

**TITLE: Temporal Bone Fracture**

**SOURCE: Grand Rounds Presentation, UTMB, Dept. of Otolaryngology**

**DATE: March 31, 2010**

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## **Introduction**

Temporal bone fracture is a frequent consultation otolaryngologists face in the emergency setting. Anatomic knowledge of the many vital structures within the temporal bone is vital to proper diagnosis and management of such injuries. Appropriate evaluation takes into account the spectrum of severity and the sometimes subtle symptoms of otologic trauma. Early involvement of the otolaryngologist/neurotologist in evaluation and management can improve long-term functional outcome.

## **Incidence and Epidemiology**

Temporal bone fractures occur in approximately 14-22% of all skull injuries. Most of these fractures are unilateral, with bilateral fractures reported in 9% to 20%. Children account for 8-22 % of patients with temporal bone fracture.

The primary mechanisms of injury include motor vehicle accident (12%–47%), assault (10%–37%), falls (16%–40%), and gunshot wound (3%–33%). With improved automobile safety technology, the incidence of fractures resulting from motor vehicle accidents has decreased. On the other hand, increasing rates of violent crimes have led to more temporal bone injury from assault.

## **Classification**

Traditionally temporal bone fracture has been classified into longitudinal fracture (~ 80%) and transverse (~ 20%) based on several cadaveric studies in the 1940s. Longitudinal fracture results from temporoparietal impact and the most frequent structures involved are the tympanic membrane, the roof of the middle ear, and the anterior portion of the petrous apex. About 15-20% will have involvement of the facial nerve, and injury occurs near the geniculate ganglion or in the horizontal portion. Facial paralysis is often delayed in onset, attributed to edema rather than direct interruption of the nerve. Vestibular involvement and sensorineural deficits are relatively uncommon and are attributed to concussive effects rather than direct trauma on the vestibular labyrinth and cochlea.

Transverse fracture results from fronto-occipital impact and courses perpendicular to the long axis of the petrous pyramid from the foramen magnum through the posterior fossa, through the petrous

pyramid, including the otic capsule, and into the middle cranial fossa. The facial nerve is involved in 50% of cases. Otic capsule and internal auditory canal are frequently involved as well.

However, this dichotomous system was deemed by some to be insufficient. It was reported that up to 90% of blunt trauma-induced fractures were more accurately described as mixed or oblique fracture.

More recently, there is also an increasing trend for categorizing temporal bone fracture into otic capsule sparing (OCS) versus otic capsule disrupting (OCD) fractures, a system showing better correlation with clinical sequelae. OCS fractures are much more common (>90%) than OCD, and the latter is associated with higher incidence of facial nerve injury (30-50%), SNHL, and CSF leak (2-4 times higher than OCS).

## **Diagnostic Workup**

In addition to gathering an adequate history of the patient and the mechanism of injury, a complete head and neck examination is necessary. The patient with multiple systems trauma must proceed according to the ATLS protocol of emergency resuscitation. Immobilizations of any cervical spine injuries are immediately performed. Before the advent of CT scan, the diagnosis of temporal bone fractures were made on the basis of physical exam alone. Blood in the external ear canal may be more representative of longitudinal than transverse fractures. Hemotympanum and blood in the external auditory canal are some of the common findings in temporal bone fractures. Evidence of a basilar skull fracture includes Battle's sign and raccoon eyes (periorbital ecchymosis). Pneumatic otoscopy may initiate the nystagmus and vertiginous symptoms of a perilymphatic fistula, or reveal a subtle fracture of the malleus. Tuning fork exam is a quick and easy way to acquire information about the type of hearing loss prior to the availability of a formal audiogram.

High resolution CT scan (HRCT) is the most commonly used radiographic modality in evaluating the patient with temporal bone trauma. HRCT provides excellent delineation of bony anatomy and allows for evaluation of the facial canal, ossicular chain, otic capsule, carotid canal, and middle cranial fossa.

