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Temporal bone fracture – case report

Złamanie piramidy kości skroniowej – opis przypadku

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Keywords

temporal bone fracture, longitudinal fracture, transverse fracture, head trauma, facial nerve trauma, otorrhagia

Summary

A temporal bone is a paired bone forming a skull, together with other bones named occipital bone, frontal bone, sphenoid bone, ethmoid bone and paired parietal bones. Although it is the thickest bone of the skull, it can still get fractured, when a significant force is applied (according to one study at least 850 kg of lateral force is required for a healthy adult bone). Depending on the localization of the applied force and therefore affected planes, we can distinguish three types of fractures: longitudinal, transverse and oblique temporal bone fracture. The range of the trauma and concomitant complications should be thoroughly assessed with the help of imaging (CT, MRI) and then specialist care aimed at encountered problems administered. We would like to present a case of a 13-years-old boy who was hit by the car. After the accident he lost his consciousness and experienced post-traumatic amnesia. By admitting to the hospital, he was alert (GCS 15) and presented with otorrhagia together with a local contusion of the right occipitotemporal area. Imaging showed a temporal bone fracture.

Słowa kluczowe

złamanie kości skroniowej, złamanie podłużne, złamanie poprzeczne, uraz głowy, uraz nerwu twarzowego, krwawienie z ucha

Streszczenie

Kość skroniowa jest parzystą kością, która stanowi jeden z elementów budowy czaszki. Pozostałe kości wchodzące w jej skład to: kość potyliczna, kość czołowa, kość klinowa, kość sitowa i parzyste kości ciemieniowe. Mimo iż jest to najtwardsza kość czaszki, może ona ulec złamaniu przy zastosowaniu odpowiednio dużej siły (według jednej z analiz, dla zdrowej kości dorosłego człowieka wymagane jest co najmniej 850 kg siły bocznej). W zależności od lokalizacji i przyłożonej siły, a tym samym płaszczyzn dotkniętych urazem, możemy wyróżnić trzy typy złamań: podłużne, poprzeczne i skośne złamanie kości skroniowej. Zakres urazu i towarzyszących mu powikłań należy dokładnie ocenić za pomocą badań obrazowych (TK, MRI), by wdrożyć odpowiednią specjalistyczną opiekę ukierunkowaną na napotkane problemy. Chciałybyśmy także przedstawić przypadek 13-letniego chłopca, który został potrącony przez samochód. Po wypadku stracił przytomność i doznał amnezji pourazowej. W chwili przyjęcia do szpitala był przytomny (GCS 15) i zwracał uwagę wyciek krwisty z domieszką płynu mózgowo-rdzeniowego z prawego przewodu słuchowego zewnętrznego wraz z miejscowym stłuczeniem okolicy potyliczno-skroniowej prawej. Badania obrazowe wykazały złamanie kości skroniowej.

Introduction

A temporal bone is an element of the skull base that comes in pairs and relates to the occipital bone posteriorly, parietal bones superiorly, and the sphenoid bone anteriorly. It has a complex anatomical structure that includes four main parts: squamous, mastoid, petrous and tympanic. It possesses three bony processes: zygomatic, mastoid and styloid. Additionally, it has several foramina and canals:

- a foramen lacerum, which contains the greater petrosal nerve,
- a carotid canal, with the internal carotid artery inside,

– an internal acoustic meatus, which houses the CN VII and CN VIII nerves,
– and the jugular foramen, that contains the internal jugular vein, the posterior meningeal artery, the CN IX, CN X and CN XI nerves.

The bony labyrinth, also known as an otic capsule, is a dense part of the petrous temporal bone that surrounds the membranous labyrinth of the inner ear. This is, in turn, surrounded by the less dense and variably pneumatized petrous apex and the mastoid segment of the temporal bone. From the front to the back, the bony labyrinth contains the cochlea, the vestibule, and three semicircular canals. During trauma all those structures may be violated what may lead to specific symptoms, in various combinations, depending on the range of the trauma.

Temporal bone fractures occur when the skull experiences a strong lateral force. Such fractures are usually caused by motor vehicle accidents, assaults, falls, sports injuries, gunshot wounds, bicycle accidents, and other miscellaneous causes (1). Patients suffering from temporal bone fractures are predominantly adult males and those fractures are typically unilateral (1-3).

