Pitfalls in the recognition and localization of brainstem signs

I want to highlight a few observations about the case presented by Nasser and colleagues regarding a man with left facial droop, ataxia and vertigo.[1]

This patient's findings are interesting, but I would dispute the authors' claim that they are "pathognomonic for lateral medullary syndrome." Perhaps the best argument for this is that this patient's magnetic resonance imaging showed a lesion in the left lateral pons instead.

A lesion here, affecting the left vestibular nucleus, explains not only the nystagmus, but also the left head tilt, which is known as an "ocular tilt reaction." This phenomenon is not a "compensation for dysfunction of ocular alignment," as the authors assert, but rather an intrinsically pathologic response to unilaterally impaired static vestibular dysfunction.[2] In the setting of impaired graviceptive inputs from one vestibular nucleus, the brain "misperceives" gravity as pulling diagonally, and the eyes, head and body reflexively tilt in a maladaptive effort to stay "righted." The authors' Figure 1 depicts this left body tilt quite well, with the left shoulder lower than the right.[3]

I further disagree that this patient has left ptosis. The authors' Figure 3A depicts not left levator dysfunction (ptosis), but rather an overhang of redundant tissue in front of the lid (pseudoptosis). This is a common superficial mimic of ptosis called dermatochalasis, most often seen as a result of increasing age-related tissue laxity.[4] (The patient's true left eyelid is seen to emerge from behind the dermatochalasis during attempted eyelid closure, as depicted in Figure 3B.) Clinically, it is important to distinguish between dermatochalsis and true ptosis. One way to accomplish this is to gently lift this redundant tissue up and out of the way to assess the true position of the underlying eyelid.

This patient appears to have left eyelid retraction (the opposite of ptosis), as suggested in Figures 1 and 3A by following the contour of the eyelid — visible at the medial and lateral canthi — and extending this expected contour behind the overhanging redundant tissue. Ignoring the left dermatochalasis, the left eye seems wider open than the right. Lid retraction in facial palsy occurs from orbicularis oculi dysfunction. The bilateral frontalis sparing in Figure 2 indeed suggests a central localization; however, from a terminology standpoint, the phrase "a central cause of seventh nerve palsy" is an oxymoron and should be avoided in favour of "a central cause of facial weakness."

Although the authors are correct that Horner syndrome is common in certain brainstem syndromes and especially the lateral medullary syndrome, they are not correct in asserting that it occurs as part of Bell palsy (an isolated palsy of the seventh nerve) or with Weber syndrome (a third nerve palsy with contralateral hemibody weakness). However, without true ptosis and with normal pupils, this patient also did not have Horner syndrome.

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