## Emotional facial paresis in a patient with a lateral medullary infarction

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Emotional facial paresis (EFP) is characterized by a weakness of inferior facial muscles evident during emotionally evoked movements but not during voluntary contraction. EFP is described in patients with lesions of the supplementary motor area, basal ganglia, temporal lobes, frontal white matter, anterolateral thalamus, anterior limb of the internal capsula, and brainstem tegmentum.<sup>1,2</sup> Voluntary facial paresis (VFP) is caused by lesions of the motor cortex or descending pyramidal tract at the level of the corona radiata, posterior limb of the internal capsula, ventral mesencephalon, and ventral pons.<sup>1</sup> We describe a patient with EFP due to a small lesion in the upper medulla oblongata.

Case report. A 60-year-old man presented with sudden onset of vertigo, dizziness, dysphagia, hoarseness, and facial paresis. He had hypercholesterolemia and a family history of stroke. Admission neurologic examination found mild gait ataxia, left Horner's syndrome, hiccups, and hypesthesia for tactile and thermic sensation involving arm and trunk on the right side. A left inferior facial paresis was evident during normal conversation and emotional movements (figure 1A), but not during voluntary activation (see figure 1B). Two cranial CT scans obtained at 5 and 48 hours after onset were unremarkable. Three days later, a brain MRI revealed a small hyperintense area on T2-weighted scans in the left upper medulla (see figure 1, C and D). Extracranial duplex ultrasonography found slight carotid atheromatosis; transthoracic and transesophageal echocardiograms were normal. On MR angiograms a basilar artery hypoplasia without a major atheromatosis was present. The patient's condition improved, but 2 weeks later the EFP, a mild hoarseness, and a contralateral hypesthesia were still present.

*Discussion.* Our patient presented an EFP associated with typical symptoms of the upper lateral medullary syndrome: ipsilateral Horner's syndrome, contralateral pain/thermic sensory impairment, hoarseness, dysphagia, gait ataxia, and hiccup.<sup>3</sup>

Facial paresis (FP) has been reported in the 23 to 56% of patients with dorsolateral medullary infarctions, mainly ipsilaterally to the lesion,<sup>36</sup> and has been attributed to the involvement of an aberrant corticobulbar tract.<sup>5,6</sup> In medullary infarctions, FP is usually noted when the lesion is located in the upper medulla, suggesting that the looping corticobulbar fibers do not descend to the level of the middle or lower medulla.<sup>3,5</sup> At the upper medullary level, FP is contralateral in ventromedial infarction and ipsilateral in dorsolateral infarction.<sup>5,6</sup> A peripheral FP was also described in patients with lateral medullary syndrome<sup>6,7</sup> and it has been attributed to a direct involvement of the facial nucleus or intra-axial facial nerve fascicles due to the extension of the lesion to the pons.<sup>5</sup> Indeed the facial nucleus is located in the lower pons beyond the vascular territory commonly involved in lateral medullary infarction. In lateral medullary infarction, series are not reported whether the FP is voluntary or emotional.

Clinical data suggest that emotional and volitional facial movements are regulated by two distinctive corticofacial tracts.<sup>1,2</sup> It is evident that VFP is found in lesions involving the pyramidal system, whereas EFP is associated with the impairment of extrapyramidal circuits, such as the "anterior fronto-thalamo-pontineconnections" that descend in the anterior limb of the internal capsula.1 EFP has been reported to be associated with lesions involving the midbrain tegmentum<sup>1</sup> and the dorsal pons,<sup>2</sup> suggesting a dorsal location of emotional corticofacial fibers in the brainstem. In the brainstem, the dissociation between voluntary and emotional motility can be explained if the lesion involves the emotional corticofacial fibers and spares the voluntary corticofacial projections. Recent studies suggest different corticofacial projections, even for voluntary movements<sup>5,6</sup>: a main ventral pyramidal tract that (in some humans) may loop into the ventral medullary region, crossing the midline, and ascending in the dorsolateral medullary region to the facial nucleus from below and "aberrant" bundle in a paralemniscal position at the ventral pontine tegmentum.

