"Uncrossed Central Facial Paralysis" Caused by Pontine Infarction

A Case Report

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Introduction: We report a patient with extraordinary pontine infarction-induced contralateral central facial palsy and weakened limb strength.

Case Report: This is a 66-year-old man with left arm movement difficulty for 10 days and worsening over the last 1 day. His left nasolabial fold flattening and left arm strength and sensory were decreased. He could not complete the finger-nose test well with his right hand. Magnetic resonance and magnetic resonance angiography tests confirmed his right pontine acute infarction but without large vessel stenosis or occlusion.

Conclusion: "Uncrossed paralysis" patients may present with contralateral face and body weakness with pontine infarcts, if the infarct occurs above the level of the facial nucleus head, and may be simmilar with the higher level pontine lesions or cerebrum semisphere infarction, which need particular attention during clinical practice.

Key Words: uncrossed facial paralysis, pontine infarction, stroke, facial nucleus

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Cerebral infarction is the most common type of ischemia cerebral vascular disease, which causes millions of people to lose labor ability and brings heavy burden to their families and society.[1,2] Among all types of cerebral infarction, pontine infarction is unique and its manifestation can be various. In general, pontine infarction can cause "crossed hemiparesis," which manifests as ipsilateral facial and contralateral limb paralysis. Here we report an extraordinary pontine infarction patient manifested as contralateral central facial palsy and weakened limb strength.

CASE REPORT

This is a 66-year-old man with left arm movement difficulty for 10 days and worsening over the last 1 day, hypertension for 20 years, and diabetes for 22 years. At the age of 44, he was diagnosed with diabetes and took acarbose, metformin, and insulin degludec, however, his blood glucose level still fluctuated between 8 mmol/L and 13 mmol/L. Two years later, his highest blood pressure could reach 190/90 mm Hg. He took amlodipine regularly but not controlled well. Ten days ago, the patient left arm occurred limited movement without barylalia, numbness, and transient unconscious. He did not take medications or go to the hospital. One day ago, he felt the left arm movement difficulty aggravated and went to our clinic. Physical examination: temperature: 36.3 °C, pulse: 74 bpm, respiration rate: 18 bpm, blood pressure: 114/67 mm Hg. Conscious with fluent speech, national institute of health stroke scale (NIHSS) score: 4 score: 15 (E4V5M6). visual field, acuity, and eye movement were normal. No signs of eye nystagmus. Facial sensory was normal, and bilateral masseter muscles are symmetrical. Bilateral frontal lines were also symmetrical. His left nasolabial fold is flattening. No sign of dysphagia, choking, and coughing when drinking water. Neck turning and shoulder shrugging are symmetrical and powerful. When he sticks out his tongue, the tip of his tongue was slightly towards the left. His left arm strength and sensory were decreased while his right side body was normal. He could not complete the finger-nose test well with his right hand. No Romberg or Babinski signs were detected. Radiology: brain-computer tomography showed a low-density lesion on the right pontine with no sign of intracranial hemorrhage. And magnetic resonance (MR) confirmed the acute infarction in the right ventrolateral pontine from the facial nerve nucleus head to the midbrain trochlear nerve level pontine. However, there was no large vessel stenosis or occlusion detected on the MR angiography (Fig. 1).

DISCUSSION

Due to the complicated structure and nucleus distribution of the pontine, the symptoms and signs can be diverse and nontypical. The ventrolateral pontine infarction mostly involved the abducens nerve, facial nerve, pyramidal tract, spinothalamic tract, and medial lemniscus. This can cause Millard-Gubler syndrome, which manifests as ocular abduction disorder, peripheral facial paralysis, contralateral central hemiplegia, and contralateral hemiparesis on the lesion side. This syndrome is often induced by anterior inferior cerebrum artery occlusion. And the ventromedial pontine infarction involved the abducens nerve, facial nerve, pontine lateral visual center, medial longitudinal tract, and pyramidal tract and manifests as ipsilateral ocular abduction disorder, peripheral facial nerve paralysis, bilateral gaze to the opposite side, and contralateral central hemiplegia, which is often called Foville syndrome. The Foville syndrome can be seen when para pontine median artery occlusion occurred. The last common syndrome of pontine infarction is Raymond syndrome. The Raymond syndrome is often caused by the vestibular nucleus, abductor nucleus, facial nucleus, medial longitudinal tract, middle cerebellar foot, inferior cerebellar foot, spinothalamic tract, and medial lemniscus lesion and usually occurs nystagmus, abduction disorder on the affected side, peripheral facial paralysis, inability to gaze on the affected side of both eyes, crossed shallow sensory disorder and deep sensory disorder on the opposite side, Horner sign on the affected side and ataxia on the affected side. Raymond syndrome is mostly seen in the anterior inferior cerebrum artery or superior cerebrum artery occlusion.[3]

