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Isolated peripheral-type facial palsy due to the central lesion:two-case reports

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Case report

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Abstract

Background Isolated peripheral facial palsy (P-FP) can lead to lesions involving the inferomedial tegmentum of the pons. However, cases with P-FP in result of a medullary lesion have rarely been reported and result from a paraventricular lesion have never been reported before. Cases presentation We described a 63-year-old man presenting with isolated P-FP due to ipsilateral pontomedullary infarction. Brain diffusion MRI revealed a hyper-intense signal on the left dorsolateral portion of the upper medulla and pontomedullary junction. And then we experienced a 77-year-old man presenting with lateral paraventricular infarction who showed contralateral peripheral type facial palsy. Brain diffusion-weighted image(DWI) showed a high-signal intensity lesion in the right lateral paraventricule and part of the posterior limb of the right internal capsule. Conclusions These two cases caution that a central nervous etiology should be considered in patients with P-FP, especially if they have high risk factors of cerebral infarction.

Background

According to the widely accepted anatomical pathways, upper facial motor dysfunction is unusually caused by unilateral hemispheric stroke thanks to bilateral corticobulbar innervation of the upper facial nerve nucleus. Isolated P-FP in the result of unilateral central nervous lesion has been reported rarely, a crucial diagnostic challenge, the differential being Bell palsy. Here we describe two hypertensive old men presenting with isolated left P-FP, one is due to left lateral medullary infarction and the other is because of right paraventricular infarction.

Case Presentation

Case 1 A 63-year-old hypertensive male presented with left peripheral-type facial palsy for two days. Neurologic examination revealed isolated left P-FP (Fig. 1). He did not have additional pontomedullary symptoms or signs, such as diplopia, abduction weakness, facial sensory loss, vertigo, nystagmus, or dysarthria. There was no limitation of ocular movement. Brain DWI showed a hyperintense signal in the dorsolateral upper medulla and pontomedullary junction area (Fig. 2).

Case 2 A 77-year-old man presented with sudden left hemiparesis before two days, and developed subsequent left P-FP one day later. He had experienced hypertension for 30 years and diabetes mellitus for 20 years. Neurologic examination showed isolated left peripheral-type facial palsy and sensory impairment of left limbs (Fig. 3). Brain DWI showed a high-signal intensity lesion in the right lateral paraventricule and part of the posterior limb of the right internal capsule (Fig. 4).

Discussion

It is not easy to meet patients with infranuclear facial palsy caused by inferolateral tegmenum of the pons infarction clinically and not to mention those caused by brainstem infarction within medulla

oblongata [1,2]. A study reported that the corticobulbar tract descends at the ventromedial lower pons, mainly at the level of the upper medulla, where the fibers then decussate and ascend into the dorsolateral medulla to synapse in the contralateral facial nucleus [3]. Therefore, the P-FP in the first patient probably developed due to the involvement of facial nerve fascicles extended from the dorsolateral upper medulla in the ascending pathway of the corticobulbar tract.

Recently, Hebant et al have described a patient caught by right peripheral-type facial palsy with precentral gyrus infarction [4]. This case broke the theory that all motor neuron superior to the facial nucleus damage can cause central-type facial palsy. Weakness of upper facial muscles can be seen in central facial paralysis, while it is often involved bilaterally [5]. The corticobulbar tract originates from the cerebral motor cortex, passes through the genu area of the internal capsule and then distribute to both sides of the upper facial nerve nucleus. Therefore, contrallateral infarction cannot result in the forehead dysfunction due to the ipsilateral corticobulbar tract fibers remained. But we believe the innervation is still attenuated slightly than before. In the second case, the P-FP was considered as the consequence of the decreased innervated effect of the corticobulbar tract fibers. Notably, the P-FP caused by central infarction is lighter than the Bell palsy, or even temporary, because there is still a small amount of fiber on the ipsilateral side remained functioning.

Conclusions

As we described above, both pontomedullary and paraventricular infarction can lead to isolated peripheral facial palsy. It has been reported rarely, and may be misdiagnosed as Bell palsy instead. These two cases suggest that clinicians should take into account the possibility of a central nervous lesion in patients with peripheral-type FP, even when they have high risk factors of cerebral infarction, including an advanced age, hypertension, diabetes, or cardiac disease history.