If someone has experienced a significant trauma to their head, particularly if it resulted in the loss of one's consciousness, it's important to consider the possibility of a cranial bone fracture. Physical examination of a person with a temporal bone fracture may reveal symptoms such as bloody or clear discharge from the ear, blood in the ear drum, dizziness, involuntary eye movements, facial paralysis, hearing loss, a wound in the ear canal, or bruising behind the ear (called the Battle's sign) (4). Concomitant other brain injuries may lead to the changes in their mental status or abnormal scores on the Glasgow coma scale.

Imaging techniques, such as computed tomography (CT) scans, are essential for the statement of the accurate diagnosis and resulting from it, proper management of the trauma. CT scans obviously have a higher sensitivity for detecting these fractures than the physical examination alone (5), but more importantly they can reveal any other associated injuries such as subdural hematoma, cerebral contusion, or subarachnoid hemorrhage. If a temporal bone fracture is identified, a high-resolution CT scan with thin slices, together with coronal and sagittal reconstructions is recommended in order to thoroughly characterize the extent of the fracture and concomitant injuries. In addition, angio-CT enables the assessment of possible vessels' injuries (like internal carotid artery or internal jugular vein injury). If there is a suspicion of a cerebrospinal fluid leak, testing the leakage for the presence of beta-2-transferrin and performing a cisternogram can help to confirm the suspicion and determine the location of the defect. Once the patient is stable, an MRI may be performed to further evaluate the brain tissue and cranial nerves, as it is more specific in the assessment of the soft tissue structures. An MRI can also differentiate between a fluid-filled mastoid and one containing herniated intracranial contents. It is also important to evaluate the cervical spine with plain film X-rays in cases of head trauma (5).

Fractures of the temporal bone can be classified based on the direction of the fissure in relation to the long axis of the pyramid. Ulrich's classification system divides temporal bone fractures into three main categories: longitudinal, transverse, and mixed types (6). Longitudinal fractures follow the length of the pyramid and constitute for the majority of all temporal bone fractures, accounting for 70-90% of cases, while transverse fractures cross the length of the pyramid and make up for a smaller portion, around 10-30%. It is rare to find a pure example of either type, as most fractures tend to have an oblique course (7). Longitudinal fractures of the temporal bone are typically caused by a blunt blow to the temporoparietal area. These fractures are usually extra-labyrinthine, meaning they do not affect the labyrinth, and thus sensorineural hearing loss is uncommon unless there is a cochlear concussion. But conductive hearing loss is more common and may be caused by ossicular disruption. In rare cases, extension of a longitudinal fracture across the sphenoid may result in bilateral injuries.

There are two subtypes of longitudinal fractures: posterior and anterior. The anterior subtype originates in the squamous temporal bone and may disrupt the middle meningeal artery, what enhances the risk of an epidural hematoma. Both posterior and anterior subtypes tend to affect the first genu or the proximal tympanic segment of the facial nerve, but facial palsy is less common and less severe in cases of the longitudinal fractures compared with the transverse fractures (fig. 1) (8).

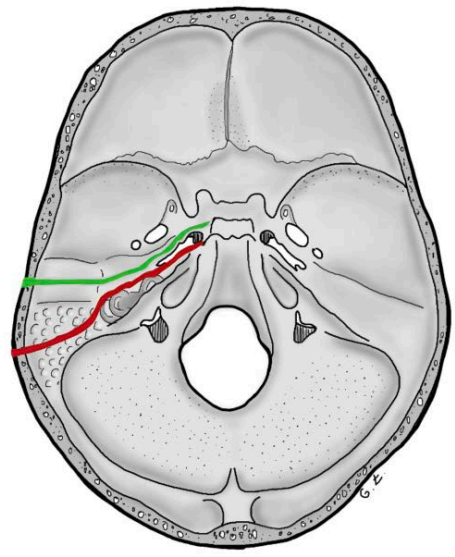

Fig. 1. Longitudinal temporal bone fracture runs parallel to the long axis of the petrous pyramid inside the middle cranial fossa. The force of the fracture approaches the vertical (sagittal) plane (author's own scheme). Green line – anterior subtype; red line – posterior subtype

On the contrary, transverse fractures are usually caused by a blunt force to the occipital area, with the point of entry often close to the vestibular aqueduct. These fractures are divided into medial and lateral subtypes. The medial subtype of the transverse fracture goes through the fundus of the internal auditory canal and can cause complete and permanent sensorineural hearing loss due to the cochlear nerve transection. The lateral subtype goes through the bony labyrinth rather than the fundus of the internal auditory canal, leading to the sensorineural hearing loss and a potential perilymphatic fistula (due to the communication between the middle and the inner ear) (fig. 2) (8).

