Management of temporal bone trauma – literature revision and algorithm proposal

Original Article

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Abstract

Introduction: Temporal bone trauma frequently occurs in the context of high-kinetic-energy trauma, often associated with multiple bodily injuries.
Objective: To present a brief literature review on temporal bone trauma and propose a protocol for approaching these situations in a tertiary hospital.
Description: Initial assessment following high-kinetic-energy trauma requires a summary ABCDE observation to ensure hemodynamic stability. Following this phase, in cases suspected of temporal bone involvement, an otolaryngology evaluation is recommended, to search for signs of hearing loss, vertigo and/or facial mimetic muscle impairment.
The proposed algorithm guides the approach to temporal bone trauma.
Conclusion: Temporal bone trauma requires otolaryngological evaluation to exclude entities that may require urgent surgical care. The use of therapeutic algorithms aims to optimize the quality of care with appropriate team training.

Keywords: trauma; temporal bone; management

Introduction

Temporal bone trauma is frequently associated with injuries to other parts of the body and occurs in approximately 20% of cases involving high-energy kinetic impact[1–3]. It typically results from blunt, unilateral trauma and may be accompanied by varying degrees of intracranial injury [4]. The leading cause is traffic accidents, particularly those involving two-wheeled vehicles, followed by gunshot wounds and falls[1]. These injuries are more prevalent in young men.

While the mortality associated with temporal bone trauma must not be underestimated, morbidity is considerably more widespread and often debilitating, leading to a substantial healthcare burden and prolonged work

absenteeism. Therefore, the management of these patients should be multidisciplinary, with otolaryngology playing a central role. Evaluation should occur in the emergency setting, guided by optimized, protocol-based strategies to facilitate the identification of cases requiring immediate intervention or close monitoring. In this study, we present a brief literature review on temporal bone trauma and its complications. Additionally, we propose a standardized protocol for the management of these injuries in a tertiary hospital setting.

Materials and methods

We conducted a literature review, along with an adaptation of the published algorithms.

A bibliographic search was conducted on the PubMed platform using the following keywords: *"trauma"* AND *"temporal bone"*; *"management"*; *"temporal fracture"*; *"temporal bone trauma algorithm"*; *"temporal bone trauma protocol"*. Subsequently, this was supplemented with a more detailed ancillary search.

For the development of the protocol, review articles and existing protocols implemented in other countries, published between 2000 and 2025, were included; all other publications were excluded. These protocols were translated into European Portuguese and adapted accordingly.

Results

The initial search identified approximately 12 studies, which were then expanded with additional data. The first assessment of a patient with high-energy kinetic trauma follows the Airway, Breathing, Circulation, Disability, and Exposure (**ABCDE**) approach for emergency evaluation[5], which focuses on hemodynamic stability and excludes life-threatening complications. Subsequently, imaging studies are typically requested to support a diagnosis or exclude complications, including computed tomography (CT) of the head. When there is a clinical and/or radiological suspicion of temporal bone involvement, **high-resolution temporal bone CT** (slice thickness < 1 mm)[6] is the preferred imaging modality. However, its availability in emergency settings may be limited[7]. In such cases, an otorhinolaryngological examination is recommended[1]. The time and place of this evaluation depend on the patient's clinical stability. In conscious and hemodynamically stable patients, a detailed history, otoscopic examination, tuning fork tests, facial mimicry assessment, and brief neurological examination can help in identifying signs and symptoms that can be grouped into distinct clinical syndromes, each requiring a specific approach and treatment. Ideally, these assessments should be performed prior to the administration of muscle relaxants, as they can impair an accurate clinical evaluation. Based on the initial otorhinolaryngological assessment, eight possible scenarios are outlined below, each with its corresponding management protocol.

1. Auricular hematoma

Acute auricular hematoma is a common external ear injury that results from direct blunt trauma to the auricular pavilion with accumulation of blood between the perichondrium and cartilage. The skin of the anterior auricular surface is adherent to the cartilage, predisposing this area to hematoma formation, often near the scaphoid fossa[8].

