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Review Article

Skull base fractures: An institutional experience with review of literature

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ABSTRACT

Fractures of the skull base are relatively common. They have been reported to occur in 3.5–24.0 percent of head injury patients. This wide variation results from differences in studied patient populations and from difficulty of obtaining radiographic verifications of the fracture.[1] The Traumatic Coma Data Bank in a prospective series of severe head injury reported that 25% had basilar skull fractures.[2] In this paper we analyze the various types of fractures and their management.

1. Bio mechanism of the fracture at skull base

Basilar skull fracture basically is a linear fracture in skull base. These fractures are often part of a convexity fracture that has extended into skull base, though they also occur alone. They occur chiefly due to either direct impact or because of propagation of stress waves through the skull as a result of remote impact. They also occur as a consequence of impact to facial bones.[3] The skull base is prone to fracture at certain anatomical sites like sphenoid sinus, foramen magnum, petrous temporal ridge, inner potion of sphenoid wings at skull base. The middle cranial fossa is weakest with thin bones and multiple foramina which predispose it to fractures.

The unique anatomical relationship of the base of skull is responsible for the particular problems that may arise after injury. The dura being firmly adherent to skull base makes it vulnerable to laceration by a fractured bone. Such a fracture places the subarachnoid space in direct contact with the sinuses, middle ear structures or mastoid cells providing a pathway for CSF leak, infection (Meningitis, abscesses) and/or persistent fistula.[4–7] Basal fractures often traverse foramina and thereby may damage cranial nerves[3,6,8] and blood vessels.[6,9]

2. Anatomy of skull base

Skull base can be divided into thee subtypes[6]:

1 Anterior skull base – is related to paranasal sinuses, cribriform plate, and orbital roof.

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2 Middle skull base – includes major parts of sphenoid bone and temporal bone.
3 Posterior skull base – includes clivus, condylar as well as portion of petrous temporal bone.

3. Radiographic diagnosis

Although, plain X-ray skull in anterior-posterior, lateral and basal projections may sufficiently demonstrate the extent of fractures, however, modern CT scans with bone windows, thin section cuts of up to 1–1.5 mm and coronal cuts provide improved detection and resolution of even tiny fractures without increased exposure to radiation.[2,10] Occasionally the fractures may be misinterpreted. False-positives may result when normal sutures are misdiagnosed as fracture lines. False-negatives may occur with subtle fractures or when suboptimal technique is utilized (as when the study is performed using section thickness > 1.25 mm collimation or without employing a bone algorithm). The commonest differential diagnosis of fracture is normal suture; hence one has to differentiate between the two. These can be differentiated by following: suture being less than 2 mm in width, lighter in color on X-rays and being present on known anatomic sites.

Helical CT is helpful in occipital condylar fractures and 3-D reconstruction in temporal bone fractures[11] in addition to providing useful information in other basal skull fractures.[12]

Indirect radiographic findings (on CT scan or plain X-ray film) that suggest a basal skull fracture include pneumocephalus in the absence of an open fracture of cranial vault and air-fluid level within or opacification of air sinuses with fluid.

MRI is of supplementary value for suspected vascular injuries and when cranial nerve palsy occurs in order to recognize neural compression. Moreover, early and late complications of basal skull fractures due to infection, brain contusion and herniation require CT scan to visualize the osseous prerequisites of complication and MRI to define the adjacent brain and soft tissue involvement.[13] Ligamentous injuries are best seen on MRI.

4. Our experience

This study was retrospective in patients with traumatic brain injury from January 2006 to January 2013. Data were collected from the case sheets of the patients. A total of 298 patients were included in the study. The inpatients of traumatic brain injury, who presented with clinical and/or radiological evidence of skull base fractures, were included.

In our result, 198 patients were male and 100 were female, the age group varies from 1 year to 75 years and was mostly prevalent among 21–30 year age group. The most common mode of injury was road traffic accident (Table 1). The most common location was anterior (51%) followed by temporal (47%) (Table 2). We here describe in detail the anterior skull base fractures. We used prophylactic antibiotics consisting of ceftriaxone & sulbactam and amikacin in all the patients for one week and in cases who developed infection, antibiotics were used as per culture sensitivity.