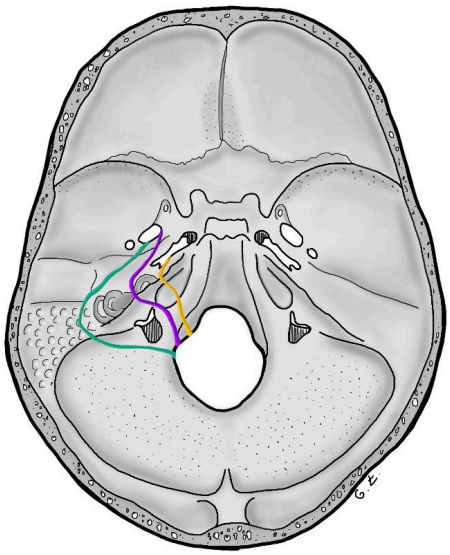

Fig. 2. Transverse temporal bone fracture runs perpendicularly to the long axis of the petrous pyramid, from the foramen magnum to the middle fossa. The force of the fracture approaches the coronal plane that is vertical to the lateral part of the temporal bone. Colored lines mark possible courses of the fracture: yellow line – medial subtype, going through the fundus of the internal auditory canal, at the same time sparing the bony labyrinth; violet line – lateral subtype going directly through the bony labyrinth (the most common subtype); green line – lateral subtype, otic capsule – sparing (author's own scheme)

Many temporal bone fractures exhibit characteristics of both longitudinal and transverse fractures and are classified as a mixed type (also called an oblique fracture). The aforementioned classification of the temporal bone fractures does not accurately predict the risk of otoneurologic complications or facilitates the choice of surgical access route, if needed (fig. 3) (8).

In contrast, the Kelly and Tami classification system is more anatomically focused and concentrates on the integrity of the bony labyrinth (otic capsule), rather than the direction of the fracture's fissure. This classification aims to improve the clinical correlation between the fracture's geometry and the clinical outcomes by dividing temporal

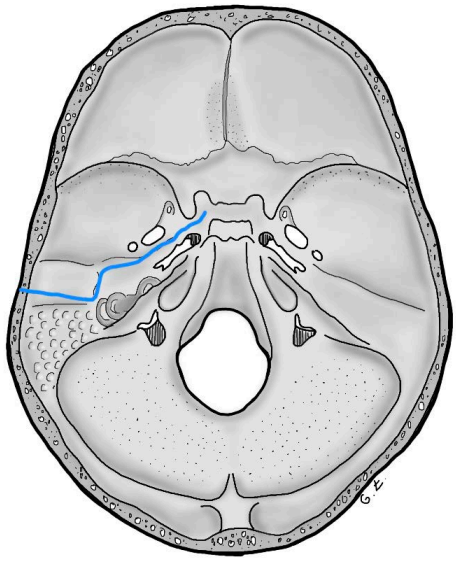

Fig. 3. Mixed temporal bone fracture involves the external auditory canal laterally, anteromedially extends to the skull base, but avoids the labyrinthine structures. The force of the fracture approaches the horizontal (vertical) plane (author's own scheme)

bone injuries into bony labyrinth sparing or violating fractures (9). Temporal bone fractures that do not involve the bony labyrinth, typically affect the squamous part of the temporal bone and external auditory canal, starting from the mastoid air cells, through the middle ear, to the tegmen tympani. These fractures tend to have lower rates of cranial nerve VII paralysis, but higher rates of mixed or conductive hearing loss due to hemotympanum or, less commonly, ossicular chain disruption. On the other hand, temporal bone fractures that disrupt the bony labyrinth have a higher association with cerebrospinal fluid leak, sensorineural hearing loss, and probable intracranial pathology (10). These fractures usually pass through the foramen magnum to the petrous pyramid and then the bony labyrinth. They are usually caused by a blow to the occipital region. In most cases they result in sensorineural hearing loss as they rarely affect the ossicular chain or the external auditory canal (1).

Additional investigations, which help with the comprehensive assessment of the extent and consequences of a temporal bone fracture, may include tympanometry with acoustic reflex testing, tonal audiometry, auditory brainstem response testing (ABR), vestibular testing for vertigo or nystagmus, as well as nerve conduction studies for facial nerve paresis or paralysis (electroneuronography).

