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Medullary infarction with central facial paralysis as the only symptom: a case report

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Abstract

The anatomical structure of the medulla oblongata is complex, its nerve fibers are dense, and its blood vessels are complex. Clinical manifestations of ischemic damage to the medulla oblongata are therefore relatively diverse, and include vertigo, dysphagia, and dysarthria. Although facial paralysis may also occur, medullary infarction with facial paralysis as the first and only symptom is rare. Herein, we report a case of medullary infarction with ipsilateral central facial paralysis as the only symptom.

Keywords

Medullary infarction, facial paralysis, tractus corticobulbaris, medulla oblongata, ipsilateral, facial nucleus

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Introduction

Both the anatomical structure and the blood vessels of the medulla oblongata are complex, and clinical manifestations of ischemic damage to this region are diverse. There are many dense nerve nuclei and nerve fibers in the medulla oblongata, such as the hypoglossal nerve, vestibular nucleus, spinothalamic tract, medial lemniscus, vestibular nucleus, vestibular fibers, trigeminal spinal tract nucleus, and spinocerebellar tract.[1] Clinical manifestations of

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ischemic damage to the medulla oblongata include vertigo, dysphagia, dysarthria, sensory disturbances, and cerebellar ataxia.[2] Notably, although there have been many reports of medullary infarction combined with facial paralysis,[3,4] cases with facial paralysis as the only symptom are uncommon. In the present report, we describe the imaging and clinical features of a patient with ipsilateral central facial paralysis caused by medullary infarction.

Case presentation

A 68-year-old man was admitted to the hospital because of a deviated mouth for 1 day. He had a history of hypertension. There was no obvious cause of mouth deviation and he had no signs of dysphagia, hoarseness, choking on drinking, vertigo, nausea, vomiting, numbness, or weakness of the right limb 1 day before. The outpatient diagnosis was facial neuritis. The patient had a 4-year history of hypertension, was taking oral nifedipine controlled-release tablets, and had a blood pressure of 164/94 mmHg at the onset of the disease. He had no other underlying diseases, such as diabetes mellitus or coronary artery disease. A neurological examination revealed clear consciousness, symmetrical bilateral frontal lines, strong binocular closure, shallow left nasolabial fold, low left angle of the mouth, tongue extension in the center, and normal pain and temperature sensations in the bilateral sides and limbs. Other tests, including speech, motor, sensory, and coordination evaluations, were normal (Figure 1). Two days after admission, the patient underwent a re-examination of head magnetic resonance imaging (Figure 2); aside from the medulla oblongata lesion, no other lesions were observed. The patient was therefore considered to have ipsilateral central facial paralysis as a result of lateral medulla oblongata infarction.

Discussion

Although medullary infarction combined with facial paralysis is relatively common,[3,4] cases with facial paralysis as the only

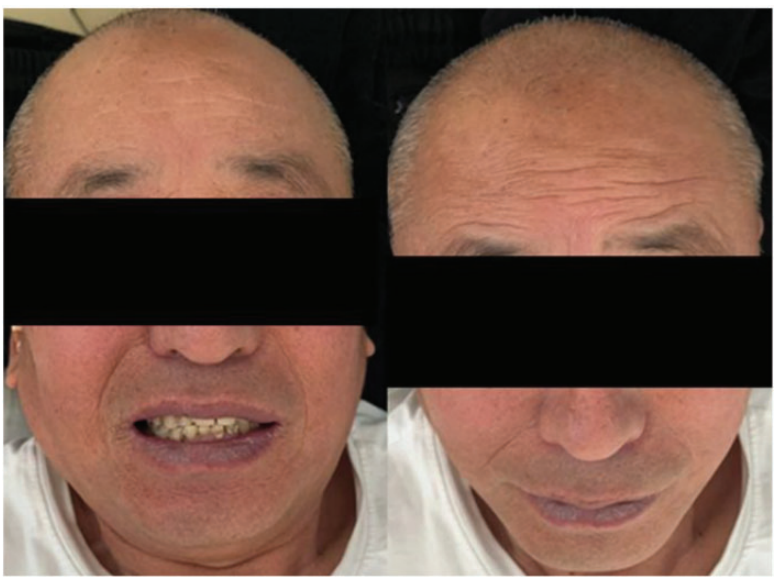

Figure 1. The patient had left-central facial paralysis.