Table 1 – Etiology of skull base fractures in the study group.

S. no	Mode of trauma	N	% Age
1	Road traffic accidents	119	40%
2	Falls	75	25%
3	Falling objects	44	15%
4	Assaults	15	5%
5	Others	45	15%

The anterior skull base fractures were the commonest and amongst them frontal sinus was the commonest to be involved followed by orbital fractures Fig. 1 (Table 3). The clinical features in them were varied and the commonest symptom was local pain and rhinorrhea was seen in 43% patients (Table 4). Surgery was done in 84 patients (60%) and rests were managed conservatively. Surgery was done for CSF leak in only 9 patients (5%) and in rest of the operative group, indication of surgery was removal of contusions or hematomas, repair of sinus or correction of deformity. The commonest complication was local deformity (12%) followed by infection (4%).

5. Discussion

5.1. *Anterior skull base fractures (Rhinobasal)*

Includes fractures of posterior wall of frontal sinus, roof of anterior and posterior ethmoid cells, cribriform–ethmoid junction, and the orbital roof. Such patients present with CSF rhinorrhea, bilateral periorbital ecchymosis (Raccoon's eyes), anosmia and damage to optic nerves and orbital contents.[14]

The anterior and posterior ethmoid foramina are located at the superior aspect of the ethmoid bone in the frontoethmoid suture. Shearing of the corresponding arteries that traverse these foramina may result in orbital hematoma. The optic canal is positioned farther posterior and is less commonly distorted by the fracture lines. However, local edema within the optic canal or free bone fragments may lead to a disturbance in the vascular plexus of the optic nerve, leading to temporary or permanent blindness. CSF leak being of high significance became of association of meningitis, abscess, and persistent fistula, needs to be actively searched for by the attending physician, especially in the presence of craniofacial or high mid facial fractures (Le Fort II and III).[13,15] Further, a

Table 2 – Table showing location of fracture.

	Skull base fracture	N	% Age
1	Temporal bone	140	47%
2	Anterior skull base	152	51%
3	Sphenoid bone	2	0.7%
4	Clivus	2	0.7%
5	Occipital condylar	2	0.7%

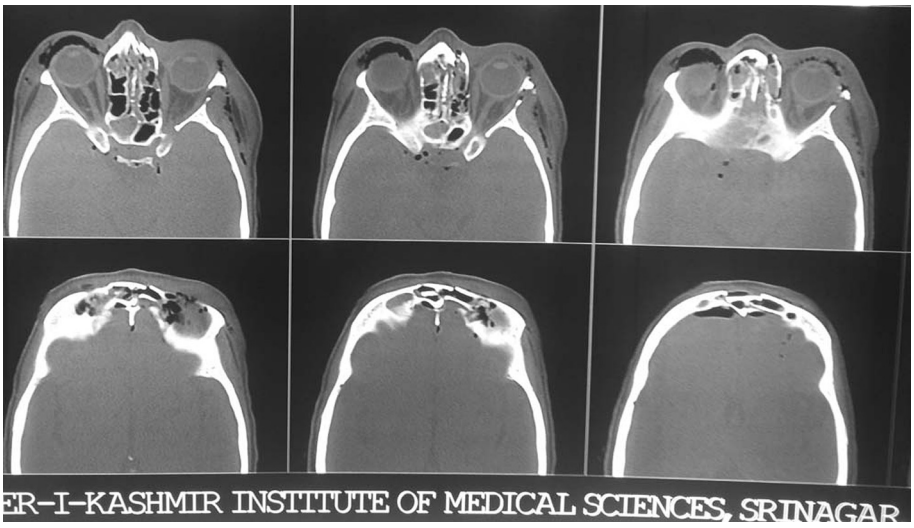

Fig. 1 – CT scan showing fracture involving frontal sinus, nasal bones and ethmoid.

