Case report

Bilateral traumatic facial paralysis. Case report

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A B S T R A C T

Although traumatic injury of the facial nerve is a relatively common condition in neurosurgical practice, bilateral lesions related to fracture of temporal bones are seldom seen. We report the case of a 38-year-old patient admitted to Intensive Care Unit after severe head trauma requiring ventilatory support (Glasgow Coma Scale of 7 on admission). A computed tomography (CT) scan confirmed a longitudinal fracture of the right temporal bone and a transversal fracture of the left. After successful weaning from respirator, bilateral facial paralysis was observed.

The possible aetiologies for facial diplegia differ from those of unilateral injury. Due to the lack of facial asymmetry, it can be easily missed in critically ill patients, and both the high resolution CT scan and electromyographic studies can be helpful for correct diagnosis. © 2012 Sociedad Española de Neurocirugía. Published by Elsevier España, S.L. All rights reserved.

Parálisis facial bilateral traumática. A propósito de un caso

R E S U M E N

Aunque el déficit de origen traumático del séptimo par craneal es una entidad común en la práctica neuroquirúrgica, la parálisis facial bilateral asociada a fracturas temporales supone un episodio infrecuente. Describimos el caso de un paciente de 38 años que sufrió un traumatismo craneocefálico severo (Glasgow Coma Scale de 7). En la TC inicial se apreció una fractura de peñasco longitudinal derecha y transversal izquierda. El paciente ingresó en la unidad de cuidados intensivos y, tras la extubación, se comprobó la existencia de una diplejía facial bilateral.

El déficit bilateral del séptimo par craneal posee una etiología distinta al déficit unilateral. En estos casos, la ausencia de asimetría facial puede dificultar el diagnóstico, por lo que la TC de alta resolución y el estudio electromiográfico son útiles para su detección. © 2012 Sociedad Española de Neurocirugía. Publicado por Elsevier España, S.L. Todos los derechos reservados.

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Introduction

Facial palsy is a common diagnosis in otolaryngology and neurosurgery, with an estimated incidence of 20–25 cases per 100,000 inhabitants/year. 1,2 The much rarer simultaneous bilateral facial palsy (SBFP) can be defined as palsy affected both hemifaces during a period of no longer than four weeks. 2–4 This type of palsy has a much lower annual incidence of approximately 1 per 5 million people. Bell’s palsy, or idiopathic facial paralysis, is the most common cause of unilateral facial paralysis, accounting for approximately 70% of cases. 3,4 The differential diagnosis for bilateral facial palsy encompasses systemic, traumatic, neuromuscular, vascular, toxic, infectious, and idiopathic causes. 1,5 While the cause of idiopathic unilateral facial nerve palsy often cannot be assessed, this is not the case for SBFP. SBFP may, for instance, appear as a symptom of Lyme disease, Guillain–Barré syndrome, leukemia, sarcoidosis, bacterial meningitis, syphilis, leprosy, Moebius syndrome, thus making the diagnosis a significant challenge for the clinician. 2,6

Case report

We report the case of a 38-year-old-patient admitted to the emergency room for a severe head trauma. The injury was sustained when the patient fell 3 m while at work. During the initial examination, the patient showed decreased consciousness (Glasgow Coma Scale [GCS] score of 7) with isochoric and slowly reacting pupils and no clear signs of neurological deficit. A bilateral otorrhea was detected. The patient was intubated and an urgent computed tomography (CT) scan was obtained, showing a right temporal epidural hematoma with a small associated pneumoencephalon, as well as a traumatic subarachnoid hemorrhage on the left temporal lobe with no midline shift (Fig. 1).

Both a right longitudinal and a left transverse petrous fracture were detected in the bone window (Fig. 1). Due to the clinical situation of the patient and the findings on the CT scan, an intracranial pressure (ICP) sensor was placed for monitoring. The patient was admitted in the Intensive Care Unit (ICU) of our hospital where good control of ICP was achieved with medical anti-edema measures. While in the ICU, the patient contracted a ventilator-associated pneumonia, resulting in a prolonged stay. During the physical examination after the extubation, a bilateral peripheric facial palsy was detected (Fig. 2a and b). The palsies were grades V and VI on the right and left sides according to the House-Brackmann classification with a bilateral Bell’s sign and no other neurological deficit.

The patient was discharged 46 days after admittance without any improvement in his symptoms. An electromyogram during the follow-up showed severe axonal injury of both facial nerves. Mixed nerve conduction velocity showed very low amplitude compound muscle action potential and increased latencies. Needle study confirmed complete denervation of all facial muscles, with profuse spontaneous muscle activity at rest, and no voluntary activation of any motor unit.

Fig. 1 – CT scan on admission, showing a right longitudinal temporal fracture (a), a left transversal temporal fracture (b), a right epidural hematoma (c, d), and a traumatic left subarachnoid hemorrhage (c, d).

Fig. 2 – Physical examination showing “mask face” (a) and a bilateral positive Bell’s sign (b).

Discussion

In this brief communication, we report our experience with a case of bilateral traumatic facial palsy, a condition only very seldom described in English scientific literature (Table 1). 3,8–16

Unlike unilateral facial palsy, SBFP is considered idiopathic in only 20–23% of cases 6,17 and usually indicates a more serious alteration. Wormald et al. 4 defined three distinct major groups for SBFP: sclerosteosis, trauma, and Bell’s palsy, although some other ailments have later expanded the list of plausible causes.