## **Hearing Loss**

More than half of patients with temporal bone trauma report some degree of hearing loss. The type and degree of deficit is related to the force of injury and location of the fracture. Transverse fractures involving the otic capsule and internal auditory canal frequently cause severe sensorineural hearing loss. Longitudinal fractures are more likely to cause conductive or mixed hearing loss. Incudostapedial joint dislocation is the most common ossicular chain injury in temporal bone fracture. Even without temporal bone fractures, concussive injuries to the cochlea or labyrinth can cause hearing loss.

Early audiometric evaluation will frequently show CHL secondary to hemotympanum. It is therefore advised that audiogram should be repeated about 1-2 months after the injury to allow hemotympanum and middle ear effusion to resolve. In the short-term management of CHL, most authors prefer to wait to determine if the loss will resolve spontaneously. However, if earlier neurotologic intervention is planned (eg, facial nerve decompression, CSF leak repair), ossiculoplasty can be used as appropriate for concomitant exploration. For patients who experience persistent CHL after the acute recuperative

3 to 4 months, ossicular dislocation or fracture must be suspected, and exploration with ossiculoplasty is indicated. Patients who have mild to moderate SNHL are usually treated with standard

hearing aid amplification. For unilateral profound SNHL, bone anchored hearing aid has been demonstrated with good outcome. Cochlear implantation has also been shown to have benefits in treating patients with bilateral profound SNHL after temporal bone fractures.

**Facial Nerve injuries**

Facial nerve is injured in approximately 15-20% of longitudinal fractures and 50% of transverse fractures. Chang and Cass’s review suggests that of longitudinal fractures; 43% had intraneural hematoma or contusion, 33% had bony impingement, 15% had transaction, and 12% had no identifiable pathology. In contrast, in transverse fractures, 92% had transection and 8% had bony impingement.

Early evaluation and thorough history are crucial in evaluating the status of facial nerve. Particular attention should be given to the time and characteristics of onset of facial weakness, whether sudden or delayed, and determination of complete versus incomplete paralysis.

The House- Brackmann grading system was designed to classify the long term degree of facial nerve deficit but is also useful to describe acute facial weakness.

|     | <b>Grade</b>      | <b>Characteristics</b>  |
|-----|-------------------|---|
| I   | Normal            | Normal facial function  |
| II  | Mild              | Slight synkinesis, no asymmetry, slight weakness  |
| III | Moderate          | Complete eye closure, noticeable synkinesis, no asymmetry at rest, obvious weakness, slight forehead movement |
| IV  | Moderately Severe | Incomplete eye closure, no asymmetry at rest, no forehead movement  |
| V   | Severe            | Asymmetry at rest, barely perceptible motion  |
| VI  | Total             | No movement   |

ENoG is considered to be the most accurate prognostic test because it provides quantitative, objective measurement of neural degeneration. An electrode is placed near the stylomastoid foramen and a transcutaneous stimulus is applied. The muscular response is then measured using bipolar electrodes placed near the nasolabial groove. The peak-to-peak amplitude wave is then measured and compared to the contralateral side. A reduction of greater than 90% amplitude correlates with a poor prognosis for spontaneous recovery. A reduction of less than 90% gives an expected spontaneous rate of recovery of 80-100%. EMG is generally used when ENoG demonstrates absent response, since degeneration and regeneration can cause phase cancellation. Development of muscular degeneration fibrillations does not develop for 10-14 days, therefore making EMG of limited value in the early detection. However, diphasic or triphasic potentials indicate normal voluntary contraction. Polyphasic potentials indicate reinnervation, which develop 6-12 weeks before clinical return of function, which is useful in the evaluation of patients seen in the late post-traumatic period.

There is general consensus supporting the conservative treatment of patients with an incomplete paralysis. In an overview by Chang and Cass, it was concluded that surgical treatment was not required in patients who had 1) documented normal facial nerve function after injury regardless of its progression, 2) incomplete paralysis as long as there was no progression to complete paralysis, and 3) less than 95% degeneration by ENoG. Treatment of a complete paralysis is much more controversial, however. In 1974,

Fisch recommended basing the decision for surgery on the time of onset of paresis, the degree of paresis, the degree and evolution of degeneration as measured by ENoG, and the degree and evolution of regeneration. He noted a poor functional outcome in patients presenting with greater than 90% nerve degeneration by ENoG within 6 days of onset of palsy. Chang and Cass suggest that if decompression surgery is anticipated it should be done within a 14 day window from the time of injury based on animal studies by Yamamoto and Fisch.