If not adequately drained, the hematoma may become secondarily infected, leading to cartilage destruction and the typical cauliflower ear deformity. Recommended management involves **aspiration or simple incision for drainage**, preferably at the level of the antihelix, followed by simple horizontal through-and-through sutures across the auricle. Reinforcement with absorbable dressing material may be performed. A compressive dressing is recommended, and sutures should be removed within 7–10 days[8]. Although scientific evidence is limited, **oral antibiotic therapy** targeting *Pseudomonas aeruginosa* and the skin flora (e.g., ciprofloxacin) is advised for 7–10 days[8,9].

2. Severe trauma to the external auditory canal

Injury to the external auditory canal may occur in the context of mandibular or temporal bone trauma, particularly involving the condyle. Diagnosis relies on careful physical examination, together with targeted imaging. Treatment commonly includes **ear packing with non-absorbable material and topical antibiotic therapy**[10]. Close follow-up is essential to monitor for complications such as canal stenosis or cholesteatoma and to guide further management.

3. Bloody otorrhea

Isolated bloody otorrhea, in the absence of other signs or symptoms, is often due to superficial disruption of the external auditory canal skin. When possible, microscopic ear aspiration is recommended to facilitate local cleaning and identify the bleeding source. For pedagogical purposes, this section refers specifically to cases where the otoscopy is normal. In mild cases, management includes **microscopic aspiration** (if possible), **topical antibiotic therapy** (preferably quinolones), and **clinical surveillance**. In cases of more profuse bleeding, **ear packing with non-absorbable material** (e.g., *Oto-wick*®) should be performed prior to initiating topical antibiotics.

4. Conductive hearing loss

This is the most common type of hearing loss associated with temporal bone trauma. The primary causes are hemotympanum, tympanic membrane perforation, and ossicular chain disruption[1,11,12]. Although these conditions may coexist, they are discussed individually for clarity. In cases of hemotympanum, blood accumulation in the middle ear increases impedance, leading to conductive hearing loss with an air-bone gap (ABG) of 10–30 dB.

In cases of **extensive** hemotympanum, confirmed by otoscopy and exhibiting generalized tympanic cavity filling on imaging, **prophylactic oral antibiotic therapy**, preferably with a beta-lactam agent, is recommended[13]. Surgical intervention alone is generally not indicated[14]. Reassessment with otoscopy and audiological tests (tympanometry and pure-tone audiometry) is recommended within 3–4 weeks (up to 8 weeks)[14]. In cases of tympanic perforation, initial management includes **topical antibiotic therapy** (quinolones) and **audiometric evaluation approximately three months** post-injury[1,3,12,15]. Follow-up otoscopy is essential to assess perforation closure. Perforations typically affect hearing at 0.5, 1, and 2 kHz, with ABG values ranging from 10–40 dB depending on the perforation size[7,10]. Perforations involving less than 25% of the tympanic membrane often heal with conservative treatment, while larger defects may require surgical repair[16]. Finally, in patients with conductive hearing loss and normal otoscopy, **outpatient reassessment with audiometry three months** post-trauma is recommended to evaluate the ossicular integrity. The most common ossicular injury is **incudostapedial joint dislocation**[17]. For persistent conductive hearing loss with ABG > 35 dB lasting for over two months, exploratory tympanotomy may be considered, with possible ossiculoplasty. However, in patients with unilateral hearing loss, surgical intervention is contraindicated [7,12].

5. Sensorineural hearing loss

In the context of trauma, sensorineural hearing loss typically affects higher frequencies, particularly between 4–8 kHz[16,17]. In the initial assessment, audiometry and evaluation of spontaneous nystagmus can aid clinical reasoning. However, a definitive diagnosis of hearing loss requires a pure-tone audiogram.