Declarations

Ethics approval and consent to participate

Not applicable

Consent for publication

Written informed consents were obtained from the patients themselves. A copy of the written consent is available for review by the editor if you want. We clarify the consent covered the publication of the information included in the case reports, as well as any associated images.

Availability of data and matarials

Not applicable. For protecting patient privacy.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

JJ: concept and design of study, interpretation of data, drafting and revising the manuscript.

XS: critical revision of the manuscript for important intellectual content, study supervision.

all authors have read and approved the manuscript.

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Abbreviations

P-FP: peripheral facial palsy

DWI: diffusion-weighted image

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Figures

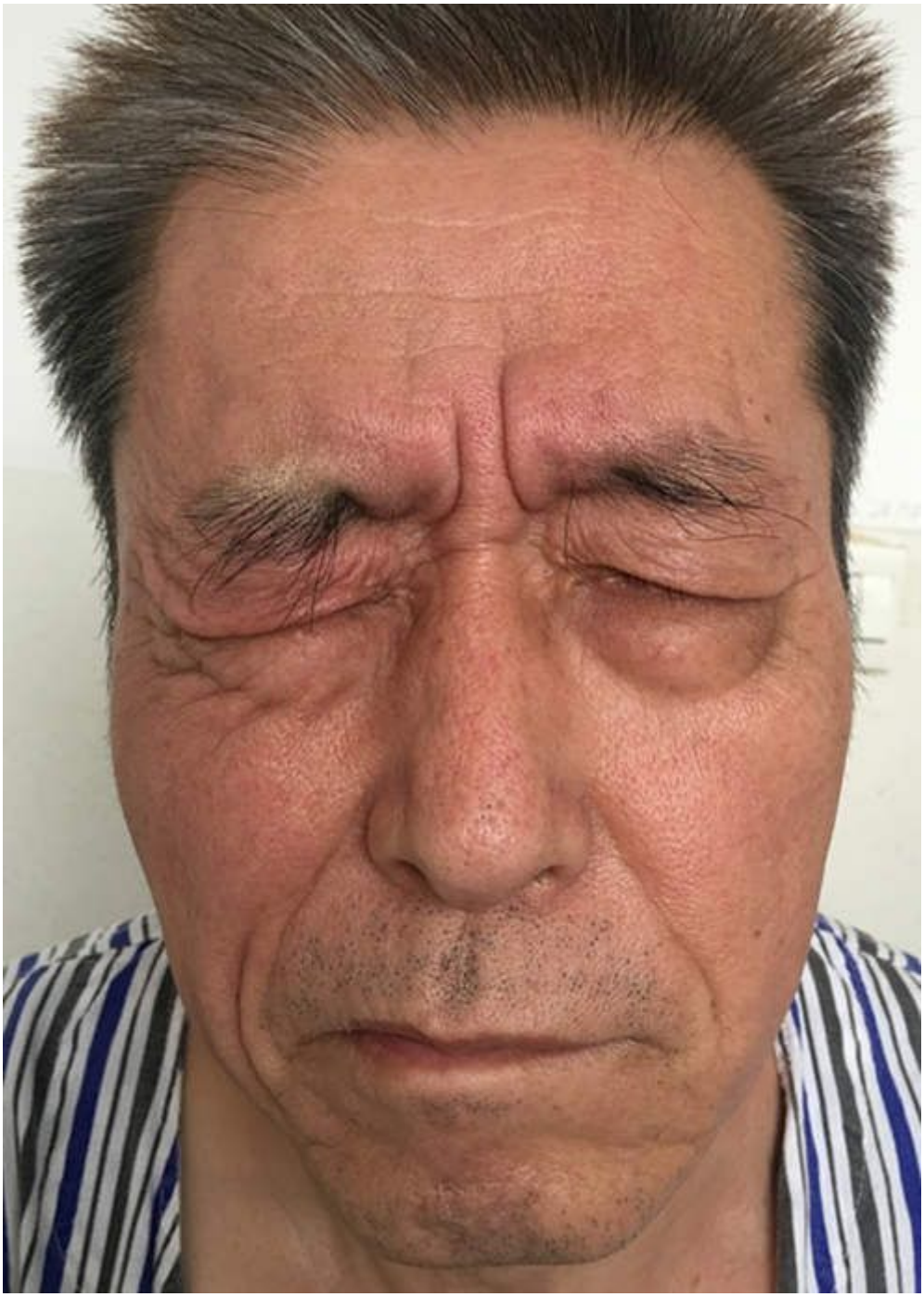

Figure 1

Photograph of the first patient demonstrates a left peripheral-type facial palsy Figure legends: Decreased movement of the left-sided forehead, eyebrow of patient in the first case with maximal effort was observed, and the patient showed drooping of the corner of the mouth in the left face.

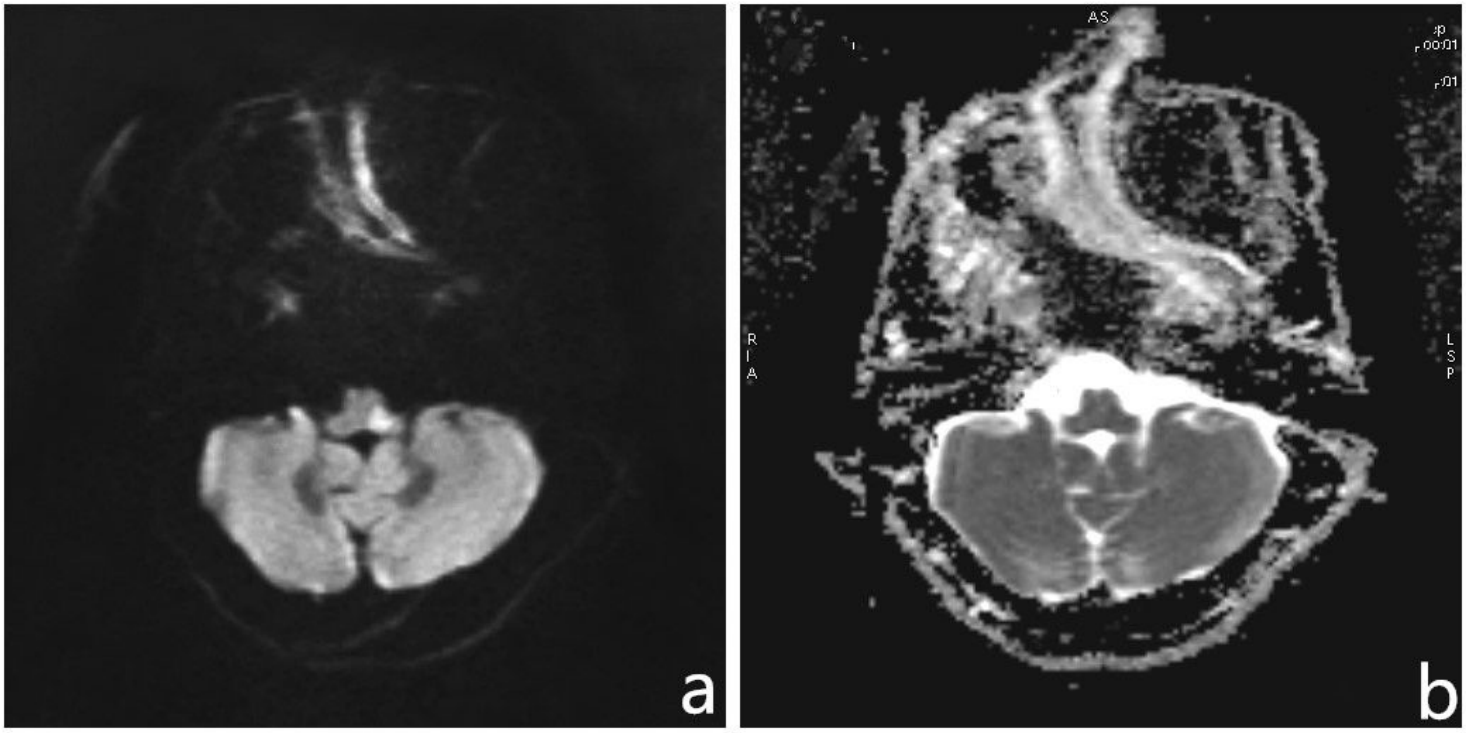

Figure 2

Brain MRI of the first patient Figure legends: (a) Axial diffusion-weighted image shows a focus of restricted diffusion in the left dorsolateral upper medulla and pontomedullary junction area;(b) Axial apparent diffusion coefficient shows a corresponding hypointensity in the left dorsolateral upper medulla confirming an acute infarction.