CSF leak may be first detected several days to weeks after trauma due to the fact that CSF leak was initially hidden in bloody nasal discharge from facial fractures or less frequently, due to delayed development of hydrocephalus with rupture of arachnoid at fracture site. A 'double-ring' sign of bloody nasal discharge which is CSF mixed with blood, can be seen on patient's pillow and is a useful bedside test. Further, association with anosmia and salty taste in mouth is highly suggestive.

5.2. *Temporal bone fractures*

One of the common basal skull fractures is categorized as transverse or longitudinal, depending on the axis of fracture in relation to petrous ridge[16] (Fig. 2).

Approximately 80–90% of temporal bone fractures are longitudinal, 10–20% are transverse and 8–10% are mixed.[17] A longitudinal fracture typically runs parallel to the external auditory canal (EAC), through the middle ear, anterior to otic capsule and parallel to petrous ridge extending to the foramen ovale. Transverse fractures run perpendicular to petrous ridge, from the foramen magnum margin across the petrous pyramid, often including the internal auditory canal and into the foramen spinosum or lacerum. Longitudinal fractures typically result from lateral blows to the temporal or parietal skull, while transverse fractures are more often associated with frontal or occipital blows. However, high resolution CT has shown that the most common fracture has elements of both types and is called mixed or oblique fracture, given its slanting course across the petrous pyramid.[18]

Longitudinal temporal bone fractures cause blood and CSF otorrhea (Tympanic membrane disruption), hemotympanum, a bony step-off in EAC, retromastoid ecchymosis (Battle's sign), conductive hearing loss due to disruption of ossicular chain, and in about 20–30% a facial nerve paralysis usually due to geniculate ganglion dissipation. In transverse temporal fractures, tympanic membrane and EAC are usually spared making hemotympanum and CSF otorrhea relatively infrequent. Vertigo, severe sensorineural hearing loss (SNHS) and nystagmus are commonly present because of disruption of internal auditory canal. Approximately 50% of the patients present with facial paralysis with the nerve usually injured in the geniculate ganglion or in the tympanic segment superior to oval window.[16,17]

In order to make it clinically more relevant and predictable, another classification divides these fractures into 'otic capsule sparing' and 'otic capsule violating' types,[19,20] the latter type was more likely to have complications like facial nerve injury, SNHL, vestibular dysfunction and CSF fluid otorrhea than those with the former. Thin section CT is diagnostic and 3-D reconstruction is helpful.[11]

The common complications are:

i) CSF leak and fistulas: occur in 1/3rd of temporal bone fractures. When the fracture tears the dura, CSF leaks into tympanomastoid space. If the tympanic membrane has been disrupted, otorrhea occurs. If membrane is intact, fluid can leak down the eustachian tube into the nasopharynx, leading to rhinorrhea. Vast majority close spontaneously within 2 weeks of trauma.
ii) Hearing loss: 20–80% of patients with temporal bone fractures demonstrate some degree of hearing loss. Patients with known or suspected temporal bone fractures should have their hearing evaluated as soon as possible with clinical and audiological (pure tone Audiometry, speech discrimination) tests. Patients with an altered level of consciousness may be tested using auditory evoked potentials.
iii) Vestibular dysfunction: usually ranging from transient non-specific dizziness to episodic vertigo with nystagmus occurring from several weeks to months after injury. It has been attributed to vestibular concussion and is generally self limited with no permanent sequelae. However, trauma can also lead to benign paroxysmal positional vertigo (BPPV) and development of hydrops of endolymphatic sac which need to be treatment on their own merits.

Table 3 – Location of fracture in anterior skull base.

	Radiological finding	N	% Age
1	Anterior and posterior wall frontal sinus fracture	56	40%
2	Ethmoid fractures	22	16%
3	Cribriform plate fractures	26	19%
4	Orbital roof fractures	48	34%

iv) Facial nerve injury: complicates about 3% of all head injuries and practically all cases of post-traumatic facial palsy occur in the setting of temporal bone fractures, with >90% located in the perigeniculate region. It is caused by a hematoma within the nerve, anatomic disruption, contusion or an impinging bony fragment. Clinical assessment and electrophysiological testing in the form of electroneuronography (ENOG) must be regularly performed to determine the timing of onset of facial weakness (i.e. immediate or delayed) and whether injury is partial or complete.