At the bedside, the presence of **acoumetry findings consistent with fluctuating or stable sensorineural hearing loss combined with spontaneous nystagmus** should raise suspicion of a **perilymphatic fistula**. These cases may also present with mixed hearing loss. A perilymphatic fistula may result from a fracture of the otic capsule (with or without pneumolabyrinth) or from stapes subluxation into the oval window[1,3,12,15]. Diagnosis is based

on correlation between **clinical and imaging findings**. Complementary tests include urgent audiometry, vestibular testing when feasible, and high-resolution imaging[18,19]. For patients with mild symptoms, clinical monitoring is recommended, including bed rest, head-of-bed elevation, and avoidance of activities that may increase the intracranial or inner ear pressure[18,19]. This approach is supported by the high likelihood of spontaneous resolution.

In cases of debilitating symptoms with progressive sensorineural hearing loss, surgical exploration is indicated to identify the site and close the defect. Various autologous grafts have shown effectiveness in defect closure, including fat, temporalis fascia, and tragal perichondrium, the latter offering the greatest resistance[18,19]. If acoumetry raises the suspicion of isolated sensorineural hearing loss, audiometry should be performed as soon as the patient can cooperate. Empirical corticosteroid therapy is recommended beforehand, including prednisolone at 1 mg/kg/day, tapered by ~20 mg every three days[12].

Persistent hearing loss may require auditory rehabilitation. Prognosis worsens with increasing severity of the hearing deficit.

6. Cerebrospinal fluid otorrhea

Cerebrospinal fluid (CSF) leaks can occur in approximately 20% of temporal bone fractures and typically present within 48 hours post-trauma. They should be suspected in cases of rhinorrhea or otorrhea that worsens with forward bending of the trunk or during the Valsalva maneuver[1,12,15,20]. The transudate should be collected in a sterile container and sent for laboratory analysis. The gold standard test is **β2-transferrin**[1,12,15,20], which is a[21]glycoprotein found in the CSF, perilymph, aqueous humor, and vitreous humor[17]. Its presence in the nasal or middle ear secretions is highly suggestive of a CSF leak [22]. If β2-transferrin testing is not immediately available, preliminary screening for glucose, protein, and potassium can be performed. High glucose (approximately 2/3 of the serum levels) and low protein and potassium levels suggest a CSF leak and require confirmatory β2-transferrin testing[22.] The test requires a minimal volume (as little as 2 µL) and involves immunofixation, isoelectric focusing, gel electrophoresis, and immunoblotting[22,23]. Samples should be analyzed within 14 days[24]. β2-transferrin testing has high sensitivity and specificity, with false negatives typically occurring when the residual fluid volume is insufficient [22,23,25].

Another diagnostic option is the detection of **β-trace protein**, a prostaglandin D synthase produced by the choroid plexus and meninges[26]. Similar to β2-transferrin, β -trace protein can also be detected in the aqueous humor, perilymph, urine, and plasma. It is abundant in the CSF and, due to its high CSF-to-plasma ratio, serves as a reliable marker of CSF leakage. However, its detection is contraindicated in conditions associated with elevated serum proteins, such as renal failure or bacterial meningitis [22,26].

β-trace protein testing is cost-effective, requires a small sample volume (200–400 µL), and offers greater sensitivity and specificity than β2-transferrin testing, although its availability may be limited[27]. In patients with inconclusive results and strong clinical suspicion, calculating the CSF-to-plasma ratio is recommended, as elevated values support the diagnosis of a CSF leak.

Small defects can often be managed with **rest**, head-of-bed elevation, hydration, use of laxatives, and avoidance of nose-blowing or sneezing. Resolution typically occurs within 7–10 days [6,10]. Topical prophylactic antibiotics may be used[11], but oral antibiotic prophylaxis is not advised, as it may obscure signs of meningitis[12]. In cases of persistent leakage, **large-volume** otorrhea, or post-traumatic encephalocele, lumbar drainage and eventual **surgical repair** of the defect may be necessary.

