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Facial Diplegia Secondary to Cranio-Maxillo-Facial Trauma: About a Case in the Cocody Teaching Hospital

Konan René Kouakou*, Konan Marc Koffi, Ahi Morel Chapo, Mamadou Atigou Baldé, Kader Morel Diarra, Aké Jonathan Lucien Yapo, Amadou Lamarana Diallo, Brice Anderson Youmbi, Koffi Laurent Boka, Bakary Ouattara, Raphiou Diallo

Oral and Maxillofacial Surgery Department of the Cocody Teaching Hospital, Abidjan, Côte d'Ivoire
Email: *rkouak1@gmail.com

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Abstract

Introduction: Facial diplegia refers to bilateral peripheral facial paralysis. It is a rare complication of craniomaxillofacial trauma that poses a diagnostic and therapeutic challenge. The purpose of this article is to highlight the diagnostic and therapeutic challenges in our practice. **Observation:** A 29-year-old patient, with no particular pathological history, was admitted to the department on 12/22/2023 for maxillofacial trauma with initial loss of consciousness following a road traffic accident. Clinical and radiological investigations revealed bilateral extra-labyrinthine fractures of the petrous bones associated with a Lefort II fracture, an intermaxillary disjunction, a fracture of the mandibular symphysis and frontal pneumencephaly. He underwent maxillofacial osteosynthesis on 02/01/2024. However, neurosurgical abstention was prescribed. Pneumo23 and anti-meningococcal vaccines were administered. The evolution was marked, on postoperative day 14, by simultaneous facial diplegia classified HB5 on the left and HB6 on the right according to the House and Brackmann classification. He was put on corticosteroid therapy and physiotherapy. After 3 months, there was a regression of facial paralysis on the left but persistence on the right still at HB5. **Discussion:** Facial diplegia is a rare complication of cranio-maxillo-facial trauma. Its diagnosis is difficult in our practice and its treatment, which is not consensual, remains a challenge.

Keywords

Facial Diplegia, Maxillo-Facial Trauma Temporal Bone Fracture, Diagnosis, Treatment

1. Introduction

The facial nerve is the 7th pair of cranial nerves (VII), it innervates all of the platysma muscles of the face. These muscles are responsible for all facial movements. Unilateral peripheral damage to the VII defines left or right peripheral facial paralysis characterized by facial asymmetry, the healthy side being responsible for a contralateral attraction to the affected side [1].

Facial diplegia is bilateral facial paralysis resulting from bilateral facial nerve involvement. Studies have shown an etiological diversity of facial diplegia including infectious, systemic, autoimmune, idiopathic, toxic, iatrogenic, metabolic, genetic, congenital and traumatic causes [2] [3]. However, facial diplegia secondary to craniofacial trauma is a rare clinical entity [4].

Its diagnosis is clinical, radiological, and electrophysiological, but it is often delayed in the context of severe craniofacial trauma. The treatment, which is multidisciplinary, is not clearly codified.

The purpose of this article is to highlight the diagnostic difficulty and discuss treatment in our practice setting.

2. Observation

The patient was a 29-year-old man living in the interior of the country and working in an informal liberal profession, admitted to the department on 22/12/2023 for maxillofacial trauma with initial loss of consciousness and bilateral otorrhagia following a road traffic accident. The patient, a motorcyclist without a helmet, was hit by a vehicle and thrown onto the asphalt ground.

The parents' interview revealed no prior medical or surgical history, either personal or family.

The general examination upon admission revealed a patient with obtunded facial features, a Glasgow Coma Scale (GCS) score of 14/15, and no motor deficits in the limbs; hemodynamic and respiratory vital signs were unremarkable, and his body weight was 60 kg.

Exo-oral examination revealed, upon inspection, a "Y"-shaped wound in the frontal region exposing the bone, global facial swelling, a telecanthus, and a periorbital ecchymosis. Vision, ocular motility, and eyelid occlusion were unremarkable.

Palpation revealed pain and a sensation of stepping on the root of the nasal bones, on the infraorbital margins and the basilar edge of the mandible on the right. The search for the Pierre Marie and Foix sign was negative. The intraoral examination revealed a dental articulation disorder, a mucosal wound between the 11th and 21st, between the 41st and 31st, a limitation of the oral opening to 2.5 cm, abnormal mobility of the mandibular corpus, mobility of the maxilla, pain on palpation of the zygomas.

The ENT examination revealed bilateral otorrhagia without tympanic membrane lesions.

Craniomaxillofacial CT scan demonstrated a Lefort II craniofacial disjunction

associated with an intermaxillary disjunction, a mandibular symphyseal fracture, a bilateral extralabyrinthine petrous fracture (Figure 1 and Figure 2), and minimal frontal pneumencephaly. Emergency medical treatment consisted of 1500 IU of tetanus serum, pneumo-23 and meningococcal A-C vaccines, and ceftriaxone at a dose of 1 g twice every 24 hours.

