

Functional outcome of the facial nerve paralysis after late surgical decompression in otic capsule-sparing fracture

Saleh K. Aboud^a, AbAziz Aini^b and Asma Abdullah^c

Departments of ^aOtorhinolaryngology – Head and Neck Surgery, ^bRadiology and ^cOtorhinolaryngology, University of Kebangsaan Malaysia, Kuala Lumpur, Malaysia

Correspondence to Saleh K. Aboud, MRCS (ENT) (Glasgow), MS (ORL-HNS), Department of Otorhinolaryngology – Head and Neck Surgery, Malaysian Allied Health Sciences Academy University (MAHSA University), Jalan Elmu of Jalan University, 59100 Kuala Lumpur, Malaysia
Tel: + 603 7965 2555; fax: + 603 7931 7118; e-mail: salehkh70@gmail.com

Received 11 February 2013

Accepted 20 July 2013

The Egyptian Journal of Otolaryngology

2013, 29:280–282

Temporal bone fracture (TBF) following head trauma is a well-known cause of facial nerve paralysis (FNP) and almost 22% of all skull fractures are TBF. It has been estimated that 25–70% of TBF are associated with FNP and otic capsule-violating fracture of the petrous portion of the temporal bone is more commonly associated with FNP, although otic capsule-sparing fracture is more common. (a) The incidence of FNP is reported to be 10–25% of otic capsule-sparing fracture. (b) With otic capsule-sparing fractures, FNP is often delayed 2–3 days, and is incomplete and temporary, with good recovery during a period of 6–8 weeks. Most clinicians seldom consider a surgical intervention in delayed FNP; however, surgical exploration may be required for complete recovery. The optimal timing for surgical exploration of traumatic FNP to best preserve facial function is currently controversial. Decompression surgery still has a beneficial effect if performed within 14 days of injury. However, late exploratory surgery is recommended in patients who do not experience recovery of facial nerve function, patients who cannot be operated on early, and who present 1–3 months after TBF with more than 95% denervation on electroneurography. Here, we report a case of delayed-onset FNP of otic capsule-sparing fracture with an excellent outcome after late surgical decompression at 6 months after trauma.

Keywords:

facial nerve decompression, facial nerve paralysis, otic capsule-sparing fracture

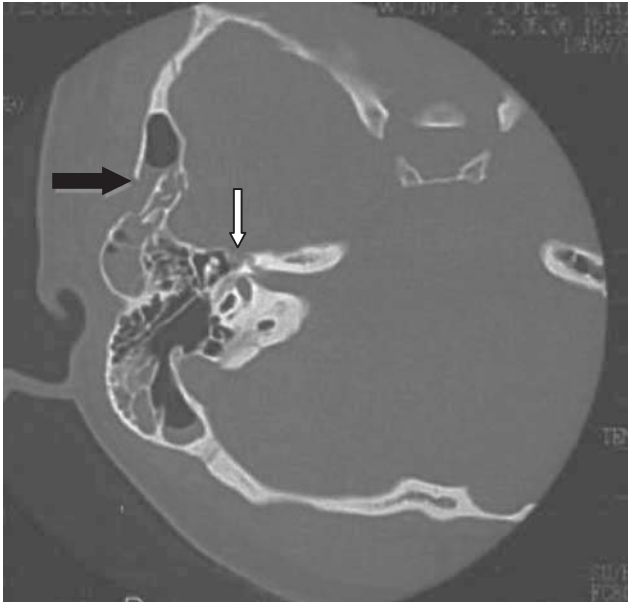
Egypt J Otolaryngol 29:280–282
© 2013 The Egyptian Oto - Rhino - Laryngological Society
1012-5574