7. Post-traumatic encephalocele

Post-traumatic encephalocele may occur following disruption of the middle fossa floor, leading to herniation of the dura mater and temporal lobe into the middle ear. It can be detected on otoscopic examination as a

whitish, vascularized mass, often pulsatile, located behind the tympanic membrane. Diagnosis is confirmed with imaging and [1,12,15,20] the mass size may increase during the Valsalva maneuver. Management is **surgical**.

8. Peripheral facial paralysis (PFP)

Facial nerve injury is most commonly associated with temporal bone fractures but may also result from direct penetrating or blunt trauma. Initial evaluation should look for the signs of facial nerve involvement.

Temporal bone fractures are classified according to the involvement of the otic capsule. The majority spare the otic capsule and are associated with a lower incidence of facial nerve injury (approximately 10%). However, in rare cases where the otic capsule is involved, the incidence of PFP may reach up to 50%[28]. **Immediate** (or acute) PFP, defined as onset within 24 hours of trauma, is typically caused by nerve transection (**neurotmesis**) and usually presents as complete paralysis (House-Brackmann grade VI)[12,15,29,30]. These cases often have fracture lines in the otic capsule and may also be associated with involvement of the vestibulocochlear nerve [31,32]. This type of fracture results from occipital trauma, with the fracture line extending from the foramen magnum (or jugular foramen) to the foramen lacerum and traversing the optic capsule and geniculate ganglion[31,32]. Delayed **(or late-onset)** PFP typically occurs between 1 and 16 days post-trauma and is frequently caused by **edema or hematoma**. It may present as either partial or complete paralysis, with partial PFP due to edema being the most frequently observed type[12,15,29,30]. Although no randomized controlled trials have established the definitive benefits of corticosteroid therapy, its use is widely recommended for both complete and partial delayed PFP. The suggested regimen is prednisolone 1 mg/kg/day, tapered by 20 mg every three days[15].

Management of traumatic PFP depends on the decision whether to proceed with surgical intervention, which is based on the assessment of nerve continuity and likelihood of a favorable prognosis. Electrophysiological evaluation of the facial nerve is indicated in the following cases:

a) Evidence of complete and immediate PFP within 3–7 days post-injury.

b) Evidence of complete and delayed PFP following an unsuccessful therapeutic trial of corticosteroid therapy.

The selection of suitable electrodiagnostic test depends on the duration of nerve degeneration. In scenario **a), nerve excitability testing (NET) or electroneurography (ENoG)** is recommended, with ENoG being the most widely used test currently[1,5,6,10,15,33]. Patients presenting with **NET thresholds ≥ 3.5 mA** or **ENoG demonstrating > 90% nerve degeneration** are considered to have a **poor prognosis**[30,34]and may benefit from surgical decompression[2,12]. It is important to emphasize that these tests do not differentiate between axonotmesis and neurotmesis, are not indicated in cases of partial paralysis, and may yield high false-negative rates if performed within 72 hours of injury due to the effects of Wallerian degeneration[30].

In scenario **b), electromyography (EMG)** is the preferred method[30]. EMG is the only electrophysiological test recommended after complete nerve degeneration. Between 10 and 14 days after the onset of PFP, EMG can identify **fibrillation potentials**, which indicate **nerve degeneration**, as well as **polyphasic reinnervation potentials**, which suggest a **favorable prognosis**. These findings are useful for follow up 4–6 weeks after symptom onset[30].