The treatment and management of any potentially life-threatening intracranial injuries and complications should be the priority. Once the patient is hemodynamically and respiratory stable, the management of further complications related to the temporal bone fracture can be addressed.

We analyzed articles available in the published literature dealing with the problem of a temporal bone fracture. The most important issues concerning the diagnosis,

differentiation, and treatment of different types of fractures are gathered and discussed below. We would also like to present a clinical case of a child with a temporal bone fracture after a traffic accident as an illustration of the problem.

Case report

A 13-years-old boy was admitted to the Emergency Department of the University Clinical Hospital in Warsaw after an accident, in which as a pedestrian he was hit by the car. He lost his consciousness right after the accident and experienced post-traumatic amnesia. On admission he received 15 points in Glasgow Coma Scale (GCS). Clinical examination revealed otorrhagia, local contusion of the right occipito-temporal area and minor abrasions of the epidermis in the right lateral ankle and the left sacral areas. He reported dizziness while changing his position. Trauma CT scan was performed. Imaging showed fracture of the squamous part of the occipital bone along the lambdoid suture with dislocation, fracture of the squamous part and the pyramid of the right temporal bone, involving the mastoid process, walls of the tympanic cavity, the external auditory canal, and the socket of temporomandibular joint, without dislocation (fig. 4-5a, b).

There were also described blood in the right mastoid process and in the right tympanic cavity (called hemotympanum), an extra-cranial hematoma in the right temporal region. No destruction of the inner ear was detected. The neurological examination did not reveal any abnormalities (fig. 6a, b).

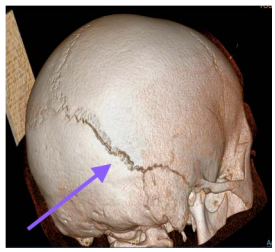

Fig. 4. CT scan, 3D-reconstruction: fracture of the squamous part of the occipital bone along the lambdoid suture with dislocation (own clinical material)

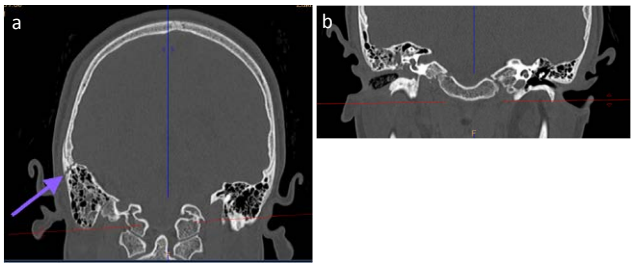

Fig. 5a, b. CT scan, coronal plane: fracture of the squamous part (a) and the pyramid of the right temporal bone involving: mastoid process, walls of the tympanic cavity and external ear canal (b). As a result, the right tympanic cavity (called hemotympanum; b) and right mastoid air cells are filled with blood (radiologically seen as impaired aeration of the mastoid process; a, b) (own clinical material)

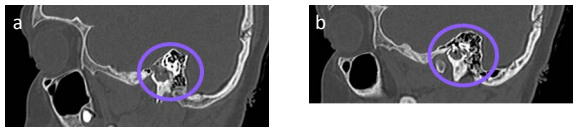

Fig. 6a, b. CT scan, sagittal plane: no destruction of the inner ear (a) and no dislocation of the ossicles, blood in the tympanic cavity (hemotympanum; b) (own clinical material)

Otolaryngological examination revealed bloody discharge from the right external ear canal. External ear canal was cleaned from cloths with suction, but some fresh discharge was still present. Canal walls were edematous and reddened, what impaired thorough examination of the tympanic membrane (fig. 7). A sterile dressing was applied. Left ear examination showed no abnormalities (fig. 8). The external ear canal was daily cleaned at the bedside, with exchange of the dressings, as long as the leakage was persistent. Bloody discharge ceased after 2 days and

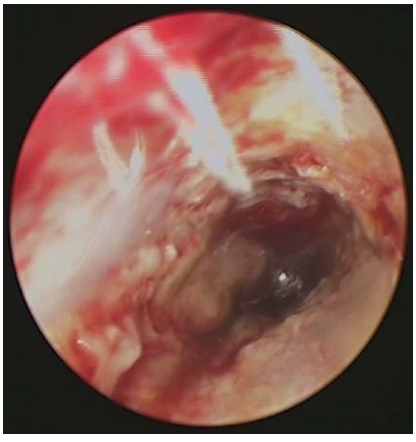

Fig. 7. Right ear, otoscopy: clinical presentation few days after trauma – narrowed ear canal due to oedema, with reddened walls and exfoliating epidermis, scar on the inferior wall corresponding the fracture line of the temporal bone, tympanic membrane partially visible, with suspected hemotympanum (own clinical material)