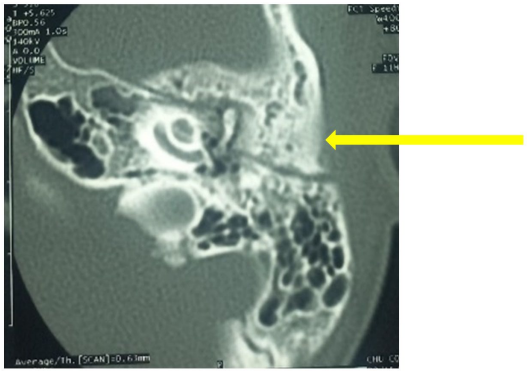

Figure 1. Left petrous bone fracture on axial CT scan.

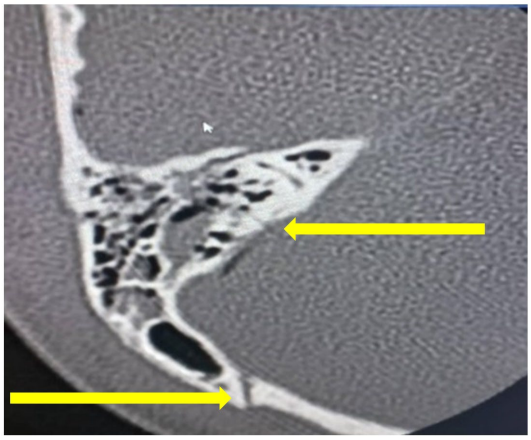

Figure 2. Right petrous bone fracture on axial CT scan.

On day 11 of hospitalization, the patient underwent maxillofacial osteosynthesis. Neurosurgical abstention was indicated for the temporal bone fracture, which was not accompanied by any obvious functional complications.

The evolution was marked on post-operative day 14 by the observation of facial diplegia estimated at grade V (HB5) on the left and grade VI (HB6) on the right according to the House and Brackmann classification (Figure 2). The neurosurgical opinion indicated a first-line medical treatment consisting of oral corticosteroid therapy based on beta-methasone 2 mg for a duration of three months was prescribed at a decreasing dose ranging from 0.2 mg/kg/day or 12 mg per day for 15 days then 0.1 mg/kg/day or 6 mg per day for 15 days and 4 mg/day for 15 days followed by 2 mg/day up to 15 days and finished with 1 mg/day for 15 days. Corticosteroid therapy was combined with vitamin B1 therapy at a dose of one tablet per day for 30 days. In addition, facial physiotherapy 2 to 3 times a day was per-

formed and consisted of massages and guided facial expressions several times a day combined with electro-stimulation once a day for 3 months. After 3 months of treatment, regression was more significant on the left side, classified as grade II (HB2), than on the right side, classified as grade V (HB5) (Figure 3 and Figure 4). The patient was subsequently lost to follow-up.

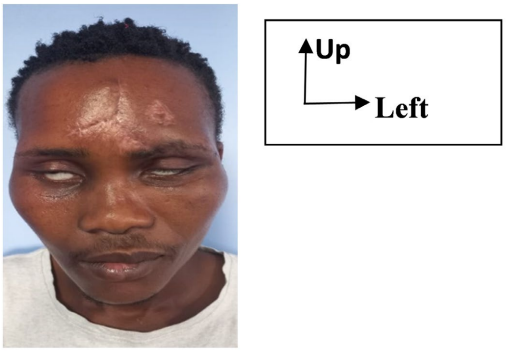

Figure 3. Facial diplegia more marked on the right.

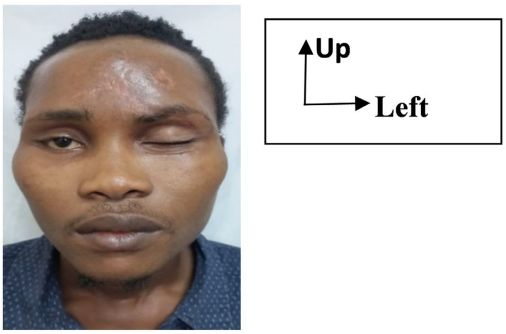

Figure 4. Regression of left facial paralysis.

3. Discussion

Facial diplegia is a rare clinical picture. Its prevalence is between 2% and 3% of facial paralysis with an estimated incidence of 1 in 5,000,000 per year [5] [6]. Its rarity could explain the existence of very few serial studies in the literature, which remains dominated by case reports [7]. The causes of facial diplegia are very varied. They can be of infectious, auto-immune, metabolic, congenital, traumatic or idiopathic origin [8]-[10]. Post-traumatic facial diplegia is rarely observed and is a consequence of a bilateral petrous bone fracture. Facial diplegia secondary to bilateral temporal bone fracture represents 3% of all facial diplegia [4]. In their study, they reported that there is a 6% to 7% risk of facial paralysis in patients with temporal bone fractures. Cranio-facial trauma can occur in the context of road traffic accidents, work accidents or domestic accidents.