In summary, **surgical intervention** is indicated when electrophysiological testing reveals **poor prognostic markers** or if CT imaging shows **evidence of nerve transection.**

After making the decision for surgical intervention, the approach and timing must be determined. The choice of surgical approach depends on whether the otic capsule is involved, which is linked to the hearing function. Surgery should be performed as early as possible, although the optimal timing remains debatable in the

literature and often depends on the surgeon's experience[15]. The surgical approach should expose the perigeniculate region and/or the mastoid segment, the two most commonly affected sites of facial nerve injury. According to the literature, a translabyrinthine approach is recommended for fractures involving the otic capsule, whereas a combined approach (transmastoid and middle cranial fossa) is preferred when the otic capsule is intact[15]. The primary goal of surgery is facial nerve decompression, either by removing bony fragments from the fracture site or by performing epineural drainage in cases where an intraneural hematoma is identified [35,36].

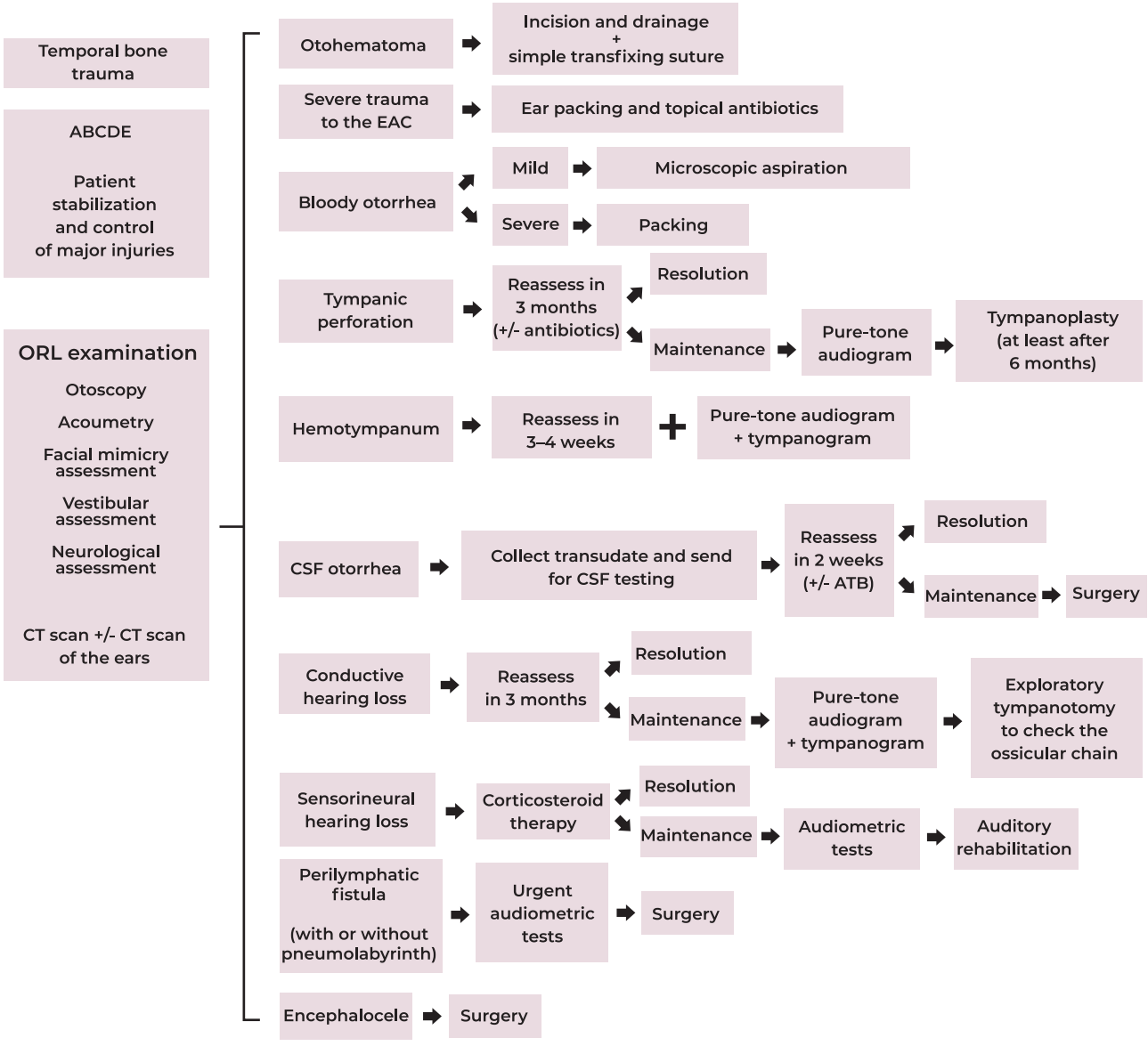
In rare cases of complete nerve transection, end-to-end anastomosis is advised. If the injury involves less than 50% of the nerve diameter, decompression without manipulation of the nerve fibers is recommended[35]. A proposed therapeutic algorithm for patients with temporal bone trauma is presented below, organized according to the clinical syndromes (Figure 1).