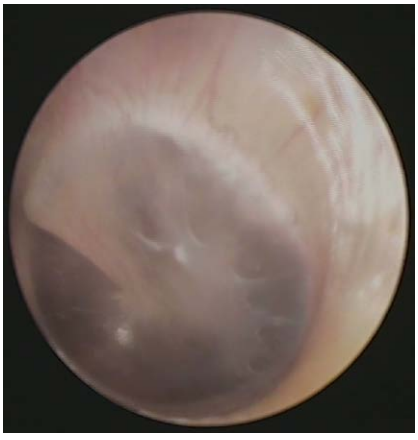

Fig. 8. Left ear, otoscopy: normal tympanic membrane (own clinical material)

hemotympanum in otoscopic examination was suspected. However, for the next few days transparent fluid persisted, what was interpreted as a CSF leak, fortunately, with a tendency do diminish. During Valsalva maneuver there was a serous-bloody discharge from the middle ear, what confirmed patent tympanic membrane perforation and the presence of hemotympanum (fig. 9, 10). Tympanometry showed a B-type curve, which confirmed the presence of fluid in the middle ear. CSF leak fully ceased one week after the accident.

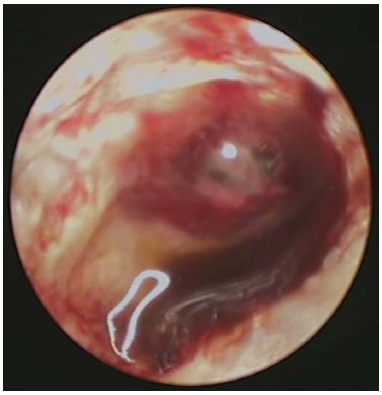

Fig. 9. Otoscopic examination of the right ear during Valsalva maneuver. The serous-bloody leakage confirmed patient's tympanic membrane perforation (own clinical material)

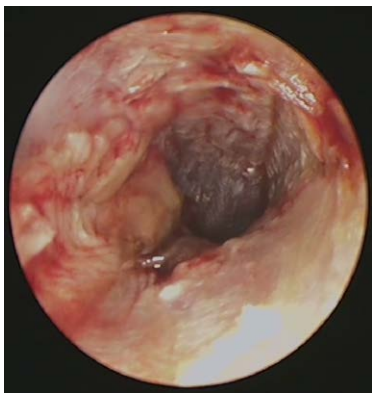

Fig. 10. Otoscopic examination of the right ear after thorough suction (under microscopic control). Narrowed, edematous, inflammatory ear canal with a scar on the bottom wall indicating the location of the fracture, thickened tympanic membrane. Gradual improvement when compared with previous examination (own clinical material)

Conservative neurosurgical treatment in a form of immobilization, observation, and adequate fluid control was recommended. He received a wide range antibiotic prophylaxis to prevent meningitis- ceftriaxone intravenously for 14 days, followed by amoxicillin with clavulanic acid orally for the next 14 days at home.

On the fourth day of hospitalization a control CT scan was performed, and a suspicion of cerebral venous thrombosis was aroused. Angio-MRI confirmed partial thrombosis of the right transverse and sigmoid sinuses. That is why, after hematological consultation, 40 mg of heparin daily was administered.

Within the time of hospitalization patient was progressively mobilized. His recovery proceeded without any further complications. There were no abnormalities in the neurological examination. External ear canal healed gradually; tympanic membrane perforation closed successfully (fig. 11, 12). He was discharged home after 2 weeks of hospitalization with the recommendation of resting lifestyle. He was controlled otolaryngologically, neurologically and hematologically in our outpatient clinic. Audiological examinations in a form of tympanometry and tonal audiometry were performed 1 month and 3.5 months after trauma. The results 1 month after trauma confirmed successful closure of the tympanic membrane perforation and good mobility of the tympanic membrane (fig. 13). However, slight conductive hearing loss on the right side was observed (fig. 14), but it returned to normal 3.5 months after trauma (fig. 15). Otoscopic examination was unremarkable (fig. 16).