The clinical case we report is the result of a road traffic accident. Medical imaging demonstrated bilateral petrous fracture. Facial diplegia following craniofacial trauma affects subjects with an average age of 27 years, regardless of gender [7] [11].

The age of the patient in this clinical case is consistent with the literature and corresponds to data on cranio-facial trauma which reports a male predominance and female sex. Post-traumatic cranio-facial diplegia is less common in children than in adults due to a relative plasticity of the child's skull [5] [6] [11].

Facial diplegia manifests clinically, in the cooperative patient at rest, by complete and symmetrical facial atony of the upper and lower facial areas, a fixed facial expression with complete disappearance of facial wrinkles and folds, widening of the palpebral fissures, drooping of the lower lip, and an inability to close the mouth. The Charles Bell sign is bilateral with an abolition of reflex palpebral occlusion [11].

In the context of severe cranio-facial trauma, facial diplegia may go unnoticed at the time of the initial clinical examination. In the study by Darrouzet *et al.*, the rate of initial search for facial paralysis at the time of management of patients with head trauma and reported that 58% of patients had not had an initial examination of facial motor skills [12] [13]. In the present clinical case, both the disturbance of consciousness and the more spectacular lesions such as swelling and facial wounds had concealed the immediate onset of facial diplegia and would explain the delay in diagnosis or the paralysis was of secondary onset. From the first examination, great importance must be given to the search for immediate facial paralysis.

The craniofacial CT scan performed on our patient demonstrated the bilateral petrous fracture. CT scan, by specifying the relationship of the fracture with the facial canal, allows us to suspect damage to the facial nerve [13] [14]. Magnetic resonance imaging is particularly indicated in post-traumatic facial paralysis where the fracture is not highlighted by CT scan [12]. Its performance was therefore not indicated in this clinical case where the fracture lines were clear on CT scan.

Electrophysiological tests are of interest for the early diagnosis of facial nerve damage and for the prognosis of facial paralysis because they provide information on the diagnosis of severity, the location and type of nerve damage, but also on possible recovery. Detection electromyography, Esslen electroneurography, Freyss muscle testing allow the initial clinical assessment of the severity of facial paralysis [15].

In some cases, electrophysiological tests can be used to decide whether surgical decompression treatment is indicated [15] [16]. However, these explorations, which are commonly performed in developed countries, are not performed in our emergency practice, due to the low level of the technical platform.

The treatment of facial paralysis secondary to a temporal bone fracture is medical or medical-surgical. Medical treatment aims to reduce nerve inflammation and combat nerve ischemia. Several authors have used, without a codified protocol, corticosteroid therapy, vasodilators, vitamin B1 therapy and facial physiotherapy, the duration of which varies depending on the study. [16]-[18]. This medical treatment was administered to the patient. However, no study reports the effectiveness of steroids in the treatment of traumatic facial paralysis, as they are in Bell's palsy [19].

Besides medical treatment, surgical decompression of the facial nerve is a therapeutic method for facial paralysis secondary to temporal bone fracture. Bougacha *et al.* [10] reported 80% improvement in facial paralysis after a 6-month follow-up. According to these authors, surgical treatment depends on the time of onset, the complete or incomplete nature of the facial paralysis and the electrophysiological data.

The patient is first placed on medical treatment and reassessed daily for the first three weeks. Clinical worsening resulting in progression to complete facial paralysis associated with electrophysiological worsening resulting in complete denervation, then an absence of signs of spontaneous reinnervation, points towards a surgical indication. If facial paralysis is complete, but the time to onset is not known, then the absence of clinical improvement associated with denervation of more than 90% of the fibers points towards surgery. If facial paralysis is immediate and complete, then surgical exploration is indicated immediately.

In this clinical case, since the time to onset was unknown, the late diagnosis and the lack of financial means were factors limiting the indication for surgical decompression of the facial nerve.

The evolution in our patient was marked by regression of peripheral facial paralysis on the left (HB2) and persistence on the right (HB5) according to the House-Brackmann classification [20] after a three-month follow-up. The improvement on the left could be explained by a simple contusion and the absence of regression on the right by a total transection.

At the sequelae stage, surgical treatment of the sequelae of facial paralysis following craniofacial trauma and secondary to a fracture of the temporal bone may involve facial reanimation, which consists of plastic procedures such as dynamic muscle transfers (transposition of the temporal muscle), commissural suspension, associated with the treatment of lagophthalmos [19] [21] [22]. These facial reanimation procedures could be proposed to the patient in this clinical case.

4. Conclusion

Facial diplegia following cranio-facial trauma is a rare complication to be investigated in cases of bilateral temporal bone fracture. Its rarity should not obscure the risk of occurrence. Its prognosis is reserved in our practice setting due to the inadequacy of the technical platform, which does not allow for accurate and early lesion diagnosis and effective therapeutic management. Access to a detection electromyograph and an electroneurograph could improve the prognosis of facial paralysis following cranio-facial trauma in our practice.

Ethical Consideration

This article benefited from the patient's free consent.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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