5.3. *Sphenoid bone fractures*

It includes fractures of body of sphenoid (via sphenoid sinus, sella turcica and upper clivus) (Fig. 3), fractures of greater wing (through orbital, basal and temporal surfaces), fractures of lesser wing (via planum and anterior clinoid) and fractures passing through pterygoid process.

Considering all fractures of sphenoid bone, they exceed in number to all other basal skull fractures including anterior basal and temporal bone fractures.[21] Such fractures may result in[6,21]:

i) CSF rhinorrhea
ii) Injury to cranial nerves III, IV, VI: causing ophthalmoplegia
iii) Optic nerve injury
iv) Chiasmal injury and endocrine abnormalities (in sellar fractures)
v) Injury to ICA: supraclinoid portion with a potential to cause cerebral ischemia, pseudo aneurysm formation and caroticocavernous fistulas. Carotid injury can also occur when a fracture line runs across carotid canal. CT angiography should, therefore, be done as soon as fracture running in proximity to course of internal carotid artery is detected on a thin slice CT and it becomes more worthwhile since appropriate and expeditious treatment of carotid injury and its complications has become safely available after enormous developments in endovascular treatments.

Table 4 – Number of patients according to the relevant clinical presentation.

	Clinical presentation	N	% Age
1	Local pain	140	100%
2	Esthetic deformity	16	12%
3	CSF leak	60	43%
4	Brain contusion	55	39%
5	Diplopia	20	14%
6	Anosmia	15	11%

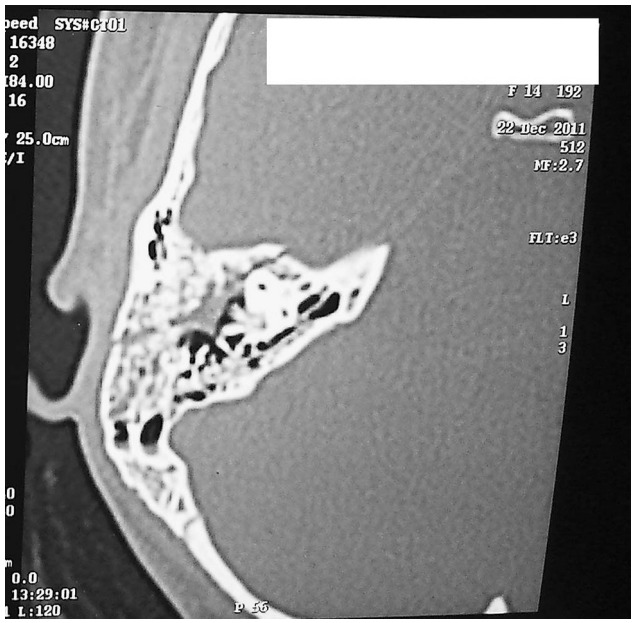

Fig. 2 – CT scan shows fracture line extending along right temporal bone and reaching the surface.

6. Clivus fractures

These rare fractures have been described in high speed motor vehicle accidents and attributed to high energy impacts therein. Longitudinal, transverse, and oblique types have been described in the literature. These are rarely diagnosed on conventional X-rays studies. Longitudinal fractures are associated with injuries of vertebrobasilar system and brain stem trauma or both and are usually highly lethal. The abducent nerve may be directly damaged in these fractures. The transverse fractures extend through the carotid canal and petrous temporal bone. While less frequently contributing directly to mortality, transverse fractures are associated with CSF leaks, cranial nerve injuries, diabetes insipidus and caroticocavernous fistulas.[22]

7. Occipital condylar fracture

Very uncommon, these result from a high energy blunt trauma and are divided into three types:

Type 1: secondary to axial compression resulting in comminution of occipital condyle.