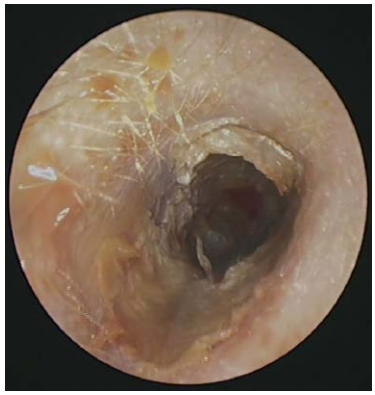

Fig. 11. Otoscopic examination of the right ear, 10th day after trauma (own clinical material)

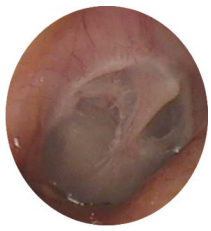

Fig. 12. Otoscopic examination of the right tympanic membrane, 3.5 months after trauma – complete recovery (own clinical material)

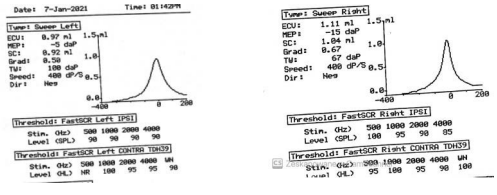

Fig. 13. Tympanometry results 1 month after trauma: tympanogram type A bilaterally (normal results) (own clinical material)

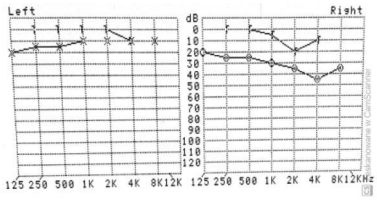

Fig. 14. Tonal audiometry results 1 month after trauma: slight conductive hearing loss on the right side (own clinical material)

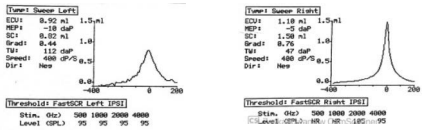

Fig. 15. Tympanometry results 3.5 months after trauma: tympanogram type A on the left side and Ad on the right side (normal results) (own clinical material)

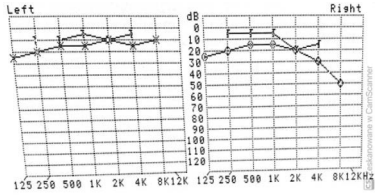

Fig. 16. Tonal audiometry results 3.5 months after trauma: earlier observed air-bone gap returned to normal, slight hearing loss on higher frequencies (4 kHz, 8 kHz) resided (own clinical material)

Discussion

Literature review shows that 31% of temporal bone fractures in the general population were caused by traffic accidents followed by assaults and falls. Other injuries, such as bicycle accidents, pedestrian injuries, gunshot wounds, off-road vehicle accidents, sports injuries, accounted for 25% of all cases (11). In children's population the most common causes of temporal bone fractures were road traffic accidents and falls, each accounting for 30 to 50% of cases (12-16). It is important to note, that men are three to four times more vulnerable to trauma than women (11, 17) and that bilateral temporal bone fractures occurred in 8 to 29% of patients (11, 17-19).

Structures, which are at risk during temporal bone fracture are either localized within the bone itself or in its direct surrounding. These include facial nerve, cochlea, labyrinth, ossicles, tympanic membrane, auricle, external auditory canal (EAC), carotid artery, jugular vein, dura, and inferior cranial nerves (20). Overall, the prognosis for a patient with temporal bone fractures depends on whether other associated brain injuries are detected.

