

Case Report

A case of lenticulostriate artery infarction presenting with peripheral type facial palsy

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Abstract: An 82-year-old woman developed a droopy right eyelid with ipsilateral hemiparesis. Her ocular symptom was caused by weakness of the right frontalis, which is usually seen in patients with peripheral facial nerve palsy. However, head MRI showed acute cerebral infarction of the left lenticulostriate artery, and electroneurography did not detect damage to the right facial nerve. To explain the pathophysiology in this patient, asymmetrical bilateral cortex innervation to the right upper face was hypothesized. This case suggested that patients with some hemispheric strokes could develop upper facial weakness mimicking facial nerve palsy, and clinicians should pay attention to this potential pitfall in the differential diagnosis of facial nerve palsy.

Key words: cerebral infarction, peripheral type facial palsy, central type facial palsy, pseudoptosis

Introduction

The facial nerve nucleus for upper facial movements is classically thought to have bilateral cortical innervation¹⁾. This theory helps explain why upper facial movements are relatively spared by hemispheric lesions, whereas they are directly dysregulated by peripheral facial nerve palsy. Whether upper facial movements are intact could be a clue to differentiating central facial paralysis from peripheral facial nerve palsy, although several exceptional cases have been reported²⁾⁻⁴⁾.

Upper facial paralysis affects eyelid opening and closing. Weakness of the orbicularis oculi could cause incomplete closure of the eyelids leading to lagophthalmos. In contrast, weakness of the frontalis could cause drooping of the periorbital structures, including the eyebrows and upper eyelids. Therefore, some patients with weakened frontalis have difficulty opening the eyelids. This condition is called pseudoptosis, which is differentiated from 'true' ptosis, defined as drooping of the eyelids due to dysfunction of levator palpebrae superioris⁵⁾. A previous study reported that this pseudoptosis was observed in 44.1% of patients with facial nerve palsy, although it is probably very rare in central facial palsy⁶⁾.

The case of a patient with left lenticulostriate artery (LSA)

infarction presenting with right pseudoptosis due to upper facial weakness that mimicked peripheral facial nerve palsy is described.

Case report

An 82-year-old, right-handed woman suddenly developed drooping of the right upper eyelid with ipsilateral hemiparesis. She visited our hospital the following day. She had no vascular risk factors including hypertension, dyslipidemia, diabetes mellitus, or a smoking habit. She had no history of right facial nerve palsy, and no facial asymmetry had been previously noted.

In the emergency department, her consciousness was intact. Her pupils were equal (right 2 mm and left 2 mm), and bilateral light reflexes were prompt. Abnormal eye position was not observed, and external ocular movements were fully preserved. Facial sensation was intact. In the resting state, she showed drooping of the right upper eyelid covering her right pupil (Fig. 1A). Right eyebrow ptosis was also observed. On the other hand, the distance between the right eyebrow and the margo palpebrae was the same as on the other side. Wrinkling of the right forehead was decreased, though she could voluntarily lift her eyebrows, resolving the drooping of the right eyelid (Fig. 1B). Mild weakness of the right orbicularis oculi was detected, although there was no

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Fig. 1 Photographs of the patient's upper face.

(A) A photograph of the patient's upper face at a resting state on the 2nd hospital day shows the right droopy eyelid. Her right eyebrow is also drooped, and wrinkles of the right forehead are decreased. (B) A photograph of the patient wrinkling her forehead on the 2nd hospital day shows that her voluntary upper facial movement is relatively preserved. (C) A photograph of the patient's upper face in the resting state on the 20th hospital day shows partial improvement of her right droopy eyelid. Fig. 1 is published with the patient's permission.

