Case Report

Pontine Supranuclear Facial Palsy

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Two patients presented with a unilateral supranuclear facial palsy. Additional dysarthria was attributed to the pontine origin documented by magnetic resonance imaging on the contralateral side. The pontine disorder also was indicated by an isolated delay of the blink reflex R1 component or of the masseter reflex. We attribute the facial palsy to a lesion of a supranuclear fiber bundle supplying the facial nucleus. The location of the lesions favors these fibers taking a separate course from the main pyramidal tract at the mid- to upper pontine level. (Stroke 1990;21:1754-1757)

Peripheral facial palsy originating from nuclear or fascicular involvement occasionally is observed in pontine ischemia and mainly presents as a supplementary sign to contralateral hemiparesis in the Millard-Gubler and related syndromes. In elderly diabetic patients, however, it may occur as the only clinical sign (unpublished observations). "Isolated" supranuclear facial palsy has been described as a lacunar syndrome of the supratentorial pyramidal tract,1 with only one report describing it as due to a pontine lesion.2 We report two patients with supranuclear facial palsy in whom the mid-pontine location of the lesion differed from present knowledge about the course of supranuclear fibers supplying the facial nucleus.3

Case Reports

Case 1

A 63-year-old man with a history of occasional right hemicrania and labile arterial hypertension suddenly developed mild nausea, dizziness, weakness of the left face, and difficulty speaking. On admission, he showed moderate left supranuclear facial palsy with sparing of the frontalis muscle. The angle of the mouth was hanging, whistling was markedly impaired, and the left palpebral fissure was 1 mm wider than that of the right. Speech was slightly dysarthric, but there were no other neurological abnormalities. Arterial pressure was 170/100 mm Hg. Electro-oculography showed saccadic horizontal pursuit movements and decreased optokinetic reaction to the left with a normal caloric response. The R1 component of the electrically evoked blink reflex was delayed on the right (13 msec as compared to 11.3 on the left), but the R2 components were not. Masseter reflex latencies were normal. Cranial computed tomography on day 1, including 3-mm sections of the brain stem, was normal. Magnetic resonance imaging (MRI) on the same day documented a mid- to upper pontine lesion contralateral to the supranuclear facial palsy and measuring 16x11x13 mm (Figure 1). Symptoms and signs resolved within 4 days. Six months later, neurological examination and blink reflexes were normal. The size of the right pontine lesion had decreased to 13x8x7 mm, which was interpreted as the residuum of a vascular lesion.

Case 2

A 70-year-old man with a history of absolute cardiac arrhythmia and moderate mitral valve insufficiency suddenly developed unsteady gait, left facial weakness, and dysarthria. On admission, there was left supranuclear facial palsy, sparing the frontalis muscle, moderate dysarthria, and left hemiataxia. Electro-oculography showed saccadic horizontal pursuit movements, normal optokinetic reaction, and normal caloric response. The masseter reflex latency was increased on the right by 0.8 msec as compared to the left. The blink reflex was normal. Computed tomography showed a right pontine faint hypodense area. A lesion of 11x12x11 mm at the mid- to upper pontine level contralateral to the supranuclear facial palsy was documented by MRI (Figure 2). At reexamination 4 months later, there were no further clinical signs.

Discussion

The left facial paresis of our patients showed a supranuclear pattern of muscle involvement. "Isolated" supranuclear facial palsy has been observed with lacunar lesions of the contralateral internal capsule and corona radiata.1 Dysarthria or disturbances of tongue movements were frequent additional signs. However, minor additional signs and symptoms may be associated with lacunar syndromes.4 In our patients, the dysarthric speech clearly was different from the speaking disturbances observed in severe

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Fig. 1. Magnetic resonance imaging of case 1, initial examination. Axial section (top panel) showing area of decreased signal intensity within the mid- to upper pontine tegmentum. Its paramedian location is shown on the coronal (middle panel) and horizontal (bottom panel) sections. Magnetom 1.0 T. Top and middle panels: TR 400, TE 17; bottom panel: TR 3,000, TE 90. Calibration, 50 mm.

Bell’s palsy. Thus, dysarthria may be of diagnostic relevance for recognizing an upper motor neuron origin of facial paresis in pontine lesions.

The lesions documented by MRI at the mid- to upper pontine level on the contralateral side could have impaired upper motor fibers only. The functional relevance of pontine lesions in this location has been indicated previously by the isolated blink reflex R1 and masseter reflex abnormalities. These abnormalities have been attributed to impairment of intra-axial trigeminal fibers passing near the principal fifth nerve nucleus on their way either to the facial nucleus or the fifth nerve mesencephalic and motor nuclei.

Only one patient with supranuclear facial palsy due to a pontine lesion has been reported so far. In this case, a paramedian lower pontine lesion contralateral to the supranuclear facial paresis was demonstrated by computed tomography. The authors postulated that the pontomedullary bundle to the facial neurons (the “fourth reich” of Dejerine) leaving the main pyramidal tract near the sulcus between the pons and the medulla was involved. In our patients, the lesions must have involved supranuclear fibers going to the facial nucleus only because pyramidal fibers supplying the limb muscles were spared. The location was contralateral, paramedian, and mostly tegmental, but extended to the basis pontis. This area comprises the central tegmental tract and medial parts of the medial lemniscus. In humans, degenerating corticobulbar fibers after medial cerebral artery occlusion or internal capsule hemorrhage are found within the ipsilateral paramedian pontine tegmentum. In the cranio caudal extension, these fibers parallel the pes lemnisci, cross the midline, and approach the facial nucleus. Because this area was involved in our patients, a bundle consisting of at least part of the supranuclear fibers to the facial nucleus is likely to descend dorso medially within the pontine tegmentum, separate from the main pyramidal tract, and remain uncrossed at the mid-pontine level. This area of the pons also contains fibers from the cerebellum, whose impairment might have caused dysarthria and saccadic pursuit movements of the eyes, as well as the hemiataxia in case 2. Dysarthria and hemiataxia as described in the dysarthria–clumsy hand syndrome have been observed with lesions at a slightly lower level and more basal location.

The extension of the lesion demonstrated by MRI in our patients was larger than expected from the clinical findings. However, large midbrain lesions causing only minor divisional third cranial nerve disorder have been reported. A size of 10–15 mm still is compatible with a lesion because of circulatory arrest of one of the long anteromedial pontine arteries of the basilar artery. The reduced size of the lesion at reexamination indicates that the initial changes on MRI partially represent some reversible effects, such as edema. Arterial hypertension, although moderate, favors a lacunar lesion in case 1. Microembolization is the likely cause in case 2, with atrial fibrillation and mitral valve insufficiency.
FIGURE 2. Magnetic resonance imaging of case 2, initial examination. Axial section (upper panel) showing area of decreased signal intensity at the mid- to upper pontine level. Its paramedian location is shown on the coronal section (lower panel). Magnetom 1.0 T. Upper panel: TR 400, TE 17; lower panel: TR 3,000, TE 90. Calibration, 50 mm.

References


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