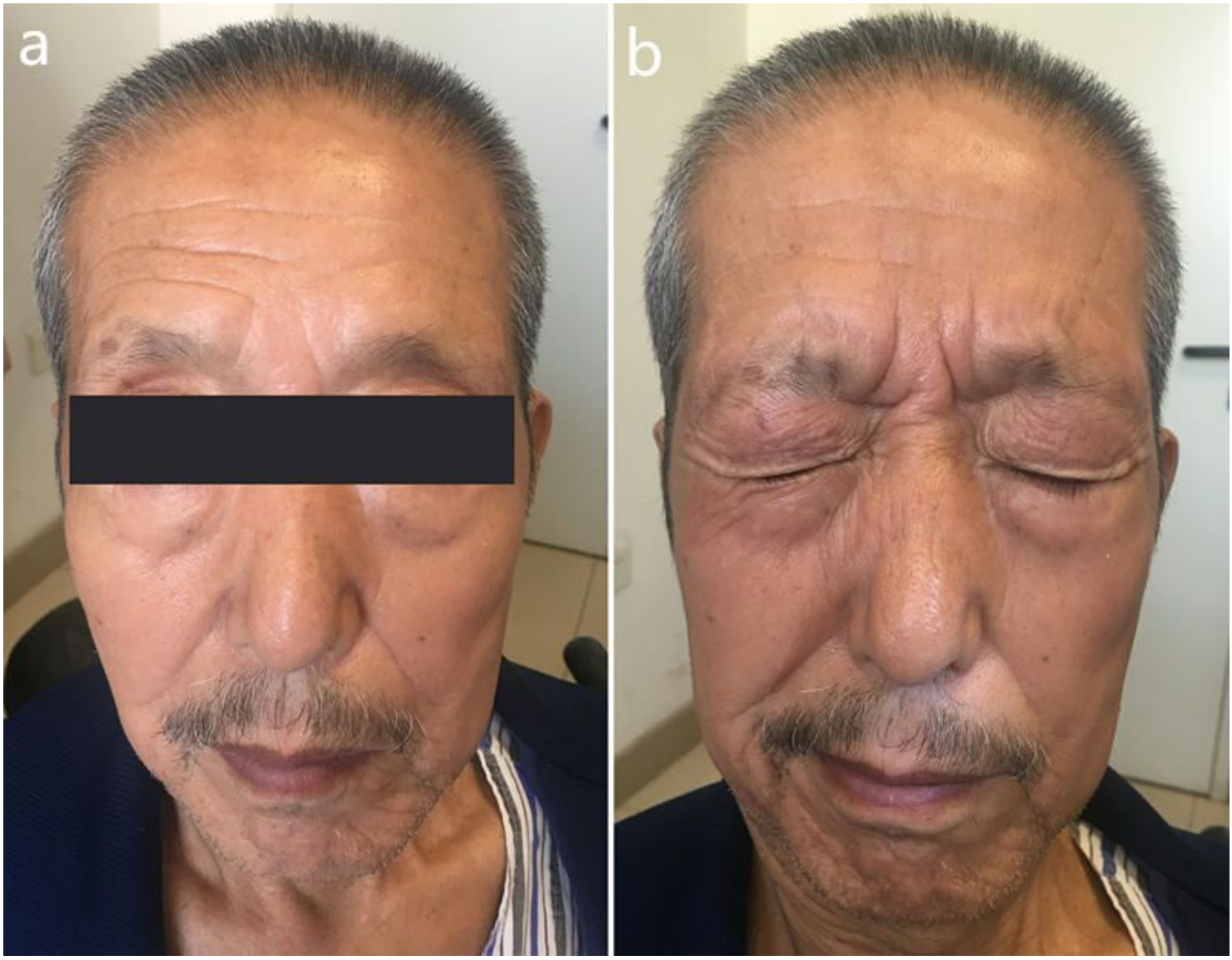

Figure 3

Photograph of the second patient demonstrates a left peripheral facial palsy Figure legends: (a) The patient showed drooping of the eyebrow and corner of the mouth in the left face at rest; (b) Decreased movement of both the upper and lower part of the face with asymmetry of eyelid closure of the second patient during maximal effort was observed.