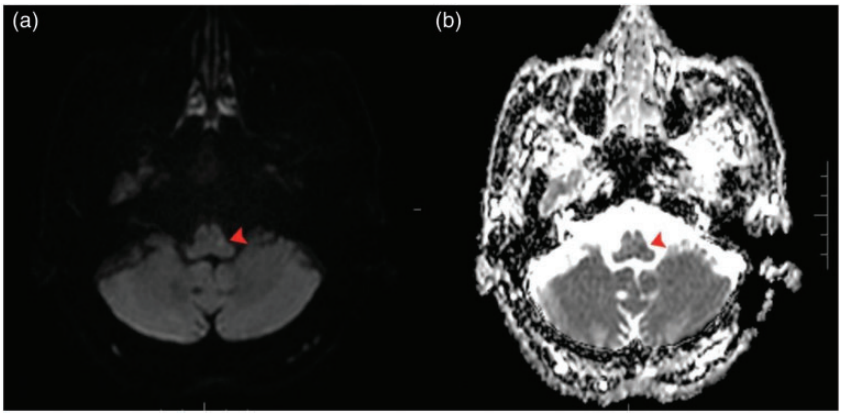

Figure 2. Brain magnetic resonance imaging showing high signal intensity on diffusion-weighted imaging and low signal intensity on apparent diffusion coefficient imaging, suggesting an acute or subacute cerebral infarction in the medulla oblongata.

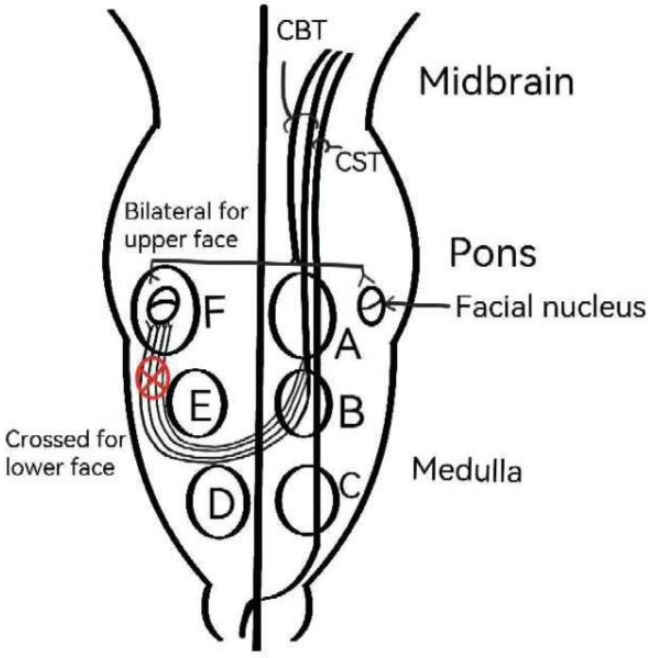

Figure 3. Diagram showing aberrant corticobulbar nerve fibers of the facial nerve.

symptom are rare; the pons is usually involved when facial paralysis is the main symptom.[5,6] In the present patient, however, imaging of the medullary infarction was consistent with Avellis syndrome,[7] and the medullary lesion was very small. The patient did not have bulbar palsy, chemosensory disturbance, or Horner's syndrome; we therefore considered that the corresponding nuclei were not damaged.

But what about central facial paralysis? Do the cortical bundles sink into the medulla, searching for a needle in a haystack, and then angle upward to the pontine nucleus? Figure 3 shows the anatomical basis of central facial paralysis. Cortical nuclear bundles first innervate the upper half of the facial nucleus. At the level of the nucleus, they split into two to innervate both lateral nuclei. The fibers that innervate the lower half first travel to the medulla oblongata and then cross to the other side, innervating only the lower half of the contralateral nucleus. Thus, the lesion in the medulla oblongata in the present case may have damaged the fibers that have already crossed over (Figure 3), meaning that it manifested as ipsilateral central facial paralysis. It seems that for a medulla oblongata lesion to cause central facial paralysis, the lesion location must be relatively high. If not, symptoms of facial paralysis will not appear, similar to the lesions on positions C and D in Figure 3.

Conclusions

Given the complex internal structure of the medulla oblongata and its relatively large number of nuclei, different manifestations are likely to occur as the result of slight differences in lesion size and/or location. In addition to its typical manifestations, lateral medulla oblongata infarction often has atypical manifestations, including the relatively uncommon central facial paralysis that was observed in the current case. Different parts of the cortico-brainstem tract, which brings nerves to the facial nucleus, have different functions. For example, damage to the fibers that have crossed after sinking into the medulla may cause paralysis of the central part of the face on the same side as the lesion. Our case report indicates that facial paralysis caused by medullary infarction is not necessarily central, and can be either ipsilateral or contralateral.

Author contributions

All the authors contributed to the care of this patient. ZZ provided the case material, WT wrote the first draft, RT revised the manuscript, YZ drew the illustrations, and all authors read and approved the final article submission.

Declaration of conflicting interest

The authors declare that there is no conflict of interest.

Ethics statement

Written informed consent was obtained from the patient to publish this case report and any accompanying images.

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