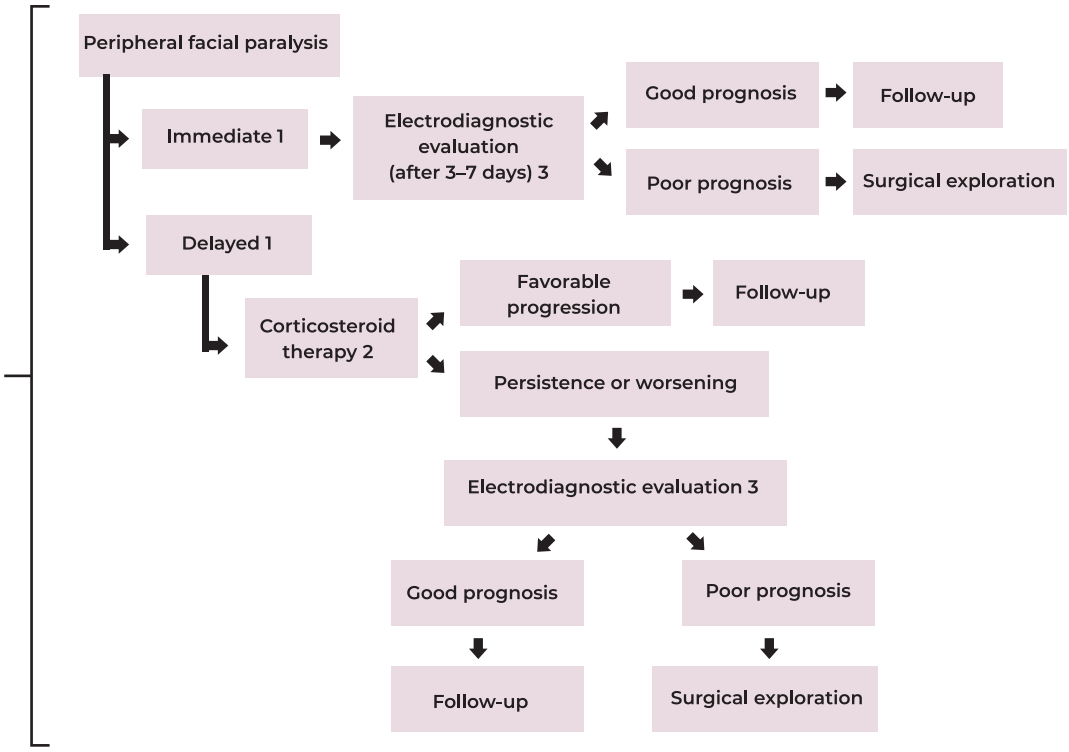
Discussion

The management of temporal bone trauma is inherently complex and requires a multidisciplinary, protocol-based approach to optimize patient care. This condition remains an important topic in otorhinolaryngology due to the potentially serious complications and morbidity. Initial treatment adheres to the ABCDE protocol, prioritizing hemodynamic stabilization and identification of immediate complications, including intracranial injuries and otologic damage using targeted imaging. One limitation is the difficulty in obtaining high-resolution temporal bone images in emergency settings, which can hamper an accurate diagnosis. This underscores the importance of referral centers equipped with the appropriate technology. In this context, high-resolution CT plays a central role in diagnosis. However, timely access to these examinations remains a challenge, even in tertiary hospitals. The proposed algorithm is based on an integration of the current literature and adaptations of the existing protocols already used in tertiary care centers. Management strategies for specific injuries, such as auricular hematoma, trauma to the external auditory canal, and bloody otorrhea, are outlined in a practical approach involving auricular aspiration and use of topical antibiotics. Reassessment of conductive hearing loss and continued monitoring in cases of hemotympanum, tympanic membrane perforation, or ossicular chain disruption are essential components of the protocol to achieve functional hearing recovery. Regarding CSF leaks, we emphasize the importance of prompt and accurate diagnosis using specific protocols for fluid collection and laboratory testing, such as β2-transferrin analysis. Conservative management comprising rest and observation is recommended for low volume leaks. Conversely, persistent encephalocele or ongoing otorrhea typically requires surgical intervention. PFP is also addressed, with a focus on assessing facial nerve function and determining the need for surgical treatment based on the lesion severity. The use of electrophysiological assessments, such as ENoG and EMG, has been highlighted, particularly in early evaluations that can inform prognosis and guide decisions regarding nerve decompression or anastomosis. Although surgery is indicated in cases of severe lesions, this decision must be carefully analyzed after considering the auditory function and duration of paralysis.