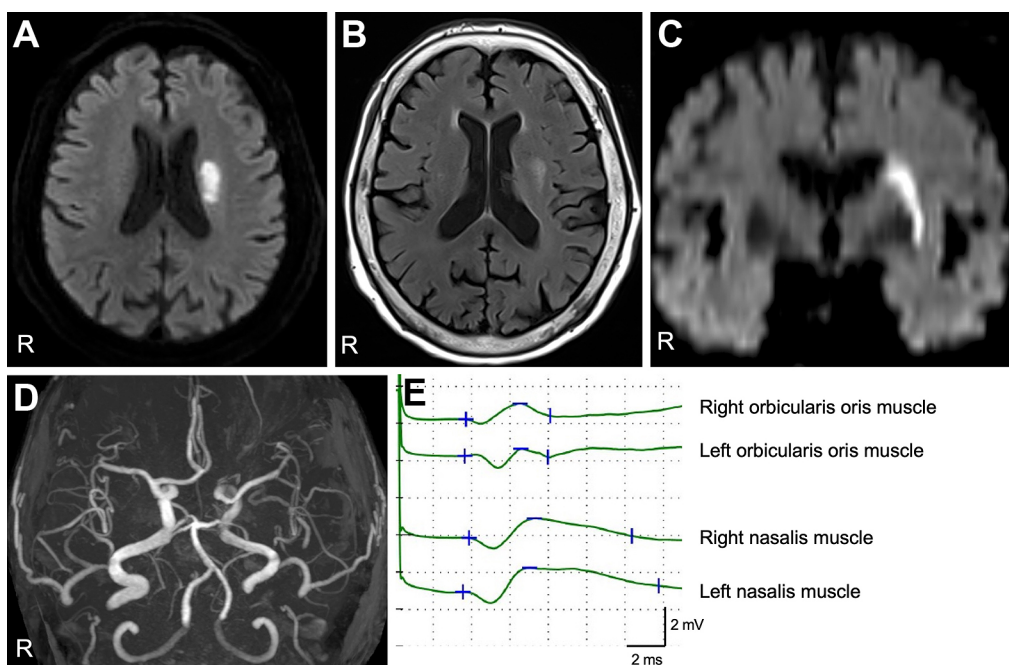


Fig. 2 Results of head MRI and electroneurography.

(A)–(C) Head MRI shows acute ischemic infarction of the left lenticulostriate artery. (A) and (B) are axial images of diffusion-weighted imaging (DWI) and fluid-attenuated inversion recovery imaging, respectively. (C) is a coronal image of DWI. (D) MR angiography shows no severe stenoses of major intracranial arteries. (E) Electroneurography shows no apparent laterality of compound muscle action potentials of the orbicularis oris and nasalis muscles. In each figure, "R" indicates the right side.

spasm at the muscle. The corner of the mouth on the right side also drooped, and mild dysarthria was present, but she had no apparent dysphagia. Gustatory and auditory sensations were intact. She showed mild right hemiparesis; pronator drift was observed on the right side, and the Mingazzini sign of the right lower extremity was present. She showed no sensory symptoms or ataxia, and she could walk alone without assistance.

Results of blood analyses were unremarkable. Hemoglobin A1c (5.5%), low-density lipoprotein cholesterol (123 mg/dl), and D-dimer ($<0.5 \mu\text{g}/\text{mL}$) were within normal limits. There

were no embolic sources detected by 12-lead and Holter electrocardiography examinations and transthoracic echocardiography. Ultrasound of the neck showed no apparent stenoses and no abnormal flows of the carotid arteries. Head MRI showed a high-intensity lesion at the left LSA territory on diffusion-weighted imaging (DWI) and fluid-attenuated inversion recovery (FLAIR) imaging (Fig. 2A–C). There were no old ischemic infarctions, hemorrhages, or brain atrophy. MR angiography showed no severe stenosis at the left middle cerebral artery (MCA) (Fig. 2D).

She was diagnosed as having a left LSA infarction and treated with intravenous argatroban 60 mg per day, intravenous edaravone 60 mg per day, aspirin 200 mg per day, clopidogrel 75 mg per day, and rosuvastatin 5 mg per day. As for her right facial palsy, overlapping peripheral facial nerve palsy was suspected. However, electroneurography on the sixth hospital day showed no laterality, suggesting that the right facial nerve was not damaged (Fig. 2E). Follow-up MRI on the 14th hospital day showed no new brainstem lesions. Her facial palsy and right hemiparesis improved gradually, and she was discharged on the 20th hospital day. The right facial palsy on discharge is shown in Fig. 1C.

Discussion

A case of left LSA infarction presenting with difficulty in right eyelid opening was described. In addition to the right-side droopy eyelid, ipsilateral eyebrow ptosis, which indicated weakness of the frontalis, was also observed. The distance between the right eyebrow and the ipsilateral margo palpebrae was not increased, implying that the right levator palpebrae superioris was intact⁷. Considering these findings, she was diagnosed as having a pseudoptosis induced by paralysis of the right frontalis. Eyelid skin usually loosens with ageing. This age-related droopy eyelid is often compensated by an unconscious contraction of frontalis. However, when paralysis of the muscle develops in these

patients, pseudoptosis could manifest.