Introduction

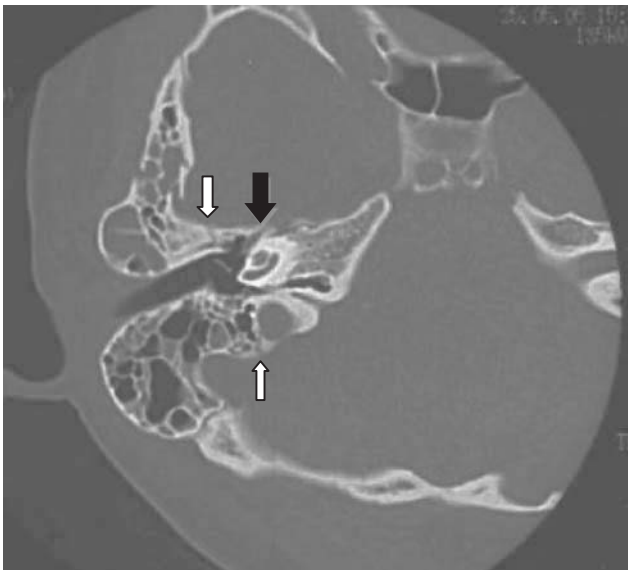
Temporal bone fracture (TBF) following head trauma is a well-known cause of facial nerve paralysis (FNP) and almost 22% of all skull fractures are TBF. It has been estimated that 25–70% of TBF are associated with FNP and otic capsule-violating fracture of the petrous portion of the temporal bone is more commonly associated with FNP, although otic capsule-sparing fracture is more common [1]. The incidence of FNP is reported to be 10–25% of otic capsule-sparing fractures [2]. With otic capsule-sparing fractures, FNP is often delayed 2–3 days, and there is incomplete and temporary, with good recovery during a period of 6–8 weeks. Most clinicians seldom consider a surgical intervention in delayed FNP; however, surgical exploration may be required for complete recovery. The optimal timing for surgical exploration of traumatic FNP to best preserve facial function is currently controversial. Decompression surgery still has a beneficial effect if performed within 14 days of injury. However, late exploratory surgery is recommended in patients who do not experience recovery of facial nerve function, patients who cannot be operated on early, and who present 1–3 months after TBF with more than 95% denervation on electroneurography (ENoG). Here, we report a case of delayed-onset FNP of otic capsule-sparing fracture with an excellent outcome after late surgical decompression at 6 months after trauma.

Case report

A 19-year-old woman was involved in a motor vehicle accident and sustained injury to the right parietal region, causing a transient loss of consciousness and bleeding from the right ear. Initially, she was evaluated and managed in another institution. Computed tomography scan of the brain showed right TBF with pneumocranium. The patient was treated conservatively and recovered well, without any adverse neurological consequences. At day 4 after trauma, she complained of right facial asymmetry and mild hearing loss of the ipsilateral ear. A high-resolution computed tomography scan of the temporal bone was performed and showed comminuted fracture of the right mastoid bone and a transverse fracture of the right petrous bone. However, no compromise to and along the course of the facial nerve was seen (Figs 1 and 2). Subsequently, the patient was referred to our medical center for further management. Upon arrival on day 6 after trauma, she was fully conscious and the neurological examination showed a right lower motor neuron lesion of facial nerve palsy (House–Brackmann grade 5) with Bell's eye phenomenon. The pure tone audiometry indicated mild conductive hearing loss on the right ear with an air–bone gap of 20 dB. Schirmer's test was negative as the stapedial reflex on the right side. ENoG of the facial nerve performed on day 21 after trauma showed evidence of 95.2% axonal degeneration of the right facial nerve. On the basis of her high-resolution computed tomography and ENoG results, the decision to

Figure 1

High-resolution computed tomography of temporal bone (axial section) showing the first genu of the facial nerve (white arrow), comminuted, and transverse fracture of the right mastoid (black arrow).

Figure 2

High-resolution computed tomography of temporal bone showing the transverse fracture of the petrous (white arrows) and tympanic segment of the facial nerve (black arrow).

perform surgical exploration using a transmastoid approach was made. Initially, the patient declined any treatment and default subsequent follow-up. Later, right facial nerve decompression was performed 6 months after trauma. Intraoperatively, the tympanic segment and first genu of the facial nerve were edematous and compressed by bone spicules. The latter were removed and the proximal and distal end of the edematous facial nerve was decompressed by facial nerve sheath slitting (neurolysis). She was being reviewed periodically on an outpatient basis and showed

complete resolution (House–Brackmann grade 1) 2 months after surgical decompression with hearing preservation.