After deciding on facial nerve exploration, the suspect location of neural injury and hearing status are the two key factors in determining an appropriate approach. Injuries of the facial nerve at or distal to the geniculate ganglion can be approached via the transmastoid procedure. Fractures can be identified laterally upon visualization of the mastoid cortex. These fractures can be chased medially to the point of injury. If there is no obvious fracture, a facial recess approach will help provide examination of the nerve from the geniculate ganglion to the second genu. Partial transections of less than 50% may be repaired with onlay nerve grafts. If transection exceeds 50%, an interposition nerve graft, such as the greater auricular nerve, should be used in approximation after the epineurium is trimmed and the nerve fascicles optimized. Of patients who undergo direct anastomosis or cable graft repair, the majority of patients (82%) will recover to a House Brackmann III or IV, and none have shown to recover to House-Brackmann I or II. If the nerve is found to be intact, decompression of the epineural sheath is performed in proximal and distal fashion until normal nerve is encountered. In Chang and Cass's review, about 50% of patients undergoing facial nerve decompression obtain excellent functional outcomes.

Injuries medial to the geniculate ganglion may be approached in several ways, depending on the status of hearing. For patients in whom hearing is not useful, a transmastoid-translabyrinthine approach is reserved. The entire intratemporal course of the facial nerve can be seen after translabyrinthine skeletonization of the internal auditory canal.

For patients with intact hearing, a transmastoid-supralabyrinthine approach or a middle cranial fossa approach is considered. Following complete mastoidectomy, the superior semicircular canal is skeletonized, thus allowing exposure of the labyrinthine portion of the facial nerve. If there are any concerns regarding adequate exposure or if grafting of the meatal portion is anticipated, the middle fossa approach is more suitable. The middle fossa approach is usually preceded by a mastoidectomy to aid in the identification of the internal auditory canal. The superior portion of the temporal bone is then exposed via an extradural craniotomy approach.

## **CSF Otorrhea**

Blunt trauma to the skull may produce fractures in the temporal bone with tearing of dura and foramina causing acute leakage. Fractures may also produce defects in the bony tegmen plate, predisposing one to encephaloceles or meningoceles with resultant delayed CSF leakage. As described previously, fractures involving the otic capsule are associated with higher incidence of CSF leak. CSF otorrhea in temporal bone fractures usually occurs within minutes of the accident but may be delayed in its presentation if it is draining through the nasopharynx. After trauma, CSF otorrhea is typically serosanguinous and can be mistaken for blood byproducts. The fluid should be sent for beta-2-transferrin, as this protein is highly specific to the CSF. Measurements of glucose and protein in the fluid have fallen out of favor for CSF identification. A high resolution CT scan can demonstrate the course of the fracture line and give information as to the likely site of CSF fistula. Contrast cisternography may increase the sensitivity of detecting CSF leak when it is active.

Management of cerebrospinal fluid leak begins with conservative measures including head elevation, bed rest with head elevation, stool softeners, avoidance of nose blowing/sneezing and other forms of straining, and, in selected patients, placement of a lumbar drain. Spontaneous resolution with this conservative management occurred in 95% to 100% of patients. In spontaneously recovering leaks, closure occurred in the first 7 days in 78%, with an additional 17% closing between 8 and 14 days. The use of prophylactic antibiotics remains controversial, although leak persists more than 7 days has been correlated with a higher incidence of meningitis. Surgical repair is recommended for those cases that persist 7-10 days after an injury. Tegmen defects may be multiple rather than single, and identifying only one defect may not be sufficient for achieving definitive repair. Because surgical repair by way of a mastoidectomy approach alone can be inadequate if there are multiple tegmen defects, a middle fossa approach alone or in combination with a transmastoid approach should be considered in most cases.