The most significant causes of SBFP are brain trauma, infectious diseases (infectious mononucleosis, syphilis, bilateral otitis media, herpes zoster, Lyme disease, meningitis), neurological diseases (multiple sclerosis, neoplasm, or stroke), and...
disorders of undetermined origin (Guillain–Barré syndrome, sarcoidosis, Melkerson–Rosenthal syndrome, leukemia, Bell’s palsy).\(^5\)\(^6\)\(^7\) A complete list for the differential diagnosis is given in Table 2.

<table>
<thead>
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<th>Table 2 – Differential diagnosis for acquired bilateral facial palsy.</th>
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**Idiopathic**
- Bell’s palsy

**Neurological**
- Multiple sclerosis
- Bulbar palsy
- Parkinson’s disease

**Autoimmune**
- Sarcoidosis
- Amyloidosis
- Wegener’s disease

**Neoplastic**
- Leukemia
- Acoustic neuroma
- Meningioma

**Metabolic**
- Diabetes
- Acute porphyria

**Infection**
- Post influenza
- HIV Infection
- Guillain–Barré syndrome
- Syphilis
- Brainstem encephalitis
- HTLV-1 infection
- Poliomyelitis

**Trauma**
- Skull base fractures
- Parotid surgery
- Mastoid surgery
- Mandibular fractures

Head trauma is responsible for almost 5% of all-cause facial paralysis. Temporal bone fracture is a relatively common cause of unilateral facial nerve injury, as the temporal bone is affected in more than one third of basilar fractures. Fractures of the petrous bone are classified as either transverse or longitudinal, in accordance to their direction with respect to the long axis of the temporal bone. Transverse fractures, in which the fracture line runs vertically through the squamous petrous bone, account for 10% of all temporal bone fractures. Approximately 30–50% of patients with a transverse fracture of the temporal bone have facial paralysis accompanied by neurosensory hearing loss.\(^5\)\(^8\) Facial paralysis is generally more severe and the prognosis is poor when compared to those from a longitudinal fracture. For longitudinal fractures, the fracture line runs horizontally along the long axis of the temporal bone. This type of fracture accounts for 90% of all temporal bone fractures, with facial paralysis occurring in 10–25% of cases.\(^7\)\(^12\)

Traumatic bilateral facial palsy are rare since they are associated with severe trauma causing extensive skull base fracture, and thus associated with a very high mortality. In a large proportion of cases the condition may remain unnoticed, as death probably occurs in many before identification of the facial weakness.\(^3\)

Traumatic facial nerve injury can be classified into two categories: immediate and delayed onset of facial paralysis. On injuries of immediate onset, the nerve is directly lacerated or contused at the site of fracture as a result of bone splinters, or otherwise an entrapment, crushing or traction of the facial nerve. Most reports indicate that the delayed onset facial paralysis is more common than immediate onset.\(^3\)\(^9\)\(^12\)\(^16\) Delayed onset occurs within an average of 4–5 days following head injury. The cause may be edema, delayed arterial spasm, arterial or venous thrombosis or external compression by hematoma fluid. The nerve is intact initially, but delayed substaned compression results in partial or complete Wallerian degeneration of the nerve.\(^1\)\(^3\)\(^12\) Since our patient could not be examined on admission we consider a delayed onset injury as the most probable origin.
Electrodiagnostic studies are often used to determine the prognosis for patients with traumatic facial palsy. The prognosis is dependent upon whether the nerve injury being regarded as neuropraxia, axonotmesis, neurotmesis or nerve transection. Neuropraxia occurs when the myeline sheath is damaged while the axon is spared, causing a conduction block. Recovery is invariably rapid and complete. Axonotmesis involves transection of axon with sparing of the nerve connective tissue sheath. Wallerian degeneration will occur 7 days later in the distal axon, followed by an attempt of regeneration from neighboring, undamaged, Schwann cells. Recovery is expected and often satisfactory once axon regeneration occurs within the intact nerve sheath. In the case of neurotmesis, the nerve is severed with loss of axonal and nerve sheath continuity, with regeneration usually being remote and incomplete. If a complete nerve transection occurs with the presence of a gap, surgical intervention with nerve grafting is required to close this gap.8,12

Patients with facial nerve injury often experience corneal irritation and ulceration. This is due to inadequate lubrication of the eyes and lid lag (Von Graefe sign), both of which are potential causes for keratitis sicca. In severe cases that go unrecognized, blindness can occur. It is, therefore, important to provide eye care such as artificial tears or eye ointment at night to protect the cornea.13

In the presence of significant intra-cranial injury, as in our case, early detection of bilateral facial nerve injury can be challenging since there may be a paucity of spontaneously generated facial or eyelid movements. However, in the presence of temporal bone fractures, a high suspicion of possible cranial nerve damage is warranted.14 When a facial nerve injury is bilateral and symmetric, the lack of asymmetry in associated movements can make clinical detection more difficult. Thus, the detection of Bell’s phenomenon and inadequate eyelid closure can be very helpful.3,8,12

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