Discussion

The use of the terms ‘longitudinal’ and ‘transverse’ is limited and does not correlate accurately to clinical sequelae. Hence, the classification of fractures using the ‘otic capsule violating’ or ‘otic capsule sparing’ system appears to be a more accurate predictor of clinical outcome. A retrospective dissection of 100 temporal bones found that the gravest damage to the facial nerve occurred in fractures involving the otic capsule [3]. FNP is uncommon in otic capsule-sparing fractures and it is usually of delayed onset, which carries a good prognosis and often does not require surgical intervention. The delayed traumatic FNP is more likely because of edema and is typically treated with high-dose corticosteroids with further intervention on the basis of the results of the electrodiagnostic testing [4]. The most common site of injury of the facial nerve is in its geniculate or proximal tympanic segment [5]. Some reviews suggest that among otic capsule-sparing fractures, 43% involve intraneural hematoma or contusion at the level of the geniculate ganglion, 33% have bony impingement into the nerve sheath, 15% have transection, and 12% have no identifiable pathology [6]. Severe traction and stretching of the greater superficial petrosal nerve can lead to the formation of an intraneural hematoma and secondary edema that extends retrogradely along the proximal part of the facial nerve. Compression injury does not produce any interruption in nerve conduction and is akin to neurapraxia, which recovers promptly in 2–3 weeks. Crushing or stretching injury causes interruption in nerve electric conduction and is similar to axonotmesis, which recovers in a period of 3–6 months. Divisional injuries shows complete interruption of nerve conduction and are akin to neurotmesis, which recovers after 6 months of injury. Delayed traumatic FNP have a more favorable prognosis than immediate paralysis. Persistence of edema is not surprising as a grossly edematous nerve can be found even in patients undergoing decompression of the facial nerve 2 months to 3 years after the onset of paralysis. Surgical treatment of post-traumatic facial [5] paralysis is useful, if well indicated, all the more because patients are often young and psychologically affected. The earliest possible decompression within the first 2 weeks has been recommended even if the facial nerve has not been severed or only compressed by a bony fragment or a hematoma. However, patients with an unstable condition may complicate the decision on the optimal timing of surgical exploration. Others have suggested that surgical exploration of the facial nerve is indicated at any time and may lead to better facial recovery even in very old injuries [7]. The choice of the approach in traumatic FNP depends considerably on the presence of serviceable hearing following trauma. The horizontal segment of the facial nerve and geniculate ganglion can be exposed using the transmastoid transattical approach. Our patient showed bony chips compression and edema of the facial nerve, which was managed by facial decompression and neurolysis

with postoperative House–Brackmann grade 1 although 6 months had elapsed before surgical decompression.

Acknowledgements

Conflicts of interest

There are no conflicts of interest.

References

- 1 Chan EH, Tan HM, Tan TY. Facial palsy from temporal bone lesions. *Ann Acad Med Singapore* 2005; 34:322–329.

- 2 Wysocki J. Cadaveric dissections based on observations of injuries to the temporal bone structures following head trauma. *Skull Base* 2005; 15:99–107.
- 3 Gladwell M, Viozzi C. Temporal bone fractures: a review for the oral and maxillofacial surgeon. *J Oral Maxillofac Surg* 2008; 66:513–522.
- 4 Işık HS, Bostancı U, Yıldız O, Özdemir C, Gökyar A. Retrospective analysis of 954 adult patients with head injury: an epidemiological study. *Ulus Travma Acil Cerrahi Derg* 2011; 17:46–50.
- 5 Bascarevic V, Samardzic M, Rasulic L, Simic V. Reconstructive surgery of facial nerve injuries. *Acta Chir Lugosl* 2003; 50:63–67.
- 6 Chang CY, Cass SP. Management of facial nerve injury due to temporal bone trauma. *Am J Otol* 1999; 20:96–114.
- 7 Sanuş GZ, Tanrıöver N, Tanrıverdi T, Uzan M, Akar Z. Late decompression in patients with acute facial nerve paralysis after temporal bone fracture. *Turk Neurosurg* 2007; 17:7–12.