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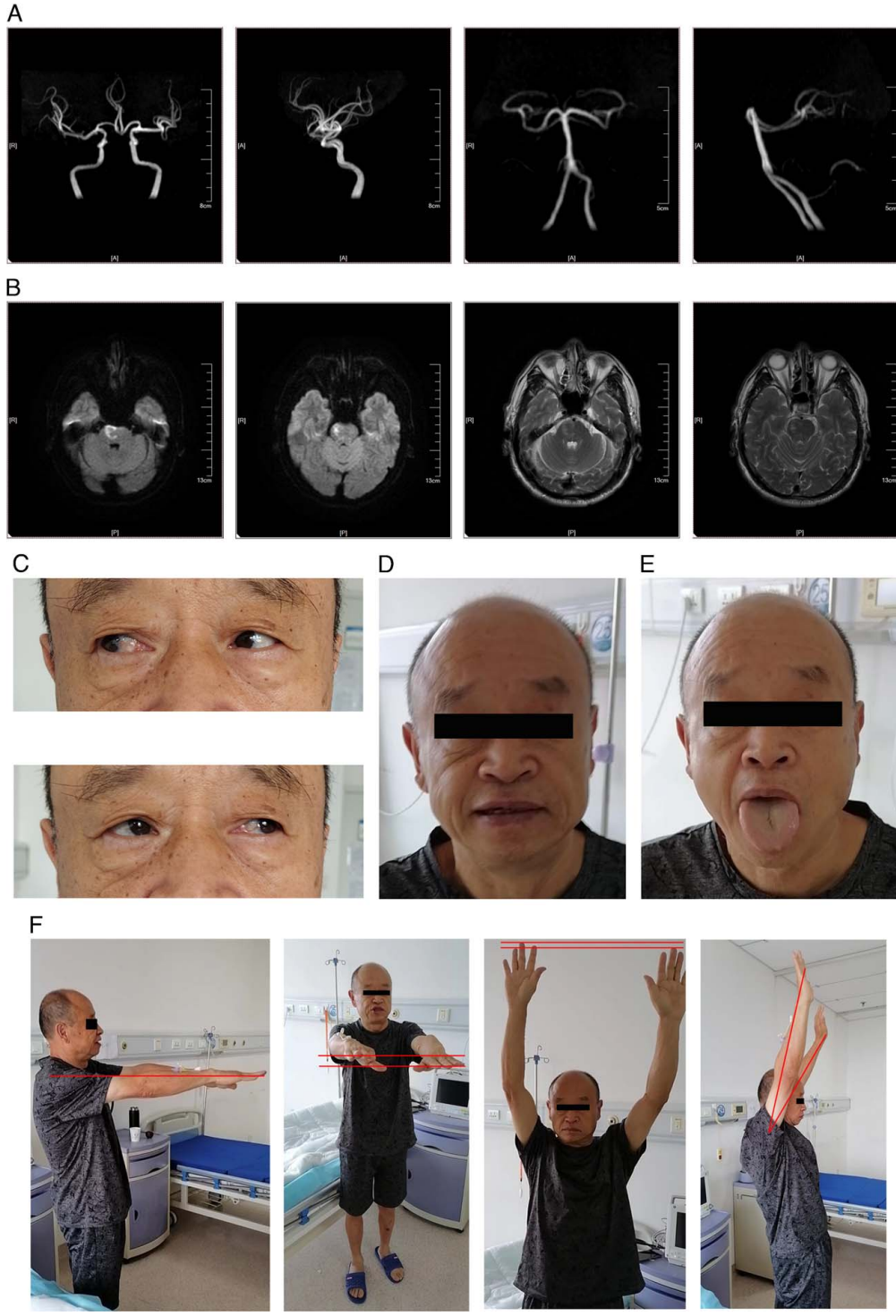

FIGURE 1. The images and clinical manifestation of "uncrossed paralysis". (A) magnetic resonance angiography images; (B) diffusion-weighted image and T2 weighed images; (C) eye movement showed normal without dyskinesia and tremor; (D) left nasolabial sulcus was shallower than the right side; (E) the tongue tip was slightly towards left; (F) Barre test and muscle strength tests. Red lines indicated horizontal or vertical positions.

All common pontine lesion syndromes include "crossed paralysis," which manifests as facial paralysis on the lesion side and contralateral limb paralysis. The basis of these special signs and symptoms is decided by the course of the corticospinal tract, cortical nucleus tract, and facial nerve pathway. And whether facial paralysis "crosses" to the contralateral may probably associate with the infarction relative position to the facial nucleus.