Facial nerve injury is a common complication after trauma of the middle cranial fossa and results in easily observed abnormalities of the face expression, aggravated by its movements. Patients have a 6-7% risk of facial paralysis, of which 25% is acute and complete paralysis, and 75% is partial/incomplete paralysis (11, 17). In pediatric population, the incidence of facial nerve injuries is 3-9% (12, 15, 16). About 27% of patients with temporal bone fractures presented with complete facial paralysis initially. In other words, an immediate onset must be assumed. Other 73% had restricted facial movements at the time of initial assessment, which may worsen over 1 to 16 days, if not properly detected and treated (11). High-resolution computed tomography (HRCT) of the head remains the gold standard for assessment of head trauma with facial nerve injuries, enabling thorough investigation of bone structures (also the bony canal of the facial nerve). However, an MRI remains a dedicated examination for the soft tissues' injuries. Surgical exploration, and decompression or less often surgical end-to-end suturing may be necessary for an immediate paralysis or significant degeneration of the facial nerve (> 90%, measured on EnOG, electroneuronography). Such explorative surgeries often reveal bony impingement, stretch injury, or edema/hematoma rather than the direct transection of the nerve. Delayed onset paralysis may be caused by neural oedema or compression by hematoma. They can usually be satisfactorily treated with a course of intravenous corticosteroids for 1 to 3 weeks, combined with rehabilitation. Serial EnOG may be performed, and surgery should be considered if the degeneration reaches or exceeds 90% (1).

Cerebrospinal fluid (CSF) leakage occurs in 17% of temporal bone fractures and is one of the most serious complications due to the risk of meningitis (11). In the bony labyrinth sparing fractures, CSF normally leaks through a fracture of the tegmen tympani or mastoid into the epitympanum, antrum, and mastoid air cell tract. It may present as clear otorrhea if the tympanic membrane is ruptured or as rhinorrhea if the tympanic membrane is unaffected. In the bony labyrinth violating fractures, CSF moves from the posterior fossa through the bony labyrinth into the middle ear (4). These fractures are exceptional as they never fully heal spontaneously. The size of the bony labyrinth remains the same at birth and in the adulthood, throughout life it undergoes only minimal remodeling (21). That's why in the process of recovery of such fractures only a thin fibrous scar is developed, with no subsequent endochondral ossification. Therefore, the patient remains susceptible to meningitis for an extended period of time after injury, possibly for the entire life (22, 23). CSF leaks in these patients are usually evident at the time of presentation, sometimes they may develop later. HRCT shows a bony defect responsible for a CSF fistula site in 70% of patients (24). Laboratory evaluations can also be helpful in the differential diagnosis, when we observe otorrhea, rhinorrhea, or otorhinorhoea. ß2-Transferrin is an isoform of transferrin that is found only in CSF, perilymph and aqueous humor (25). This protein can be found even in small volumes of CSF with high sensitivity and specificity, and therefore it can be crucial for CSF leak diagnosis.

The treatment of the cerebrospinal fluid leak should include conservative measures such as elevating the head,

bed rest, and avoidance of straining. Wide range antibiotic prophylaxis is a standard element of most skull base injuries, especially with associated CSF leak. Most CSF leakages usually tend to close spontaneously. If conservative measures are unsuccessful, a lumbar drain may be placed to reduce intracranial pressure. In a small number of cases, when CSF leak persists for more than 7 to 10 days, surgical repair may be necessary (1, 20).

Auditory dysfunction has long been recognized as a potential consequence of a head injury and the temporal bone fracture. It has been reported to occur in about 30-70% cases of patients with skull base fractures or with head trauma history (26). Temporary conductive hearing loss is caused by initial hemotympanum or effusion, while permanent deficits are caused by disruption of the ossicular chain, which occurs in approximately 20% of patients (27). The most common injuries of the ossicular chain include subluxation of the incudostapedial joint (82%), incus dislocation (57%) and stapes fracture (30%) (28). One-third of patients experience multiple middle ear injuries due to the trauma. Among cases of temporal bone fractures, damage to the middle ear or sensory neuroepithelium of the inner ear may be the cause of sensorineural hearing loss. Those hearing disorders can occur in both bony labyrinth sparing and bony labyrinth violating fractures, as well as in cases of head trauma, not directly related to a temporal bone fracture; the hearing loss can progress with time (29). There are different mechanisms that can lead to sensorineural hearing loss, like disruption of the membranous labyrinth, avulsion or trauma to the cochlear nerve, disruption of the cochlear blood supply, bleeding into the cochlea, perilymphatic fistula. Another described mechanism is endolymphatic hydrops, which results from the obstruction of the endolymphatic duct by the bones disrupted due to fracture (30). Complex and thorough audiological evaluation should be delayed until a few weeks after the injury to ensure that oedema and blood in the eardrum have fully resolved. If the hearing loss persists and is conductive in nature, ossiculoplasty (ossicular chain reconstruction) may be required. If the hearing loss persists and is sensorineural, treatment options such as hearing aids or a cochlear implant should be considered, depending on the severity of the loss (1).