The ischemic lesion in our patient corresponds to the ascending tract of such looping medullary corticofacial projections, whereas the FP ipsilateral to the lesion suggests that corticofacial projections have been decussated at the level of the dorsolateral upper medulla.<sup>5,6</sup> In addition, the clinical features of our patient suggest that this looping corticofacial tract may contain emotional fibers.

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Received April 5, 2002. Accepted in final form October 8, 2002.

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Figure. (A through D) Central facial paresis is evident when the patient smiles (A) and disappear almost completely during voluntary contraction (B). A hyperintense lesion, corresponding to the ischemic area, involves the left dorsal side of the medulla oblongata. The lesion is located immediately ventral to the profile of the inferior cerebellar pedicle (MR scan, T2-weighted. Axial slice) (C) and is located in the upper medulla oblongata. (D) Coronal slice.

February (2 of 2) 2003 NEUROLOGY 60 723

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## Case report: Recurrent temporalis muscle swelling and headache

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A 43-year-old man presented in December 2001 with a 1-week history of worsening temporal and frontal headaches. On examination he was afebrile with mild tenderness to palpation over the temporalis muscle bilaterally. Neurologic and systemic examination was normal.

Laboratory investigations including hemoglobin, differential white cell count, erythrocyte sedimentation rate (ESR) and electrolytes were unremarkable. A CT scan of the head was normal. A diagnosis of muscle contraction headaches was made and he was started on dothiepin (a tricyclic antidepressant).

Over the next week the headaches increased in severity with pain on jaw opening and required treatment with IM morphine sulfate. Over a 12-hour period he developed remarkable bilateral temporalis muscle swellings.

There was a nonfluctuant, diffuse swelling of the left temporalis muscle. A more nodular thickening was palpated over the lessswollen right temporalis muscle. There were no associated skin changes. Thickening of the left upper buccal mucosa was noted

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and the temporal arteries were pulsatile and normal. Neurologic examination remained unremarkable.

Hematologic investigations as outlined above remained normal. A CT scan of the head now revealed marked swelling of both temporalis muscles (figure, A).

Bilateral temporalis muscle biopsies identified acute-onchronic fasciitis in the fascial layers of both specimens (see figure, B) with a predominantly mononuclear inflammatory infiltrate. A diagnosis of recurrent eosinophilic fasciitis was made. This diagnosis had been previously made in 1994 when this man presented to another hospital with similar headaches. On that occasion, he had also developed bilateral swelling of his temples. Temporalis muscle biopsy revealed eosinophilic fasciitis. Eosinophils were identified in the inflammatory infiltrate and his serum eosinophilic cationic protein was elevated at 63 µg/L (normal range: 2 to 16  $\mu$ g/L). Elevation of the peripheral eosinophil count was not noted during either episode. He was successfully treated with oral corticosteroids. There was a rapid response to oral corticosteroids on both occasions, with resolution of symptoms and signs over a 7-day period. The patient had not experienced headaches in the intervening 7-year period between presentations.

This patient had two episodes of eosinophilic fasciitis affecting the deep fascia of the temporalis muscles within a 7-year period. Eosinophilic fasciitis is an uncommon disorder first recognized by Shulman in the 1970s.<sup>1</sup> It is usually characterized clinically by



Figure. (A) CT scan of head. Axial cut showing bilateral temporalis muscle swelling. (B) Biopsy of left temporalis muscle revealing acute on chronic fasciitis (original magnification ×125).
724 NEUROLOGY 60 February (2 of 2) 2003

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### Emotional facial paresis in a patient with a lateral medullary infarction P. Cerrato, D. Imperiale, M. Bergui, et al. *Neurology* 2003;60;723-724 DOI 10.1212/01.WNL.0000048564.05351.09

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