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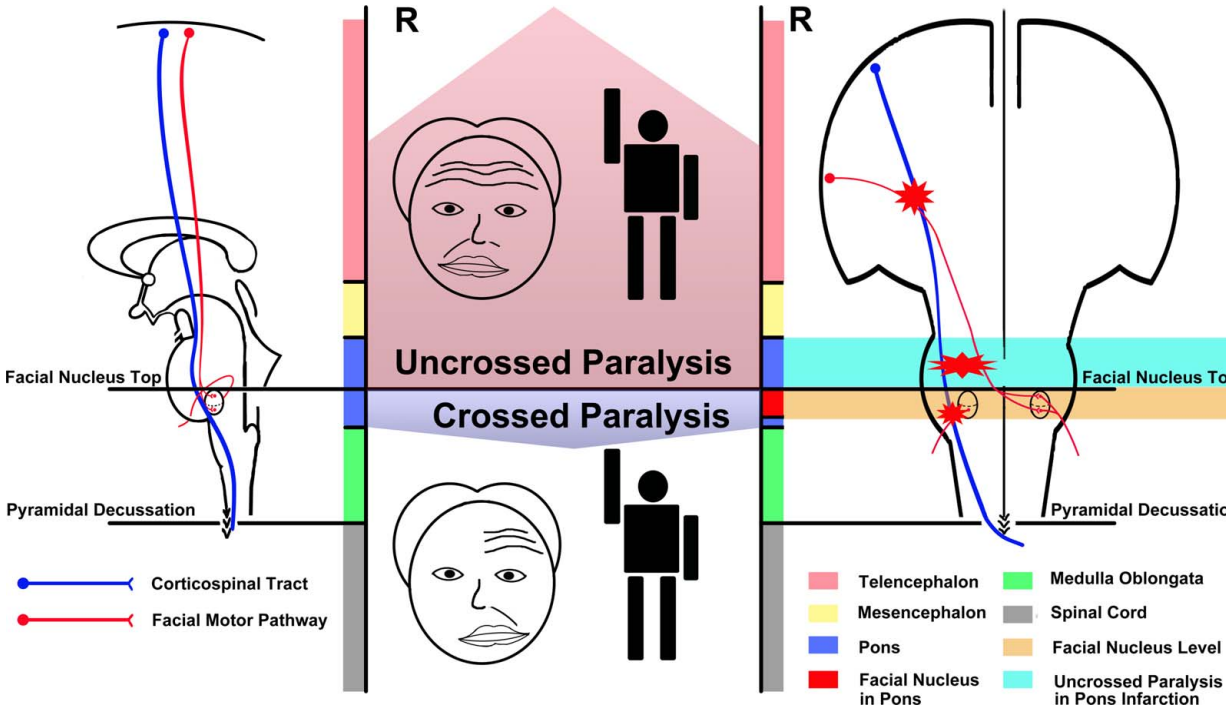

FIGURE 2. The mechanism of "uncrossed paralysis". When the damage level was just between the cortex and facial nucleus top level, it often manifests as "uncrossed paralysis". And if the damage level was between facial nucleus level and pyramidal decussation, it always showed classic "crossed paralysis" signs and symptoms.

As is well known, the upper half of the facial nerve nucleus takes facial muscle movement above the ocular fissure in charge, whereas the lower part controls facial movement below the subjective ocular fissure. However, the upper half of the facial nucleus is innervated by bilateral cortical nuclear bundles, whereas the lower half is only innervated by contralateral cortical nuclear bundles instead. According to the lesion focus relative position to the facial nucleus, facial paralysis can be divided into supranuclear and subnuclear paralysis. When the infarction focus lies above the facial nucleus such as the corticospinal tract or cortical nuclear tract in corona radiata, it can be performed as contralateral central facial palsy. And if the infarction lies on or beneath the facial nucleus level, ipsilateral peripheral facial paralysis may be the main manifestation.[4,5]

The corticospinal tract crosses to the contralateral at medulla oblongata pyramids decussation, whereas the cortical nuclear tract innervating lower face muscle crosses to the contralateral at pontine. Therefore, when the pontine infarction level does not reach the facial nucleus, infarction can manifest as "uncrossed" paralysis just like in mesencephalon or cortex infarction.

Till now although "uncrossed" central facial paralysis can be seen in clinical practice, seldom literature reported or analyzed the potential mechanism of this phenomenon induced by upper pontine infarction. Ogawa[6] and his colleagues reported a patient with Raymond syndrome and uncrossed central facial and limb palsy caused by bottom pontine infarction in 2008. They owe the symptoms to the ischemia of perforating branch of the basilar artery.[6]

In this case, the patient manifests as contralateral central facial palsy and arm slight sensor-motor abnormalities. From the diffusion-weighted image, we can see the "*C*-shaped" infarction pontine did not involve the medial longitudinal fasciculus and abducens nucleus. It is consistent with the clinical manifestation of no eye movement disorder. The "uncrossed facial palsy" is mostly associated with the focus lying above the facial nucleus level (Fig. 2).

In general, pontine infarction often causes "crossed paralysis". However, the rare situation of both facial and limb palsy lying on the same contralateral side cannot be ignored. "Uncrossed paralysis" may be caused by pontine infarction above the top of the facial nerve nucleus level, which needs particular attention during clinical practice. Though MR angiography fails to detect crime arteries, it cannot be excluded whether the infarction is caused by small perforating branches or blood flow dynamics factor. Digital subtraction angiography may be the best choice and long-term follow-up is also needed.

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