It can be challenging to anticipate whether a person will experience permanent hearing problems when there are not any abnormal findings on radiological studies. What is more, some hearing disturbances are the result of a local trauma and inflammatory (post-traumatic) changes. The terms “labyrinthine concussion” and “inner ear concussion” have evolved to mean auditory and/or vestibular dysfunction of the inner ear following head trauma in the absence of the evident fracture. Temporal bone concussion is more common in patients with head trauma caused by assaults or sports’ injuries than temporal bone fractures. The most frequently described mechanisms of labyrinthine concussion in literature included: labyrinthine hemorrhage, cochleovestibular nerve traction injury or direct damage due to the labyrinthine fluid wave (31, 32). There is no specific treatment for labyrinthine concussion. The use of corticosteroids is controversial, and most cases are managed individually, according to the observed symptoms (33).

Vertigo is also considered as a possible consequence of temporal bone fracture. Usually, it occurs as a benign paroxysmal positional vertigo (BPPV). BPPV occurs in 4.1-14.9% of head traumas, but some argue that it may appear more frequently after temporal bone fracture (34). It is caused by the damage of the otolith organ which is localized in the temporal bone. Post-traumatic BPPV has several peculiarities, such as a high incidence of bilaterality, involvement of multiple semicircular canals, comparable gender occurrence, frequent occurrence among young people, more treatment difficulties, and frequent recurrences (35). The results of studies show that the treatment of post-traumatic BPPV in not as beneficial as idiopathic one’s. It requires more frequent canalith repositioning procedures (CRPs) and tends to reoccur (36).

Also, post-traumatic Meniere’s disease (PTMD) was reported in the literature. Usually, full-blown Meniere syndrome with unilateral tinnitus, feeling of fullness in the ear, hearing loss, and recurring episodic vertigo lasting at least 20 min develops many years after trauma (up to 20). However, up to 30% can occur in first month (37). PTMD is treated as a Meniere’s disease, starting with preventive measures (no rapid head movements, low-salt diet etc.). Medication such as diuretics, steroids, betahistine hydrochloride, can be administered in cases with severe vertigo. If such treatment is not beneficial, intratympanic gentamycin injection or surgical treatment, such as ELSD, labyrinthectomy, cochleosacculotomy or vestibular nerve dissection should be considered (38).

Temporal bone fracture can cause both immediate and time-delayed complications. Therefore, specialist and extensive post-accident care is required. Patient in our case presented with CSF leakage, hearing loss and vertigo. All detected complications appeared shortly after the accident and gradually receded over time. He got wide range antibiotic prophylaxis to prevent meningitis. As for CSF leakage regular suction and sterile dressings were applied. Dizziness subsided during hospitalization; hearing returned to normal after 3.5 months. Our patient hasn’t developed any delayed in time complications yet. He remains under ambulatory control.

Conclusions

Temporal bone is a complex structure located on the sides of the skull base and protects several important structures, including the middle and inner ear, the facial nerve as well as other nerve structures. Temporal bone fractures are a type of head injury that results from a high-force trauma, such as a fall or a motor vehicle accident, when an immense amount of lateral force results in fracture.

Therefore, temporal bone fractures are often associated with significant general trauma and can lead to serious complications. Such patients definitely require multi-specialist care. When evaluating a patient with a suspected temporal bone fracture, healthcare providers should look for hemorrhagic or clear otorrhea, hemotympanum, hearing loss, vertigo, nystagmus, facial paralysis, and/or Battle's sign. Confirmed fractures should be classified as either bony labyrinth sparing or violating, with violation being more likely to cause SNHL, perilymphatic fistula, facial nerve paralysis, and most often requiring surgical intervention. High-resolution non-contrast CT with slice thickness equal or less than 1.5 mm is the modality of choice for the evaluation of temporal bone fractures. It is also important to look for associated intracranial and cervical pathologies. Once stabilized, temporal bone fracture complications can be treated. Complications may include facial nerve paralysis, CSF leak, hearing loss, and vertigo. Hearing loss requires differentiation of its origin (conductive versus sensorineural hearing loss), with audiological examinations being helpful in assessing its grade, and then monitoring the healing processes or effects of our procedures. Facial nerve paralysis assessment should include an EnOG and EMG, sometimes surgical procedures are required. CSF fistulas usually resolve spontaneously but may require lumbar drainage or surgical repair if persistent.

Conflict of interest
Konflikt interesów

None
Brak konfliktu interesów

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