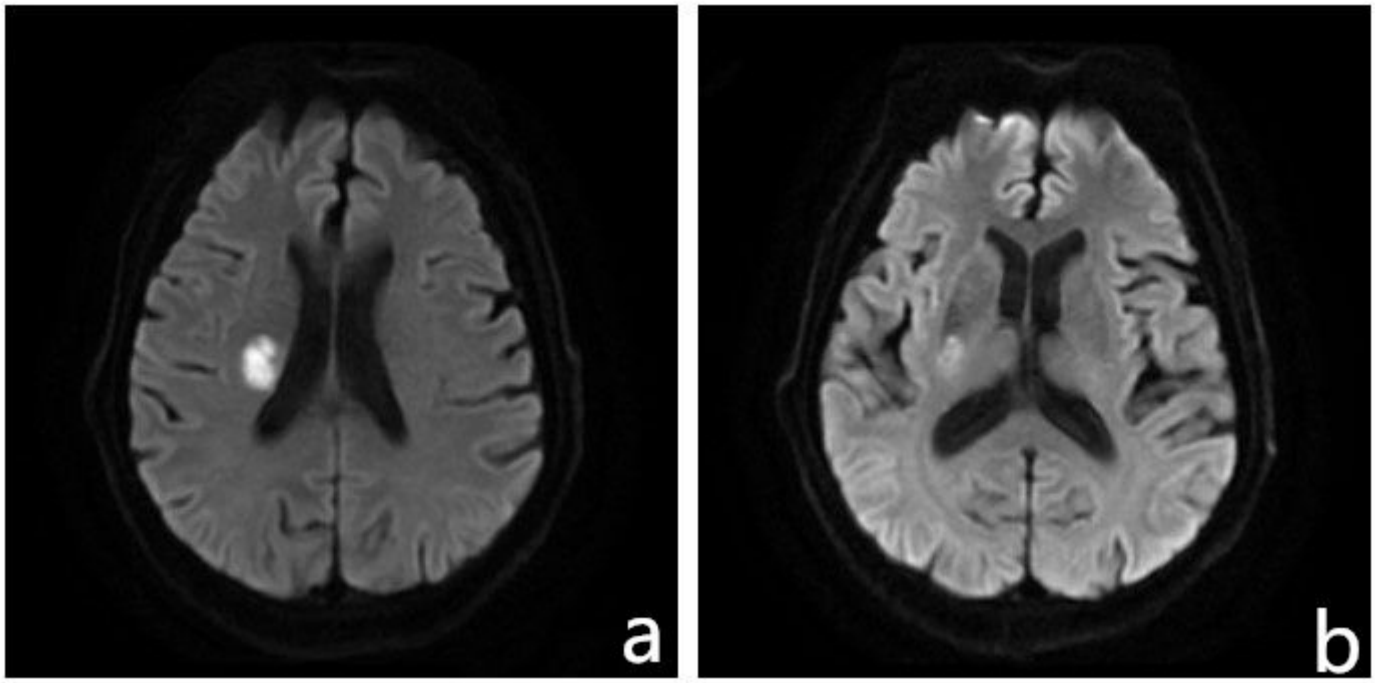

Figure 4

Brain MRI of the second patient Figure legends: (a) Axial diffusion-weighted image shows a focus of restricted diffusion near to the right lateral ventricle. (b) Axial diffusion-weighted image shows a hyperintensity focus in the posterior limb of the right internal capsule.

Supplementary Files

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