Type II: result from a direct occipital blow, where condylar fracture is an extension of linear basal skull fracture.

Type III: avulsion of condylar fragment occurs during rotation, lateral bending or a combination of mechanisms. This is an unstable fracture.

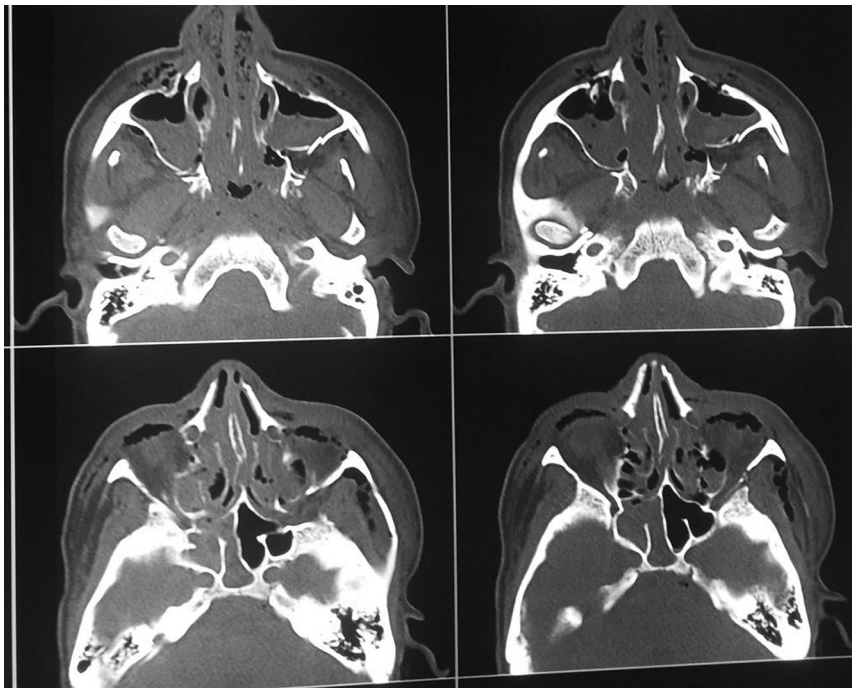

Fig. 3 – Fracture involving sphenoid body, maxilla with hemo maxilla and blood is also seen in sphenoid sinus.

They should be suspected in any blunt trauma with craniocervical injuries causing altered mental status/coma, quadriparesis, occipital pain or tenderness, lower cranial nerve palsies or retropharyngeal soft tissue swelling. They are rarely diagnosed on plain X-rays and CT scan is recommended for establishing the diagnosis. MRI is recommended to assess the integrity of craniocervical junction.[23]

8. Management

8.1. *Ethmoid facture*

Use of antibiotics has not proven effective in changing the incidence of meningitis in post-traumatic CSF leaks and therefore, they are no longer recommended routinely.[24–26] Most acute post-traumatic leaks stop spontaneously within 10 days of injury. This is also true of leaks or dural tears associated with midface fractures when they are reduced.[26] The use of lumbar drainage may further increase the rate of healing. Persistence of CSF leak beyond 10 days, recurrent CSF leak, a bout of meningitis and pneumocephalus are indications for neurosurgical intervention. CT cisternography or MRI is done to precisely localize the fistula, followed by obliteration of fistula by dural patch graft or fat plus or muscle pledgets or injection of fibrin or other glues or tissue transfers may be needed. Usually, subfrontal or frontobasal approaches with intradural or extradural repair (intracranial) or combination of two is done.[5,6] Endoscopic repairs have also been a common practice at many centers.[27]

Regarding optic nerve injury following anterior basal fractures, it has been seen that there is no difference in improvement of vision in patients treated with surgical decompression versus non-surgical methods (steroids and expectant management).[28] The recovery of VEPs from no response to abnormal wave or abnormal wave to normal VEP is both indicators of relatively good visual prognosis. The surgical decompression is indicated for documented deterioration of initially good vision following head trauma especially in case of retrobulbar hematoma and CT/MRI documented bone fragment dislocation into the orbit, comprising retrobulbar part of optic nerve.[6,28]

8.2. *Temporal fracture*

Most of the patients can be managed conservatively. Mortality in the setting of temporal bone fracture typically occurs from an associated injury (like, closed head trauma, abdominal or thoracic injury) caused by blunt trauma. Mortality may also result, rarely, from delayed complications of fracture, such as meningitis.