### **Vascular injury**

Carotid injury is uncommon (1-4%) in temporal bone trauma. A recent article by Dempewolf describe 44 of 127 (35%) temporal bone fracture patients had carotid canal fractures whereas only 5 of 127 (4%) had carotid artery injury. Both CT temporal bone and CT maxillofacial have been shown to be very sensitive in detecting carotid canal fracture, with a negative predictive value > 100%. CT angiography and MRA have been used more frequently than standard angiography when CT scan demonstrates evidence of vascular injury. Physical exam has not been shown to be very sensitive as only 2 of 5 patients in the Dempewolf series displayed physical findings of vascular injury (epistaxis, focal neurologic deficit).

### **Vertigo**

Vertigo after temporal bone trauma may be secondary to either vestibular concussion in OCS or vestibular destruction in OCD setting. It is usually self-limiting and resolves within 6 to 12 months from central adaptation. Perilymph fistula after otic capsule injury can also cause vertigo and SNHL. Another cause of vertigo after temporal bone fracture is posttraumatic endolymphatic hydrops. These patients present with aural fullness, tinnitus, fluctuating hearing loss, and vertigo similar to patients who have Meniere's disease. Brief vertigo episodes may be attributed to benign positional paroxysmal vertigo. It is presumably caused by traumatic displacement of otoconia from the vestibule into the ampulla of the posterior semicircular canal. Treatment of BPPV includes standard rehabilitation and repositioning maneuvers.

### **Other complications**

Some of the late rare complications after temporal bone injury include meningocele, encephalocele, meningitis, and cholesteatoma. Treatment is often surgical to prevent the development of further intracranial complications.

### **Summary**

Temporal bone fracture is a common injury in acute head trauma. Early management involves stabilization of the patient and collaboration with the trauma service. Early conservative management is recommended for hearing loss, CSF leak, and facial paresis. Long-term follow up is necessary to address hearing loss and monitor for intratemporal and intracranial complications.

## **Grand Rounds Temporal Bone Fracture – Discussion by Tomoko Makishima, MD**

Thank you, Dr. Ho. That was an excellent presentation. There's not much I need to add but I would like to comment on facial nerve injuries. Whether or not to go proceed with surgical treatment is a very controversial topic because people don't have a lot of experience in general. It is often difficult in the first place to assess whether the patient has definite indications for surgery. One of the reasons is that, in these patients, there are often other life-threatening events going on that needed to be taken care of and the diagnosis of the facial nerve injury are often delayed than you would like.

And then, another difficult decision is, what kind of surgical approach to choose. Let's say you suspect a transection of the facial nerve. One of the methods to repair it is to have the nerve ends in proximity. As long as the ends are close together, most of it will most likely re-anastomose on its own. So, those milder non life-threatening cases which go into surgical treatment of facial nerve injury, probably do not have a big gap between the transected nerve ends. And perhaps that is why there is not a lot of difference in outcome in the long term in whether you treat it surgically or not. And if you really need to do an allograft (nerve grafting) that means there is more than a few millimeter difference separation of the nerve ends – which also indicates that the temporal bone is dislocated and there is more severe events taking place in the patient, and thus will not likely be seeing us for facial nerve repair.

In terms of doing surgical decompression, my personal opinion is that I don't necessarily think that you need to go all the way to the middle fossa because the whole idea is to decompress the swelling or make some room so that the nerve ends can be close to each other, or to relieve the nerve from being stretched or pulled. Decompressing in the entire mastoid portion and tympanic segment will generate a lot of such space because you will be releasing the nerve from the surrounding bone. During my practice here at UTMB since 2005, there was only one case in which I had to do the decompression, and I only did the mastoid portion but it did turn out pretty well. This was a young boy involved in an accident – he was thrown out of a car and the car landed on his head. He had bilateral temporal bone fractures, with unilateral complete facial nerve palsy, and profound deafness on the same side.

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