The findings for the present patient suggest that pseudoptosis due to a weakened frontalis, which is usually seen with peripheral facial nerve palsy, could occur with a supranuclear lesion. Clinicians should pay attention to this pitfall in differentiating central nerve palsy from Bell's palsy, especially in the emergency department. How often patients with hemispheric strokes show upper facial weakness is not fully understood. A previous study reported that 6.6% of hemispheric strokes showed weak eyelid closing⁸. Furthermore, there have been several case studies describing hemispheric strokes mimicking facial nerve palsy (Table 1)²⁻⁴. Compared with them, the present case was unique in that it showed pseudoptosis as an ocular symptom and its development with a subcortical infarction; other cases showed incomplete eyelid closing, and the causative strokes mainly affected cortical areas.

The pathophysiology of the peripheral-type facial palsy in the present patient is largely unknown. One possible mechanism is the effect of a pre-existing right Bell's palsy. However, she had no apparent history of the disease, and no damage to the right facial nerve was detected on electroneurography. Therefore, we hypothesized that the bilateral cortical innervation to the right upper face was asymmetrical. The present patient might have had a congenital neuroanatomical anomaly with the right upper face dominantly controlled by the left hemisphere, and the LSA

Table 1 Cases of hemispheric strokes mimicking facial nerve palsy.

Reference	Patient (age, sex)	Lesions	Ocular symptoms	Other neurological symptoms
Hebant et al. ²⁾	78 y, female	Left precentral gyrus	Right lagophthalmos	None
Hebant et al. ³⁾	35 y, female	Hemorrhage of left precentral gyrus hemangioma	Right lagophthalmos	None
Onder et al. ⁴⁾	69 y, female	Infarction of right frontal lobe cortex and subcortex	Left lagophthalmos	Left hemiparesis
Present study	82 y, female	Infarction of left lenticulostriate artery	Right pseudoptosis	Right hemiparesis

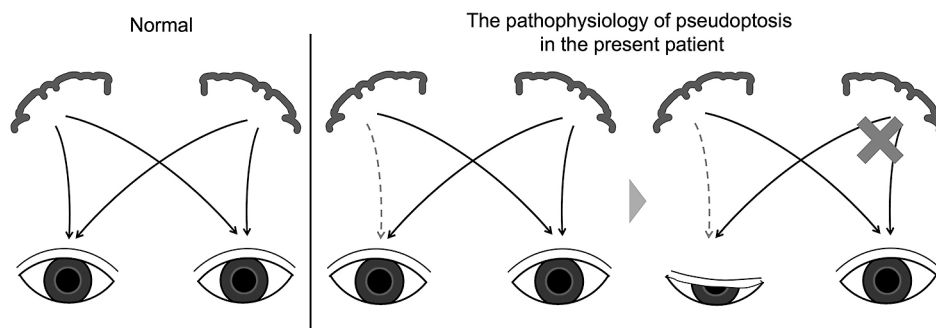


Fig. 3 A hypothesis for the pseudoptosis in the present patient.

(Left) Normal bilateral innervation of facial nerve nuclei for upper facial movements. (Middle and right) The right facial nerve nucleus in the present patient might have had congenitally dominant innervation from the left hemisphere. The dotted arrow means insufficient innervation from the right hemisphere. LSA infarction damaged the tract, resulting in upper facial weakness with pseudoptosis.

infarction might have damaged the responsible tract (Fig. 3). However, to the best of our knowledge, there have been no studies investigating the prevalence of this anomaly. Interestingly, several studies cast doubt on the classical theory of cortical innervation for upper and lower facial movements²⁾³⁾⁹⁾¹⁰⁾. In particular, Cattaneo et al. suggested that sparing of upper facial movements in MCA stroke was due to the presence of an upper face motor representation in both the MCA and anterior cerebral artery (ACA) territories¹⁰⁾. Based on this hypothesis, the LSA infarction in the present case might have damaged the tracts from both the MCA and ACA territories (Fig. S1). Nonetheless, why the present case developed upper facial weakness, whereas most LSA infarcts do not, is still unknown. Overall, the precise mechanism of facial movements is not fully understood, and further case reports and radiological and physiological studies will be needed.

In conclusion, a case of peripheral-type facial palsy with pseudoptosis due to LSA infarction was described. This case suggests that some hemispheric strokes could mimic facial nerve palsy, and clinicians should pay careful attention to them. To understand the detailed mechanism of upper facial movements, further clinical and biological studies will be needed.

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COI : The authors declare that there is no conflict of interest relevant to this article.

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