8.3. *CSF leak*

Bed rest, elevating head 30°, stool softeners and occasionally acetazolamide are used as conservative treatment. Lumbar subarachnoid drainage is helpful in selected group of patients. Prophylactic antibiotics are controversial and many centers have policy of using these and many do not use them till evidence of infection is there. Surgical therapy is indicated for persistent (>2 weeks) fistulas, persistent or enlarging pneumocephalus, and meningitis. Through a subtemporal extradural or intradural approach, dura can be closed primarily or more commonly a dural substitute graft is placed.[5,6]

8.4. *Hearing loss*

Conductive hearing loss can result due to injury to the auricle, external auditory canal, tympanic membrane, or ossicular chain. About 80% of such patients improve spontaneously. Conductive hearing loss greater than 30 dB persisting beyond 3 months in a patient with intact tympanic membrane is highly suggestive for ossicular chain disruption and an otolaryngologist should be consulted for a possible ossicular chain reconstruction. SNHL can result due to trauma to cochlea, cochlear nerve or central auditory pathways. The prognosis of recovery of significant SNHL is poor. Most authors recommend amplification with a hearing aid.[29,30]

8.5. *Vestibular dysfunction*

Electronystagmography or MRI can be valuable in the work up of a patient with post-traumatic vestibular dysfunction to identify or exclude central lesion. Leakage of perilymph from inner to middle ear (perilymphatic fistula) can cause both hearing loss and vestibular dysfunction. Surgical exploration and repair of fistula with muscle, fat, autologous or allograft material will be needed.[30]

8.6. *Facial nerve injury*

Approximately 90% of patients with delayed onset of facial paralysis or those who have partial weakness will fully recover spontaneously. Thus conservative treatment is indicated in these patients which includes short term oral corticosteroid therapy to reduce edema of the nerve within the fallopian canal. Surgical intervention is warranted in patients with penetrating injury or in whom radiography (high resolution CT) demonstrates an obviously discontinuous fallopian canal with immediate complete facial paralysis. In case of delayed facial palsy, if ENOG drops to less than 10% of the contralateral side, surgical decompression may be considered. Removal of hematoma and bone fragments (decompression) facilitates nerve regeneration. Severely contused or disrupted nerves require resection of the damaged portion with end to end anastomosis, in case resulting nerve is not under tension or cable grafting with sural or greater auricular nerves in case segment loss is significant. Facial nerve grafting, when successful, can require upto 2 years for full regeneration.[6,30,31]

8.7. *Sphenoid fracture*

CSF rhinorrhea: in sphenoid sinus fracture is managed like an anterior basal fracture. In addition repair can be done via trans-sphenoidal route.

Injury to ICA: supraclinoid portion with a potential to cause cerebral ischemia, pseudo aneurysm formation and caroticocavernous fistulas. Carotid injury can also occur when a fracture line runs across carotid canal. CT angiography should, therefore, be done as soon as fracture running in proximity to course of internal carotid artery is detected on a thin slice CT and it becomes more worthwhile since appropriate and expeditious treatment of carotid injury and its complications has become safely available after enormous developments in endovascular treatments.[21]

Endocrinopathy: usually responds to hormonal replacement.

8.8. *Clivus fracture*

These are invariably fatal. However in the surviving patients basic management is as in other basal fractures.[22,32]

8.9. *Occipital condylar fracture*

These are treated with external cervical immobilization (hard cervical collar/halo) for 6–8 weeks; however, those type III injuries that involve total ligamentous disruption are best managed with a posterior occipitocervical arthrodesis.[23]

Conflicts of interest

All authors have none to declare.

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