The proposed protocol is comprehensive and reflects the need for specialized management of temporal bone trauma. However, its implementation may pose challenges in resource-limited settings, where restricted access to advanced diagnostic tools and lack of continuous professional training can hinder optimal care. Effective implementation requires not only the availability of the appropriate equipment but also ongoing training of healthcare professionals to manage the complexity and potential complications associated with these injuries.

Figure 1
Proposed algorithm for temporal bone trauma. Following initial evaluation and stabilization of major injuries, an otorhinolaryngological evaluation is recommended, supported by imaging studies. For each identified pathology, a corresponding treatment is proposed.
1. Immediate or acute PFP is defined as PFP occurring at presentation or within the first 24 hours of injury. Delayed PFP refers to onset between 24 hours and 16 days post-injury
2. A two-week course of oral corticosteroids is recommended (prednisolone 1 mg/kg/day), followed by a gradual tapering schedule of 20 mg every three days
3. NET or ENoG is also advised. NET compares the neural stimulation threshold between the healthy and affected sides; a difference ≥ 3.5 mA indicates nerve degeneration. ENoG evaluates the percentage of degenerated nerve fibers; a denervation rate < 90% indicates a favorable prognosis. Electrodiagnostic evaluation should be conducted within the first 2 weeks post-trauma. After this window, EMG is recommended.





Abbreviations: CSF, cerebrospinal fluid; CT, computed tomography; EAC, external auditory canal; NET, nerve excitability testing; ENoG, electroneurography; ATB, antibiotics; PFP, peripheral facial paralysis; EMG, electromyography.

Conclusion

Temporal bone trauma is a common condition in emergency departments and requires assessment by an otorhinolaryngologist after initial patient stabilization. Specialist evaluation should aim to exclude conditions requiring urgent surgical intervention through a focused clinical examination and appropriate diagnostic tests. The existence and adherence to a therapeutic algorithm in emergency settings can improve the overall quality of care. However, the successful implementation of such protocols depends on resource availability, the training of medical teams, and the institution's ability to adapt protocols to local conditions.

Conflicts of interest

The authors declare that there is no conflict of interest regarding the publication of this paper.

Data Confidentiality

The authors declare having followed the protocols used at their working center regarding patient data publication.

Protection of humans and animals

The authors declare that the procedures were followed according to the regulations established by the Clinical Research and Ethics Committee and the 2013 Helsinki Declaration of The World Medical Association.

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