Letter to Editor

Eyebrow ptosis: Barely mentioned and frequently overlooked

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A 65-year-old man with a history of diabetes mellitus, hypertension, and dyslipidemia presented to the neurology outpatient department with a 5-day history of the left post-auricular pain and difficulty in spitting with deviation of the angle of the mouth. Pain was of moderate severity, almost continuous pricking type with no obvious aggravating or relieving factors, without any vomiting. There was a history of epiphora in the left eye. There was no history of deafness, vertigo, taste disturbances, or hyperacusis. There was no history of limb weakness, ataxia, or paresthesia. There was no history of generalized headache, vomiting, or fever. His blood pressure was 144/80 mmHg and pulse rate was 68/min. Examination showed evidence of the left lower motor neuron (LMN) type facial palsy (grade III House Brackmann Grading). The palpebral fissure was smaller on the left side with eyebrow ptosis (Figs 1 and 2). He was treated with deflazacort, pantoprazole, valaciclovir, and physiotherapy, with which his weakness improved markedly.

Bell's palsy (BP) is acute facial paralysis caused by dysfunction of the peripheral part of cranial nerve VII (facial nerve) of unknown etiology. It is the most common acute unilateral peripheral facial paralysis encountered in clinical practice. LMN facial palsy can be due to different causes [1]. BP represents 46.4–69.2% of all LMN facial palsies. Other causes of LMN facial palsy are iatrogenic (most often after ear, parotid, or vestibular schwannoma surgery) in 14.4–17.4%, trauma in 8.9–10.5%, herpes zoster infection (Ramsay-Hunt syndrome) in 7.5%, and miscellaneous in 5.1% [2]. Reactivation of the latent herpes simplex virus (HSV) infection in the geniculate ganglion of the facial nerve is commonly attributed. HSV-1 and varicella zoster virus (VZV) are found in 52% and 22% of geniculate ganglia in cadavers [3]. Thrombosis of the vasa nervorum of the facial nerve resulting in ischemic facial neuropathy has also been hypothesized [4]. The interleukin (IL)-6, IL-8, and tumor necrosis factor-alpha levels were significantly higher in Bell's palsy than in controls, but did not correlate with recovery [5]. The onset of Bell's palsy during pregnancy or the puerperium is probably associated with the development of the hypertensive disorders of pregnancy.

Figure 1: (a) Left eye brow ptosis causing left eyelid pseudoptosis with slanted forehead wrinkles and absence of nasolabial fold on the left side. Left lower lid is deviated away from limbus; (b) left eye brow ptosis with deviation of the angle of the mouth to the right, causing a horizontal V sign pointing toward the paralyzed side

Figure 2: Incomplete eye closure on the left side with disappearance of eyebrow ptosis

LMN facial palsy generally results in incomplete eye closure (lagophthalmos) and upper and lower lids move away from the limbus and cause a widened palpebral fissure. Blink excursion will be reduced on the affected side, in contrast to upper motor neuron facial palsy, where blink will be normal. Sometimes, the affected upper lid will be lower than the normal one, causing pseudoptosis due to associated eyebrow ptosis.

Ramsay hunt syndrome (RHS) is a peripheral facial nerve palsy accompanied by an erythematous vesicular

Access this article online	
Received - 31 August 2025 Initial Review - 16 September 2025 Accepted - 16 October 2025	Quick Response code
DOI: ***	

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rash on the ear (zoster oticus) or in the mouth. Other frequent symptoms and signs in RHS include tinnitus, hearing loss, nausea, vomiting, vertigo, and nystagmus. Finally, some patients develop peripheral facial paralysis without ear or mouth rash, associated with either a fourfold rise in antibody to VZV or the presence of VZV DNA in auricular skin, blood mononuclear cells, middle ear fluid, or saliva. This indicates that a proportion of patients with Bell's palsy have RHS (zoster sine herpete). Treatment of these patients with acyclovir and prednisone within 7 days of onset has been shown to improve the outcome of recovery from facial palsy [6].

BP is more common and more severe in diabetics than in non-diabetics. Taste disturbances were less common in diabetics than in non-diabetics and some cases of BP may be a diabetic mononeuropathy. Symptomatic improvement has been noted in non-recovering facial palsy with facial nerve enhancement after high-dose intravenous (IV) methylprednisolone [7]. This case is presented to highlight a barely mentioned sign, eyebrow ptosis, and its presence supports the diagnosis of LMN facial palsy.

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Funding: Nil; Conflicts of interest: Nil.

How to cite this article: Anandan S, Rajendran SS, Joy J, Suresh A. Eyebrow ptosis: Barely mentioned and frequently overlooked. Indian J Case Reports. 2025; October 24 [Epub ahead of print].