This hypothetically shows the possibility that mixing creams of varying vitamin A concentrations coupled with the patient's other risk factors for BIH may have contributed to the development of the clinical picture seen.

Figure 1
Evidence of left-sided seventh nerve palsy

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