TRAUMATIC facial palsy is a common entity, in the majority of cases resulting from fracture of the ipsilateral petrous bone. Patients with this pathological entity present immediately or later due to the gradual development of peripheral paresis or paralysis. Central facial palsy can be a consequence of a contralateral hemisphere injury or compression in patients with trauma, stroke, or tumor, usually combined with hemiparesis.¹

To our knowledge, traumatic, isolated central facial palsy has not been previously reported. Moreover, the dissociation between voluntary and emotional movement, which we observed in the patient in the present case, rarely has been described in cases other than those of internal capsular lesions.¹ In the present case, however, after a mild head trauma, this symptom developed as a result of a lesion inferior to the basal ganglia. Finally, we present this case as an exception to the general rule that correlates midline traumatic brain lesions with a prolonged comatose or vegetative condition and poor outcome.

Abbreviation used in this paper: MR = magnetic resonance.

Case Report

History and Examination. This 23-year-old man with an unremarkable medical history was admitted after a motorbike accident (ran into by another vehicle) that occurred while he was driving unhelmeted. Because he was mildly amnesic yet fully oriented and opening his eyes to commands, his condition was graded as a Glasgow Coma Scale score of 14. His pupils were equal in size and reactive to light. Neurological examination revealed ptosis of the left margin angle, noticeable with every mouth movement. There was neither paresis of the left orbicularis oculi nor any other sign of peripheral facial palsy. Motor and sensory function in the upper and lower extremities was intact as well as in all other cranial nerves, and tendon reflexes were produced normally. A computed tomography scan revealed a small (1.5 cm in diameter) hematoma extending from the right thalamus to the cerebral peduncle of the midbrain with no skull base fracture or other parenchymal lesion (Fig. 1).

Treatment. Although there were no major thoracic, abdominal, or bone injuries, there was a chin laceration requiring sutures.
Posttreatment Course. During the next 24 hours the patient recovered to a Glasgow Coma Scale score of 15. Repeated examination of the cranial nerves showed central paresis of the left facial muscles, visibly sparing the emotion-related movements (Fig. 2). Given the atypical pattern of this lesion, we also performed MR imaging, and no underlying lesion was revealed (Fig. 3).

At the 1-month follow-up, mild facial paresis was still detectable with only slight improvement, and the patient reported a left lower-extremity dysfunction, which had developed progressively since his discharge. Neurological examination revealed mild pyramidal weakness in the lower limb. The patient still was able to walk without visible movement disorder.

Discussion

Acute traumatic midbrain hematoma is an uncommon finding in patients after head trauma. Some authors have described it as a discrete entity attributable to a hyperextension mechanism of injury in persons who have sustained an impact on the forehead along the rostrocaudal axis. This mass often coexists with other brain lesions, and affected patients are in a severe state (comatose) with a poor disease prognosis and thus subsumed in the category of those with severe brainstem injuries. In some cases, however, an isolated type has been described with unexpectedly good recovery. The clinical presentation of these patients has included motor disorders (for example, ataxic hemiparesis).

Brainstem contusions have a poor prognosis in terms of survival and functional outcome; apart from representing lesions of critical structures, they indicate the existence of diffuse axonal injury resulting from the classically described mechanism of violent strophic acceleration of the head. In cases of direct impact on the rostrocaudal axis, hyperextension of the neck causes cervical spine injuries. Uncommonly, sudden displacement of the brain can cause contusion of the anterior rostral midbrain.

The patient in the present case sustained a strong blow on the chin, as implied by the skin laceration. According to the hypothesis cited earlier, acute axial displacement of the brain resulted in tissue microlaceration in the region of the rostral midbrain, with the hematoma extending to the contiguous thalamus. The absence of strophic movement of the brain protected the midline structures from axonal injury; therefore, the level of consciousness in the patient was normal. Data in this case suggest a discrete, albeit rare, type of head injury related to a rostrocaudal impact resulting in midbrain contusion, which when isolated may have a benign course in accordance with other cases previously described. Moreover, the preservation of a high level of consciousness clarifies the discrete role of brainstem lesions and diffuse axonal injury on the development of coma in head trauma. The absence of an underlying lesion (that is, a cavernoma) on the MR image along with a clear history of forced injury confirms the hypothetical trauma-induced isolated midbrain hematoma.

Apart from amnesia, the single presenting neurological finding in the patient in the present case was unilateral central facial paresis. The absence of other motor disorders at first was striking, considering the importance of the thalamus and midbrain for extrapyramidal and pyramidal pathways, respectively. The appearance of a slight motor deficiency in the left lower extremity a few days later corroborates the proximity-attributed vulnerability of these pathways.

The corticobulbar fibers for face movement lie medially to the ones for the extremities in the cerebral peduncle, are
responsible for conduction of voluntary facial movements, and end at the motor nucleus of the facial nerve under the floor of the fourth ventricle, which receives corticobulbar fibers for upper face movement from both hemispheres. Thus, a lesion of the midbrain, as in the featured case, results in palsy of the mimetic muscles of the lower face only (central facial palsy).\textsuperscript{1,12}

The cause of supranuclear facial palsy in the majority of cases is an ischemic lesion on the course of the corticobulbar tract. Because of the proximity of the corticospinal tract in the region of the mesencephalon and the pons, stroke affecting these areas results in hemiparesis. Even in the absence of hemiparesis, in small lesions, dysarthria is always present.\textsuperscript{7,9} Presumably, isolated supranuclear facial palsy is more frequently observed in lesions of the precentral cortex, where the zone responsible for facial movement is large enough. Another cortical location for a lesion causing volitional movement–related facial palsy—the supplementary motor cortex—has been reported once.\textsuperscript{8} To our knowledge, isolated supranuclear facial palsy attributed to a brainstem lesion has been described only once in a patient with a small pontine infarct and is thus considered extremely rare.\textsuperscript{10} This information renders our case unique not only in terms of the cause of the paresis (trauma) but also the lesion site.

The pathway for emotion-related facial movement remains unclear because of the difficulty in producing a relevant experimental model for its neurophysiological documentation. It is considered to involve the limbic system and the basal ganglia, but the exact course of its efferent fibers (possibly originating from the hypothalamus and globus pallidus) to the facial nucleus remains unknown.\textsuperscript{1,6,12} Hence, the cases in which only one pathway is affected remain valuable. Although isolated emotional facial palsy has been described in lesions in the basal ganglia, thalamus, temporal lobes, frontal white matter, or supplementary motor cortex, the converse phenomenon is a classic symptom produced by lesions affecting especially the internal capsule (along with ipsilateral hemiparesis) and is rarely attributed to other structures; but pontine lesions have been reported as the cause in some cases.\textsuperscript{6,14,15} In accordance with these cases, the currently described patient presented with isolated voluntary paresis due to a lesion in a higher brain level (midbrain), where the fibers of the two pathways are expected to be separate.

Conclusions

In summary, two unusual entities are associated with this patient. After a rostrocaudally induced head trauma, he presented with a single midbrain lesion, which caused no severe consciousness disorder, implying the discrete character of this type of injury. His injury did result in isolated supranuclear voluntary movement–related facial paresis, a rare reminder of the neurophysiological elements of facial movement.

References