Surgical Management of Non-Missile Penetrating Brain Injury

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Abstract

Introduction: Non missile penetrating brain injury secondary to car crashes or industrial accidents are not infrequent neurosurgical emergencies. The management of such cases is characterized by many challenges, not only from the surgical point of view, but also for the different and sometimes bizarre dynamics by which they present. We report our experience in surgical planning to treat such.

Material and Method: Total of 32 patients were treated at Neurosurgery Department, Menofyia University, all of them were presented by non missile penetrating brain injury age ranged from 5 to 52 years, 17 males and 5 females, industrial injury was the most common followed by assault and car crashes. Plain skull X-ray standard views, plain CT brain, CT brain angiography done for cases where the foreign body is suspected to be in the vicinity of major vessels.

Results: Wide craniotomy under broad spectrum antibiotics, with preservation of the non damaged brain tissues, adequate haemostasis, packing of the air sinuses, good reconstruction of the skull base if involved, dura reconstruction are the main surgical tricks to grantee excellent outcome.

Conclusions: Adequate preoperative planning aided with full radiological assessment with preservation of the non damaged brain tissues and proper surgical techniques are the main keys of getting surgical outcome in such challenging neurosurgical emergency.

Key Words: Non-missile – Brain injury.

Introduction

PENETRATING Brain Injury (PBI) includes any traumatic injury where an object pierces the skull and breaches the meninges surrounding the brain. PBIs are less prevalent than blunt head injuries, representing 0.4% of injuries, however, they often have more complex damage, worse prognosis and higher rates of morbidity and mortality [1,2].

PBI can be classified as missile or non-missile; the main difference between the 2 is the velocity of impact [3]. Non-Missile Penetrating (NMP) lesions are defined as having an impact velocity of <100m/s, causing injury by laceration and maceration, whereas missile projectiles cause lesions by kinetic and thermal energy [4]. In addition, NMP injuries may classify in 2 types: Those entering through a natural orifice (orbit, nose, mouth, or ear) and those whose object really crosses the skull, causing a fracture and creating an artificial orifice [5]. It is important to note that there are two distinct types of damage that can result from a PBI, namely primary brain injury and secondary brain injury.

Primary brain injury refers to injury caused by the trauma of the penetrating object itself. The effects are generally less devastating than secondary brain injuries and include hemorrhage, intracranial lesions and skull fractures. Primary axonal damage can trigger events that can lead to secondary brain injury, such as neurotoxic biochemical cascades, hematoma formation, blood loss, infection or seizure [6]. These effects usually occur around 24-48h post-initial injury and are generally more devastating, resulting in brain herniation infarction and/or post-traumatic atrophy. secondary brain injury [2,3].

There are two goals in PBI imaging. The first is to assess primary brain injury in the acute setting to aid management and treatment planning, and secondly to detect secondary brain injury in the sub-acute setting which helps predict long-term effects and patient prognosis. A non-contrast cranial CT is typically the primary imaging for PBI, MRI plays an important role in providing an extensive and precise evaluation of tissue status. MRI is more sensitive than CT, both acutely and later post-injury, in the detection of haematomas, haemorrhages and white matter injuries such as Diffuse Axonal Injuries (DAI) which affects a greater area of the brain [3,5]. Cerebral angiography is strongly recommended in PBI patients with an increased risk of vascular injury. MR Angiography (MRA)
and CTA have been shown to be equivalent in the
detection of carotid and vertebral artery dissection,
luminal narrowing, pseudo-aneurysm [6,7].

PBI management differs greatly from that of
non-penetrating brain injury due to the mechanism
of injury and subsequent pathophysiology of the
trauma [8]. Initial management involves surveying
and stabilizing the patient to reduce the risk of
secondary brain damage, resulting from increased
intracranial pressure or reduced cerebral perfusion.
These injuries present a significant management
challenge as extensive surgery may be required,
which can result in significant morbidity and mor-
tality [5,7]. So the neurosurgical management of
NMP injuries is based on the correction of entry
point to avoid Cerebral Spinal Fluid (CSF) leakage
(primary lesion) and to treat secondary lesions
caused by the penetrating object, such as brain
parenchyma and vascular lesions [6,8].

Material and Methods

Total of 32 patients were treated at Neurosurgery
Department, Menofyia University, during the period
from January 2008 to December 2012. Admission
records were reviewed, all of them were presented
by non missile penetrating brain injury.

Patients were admitted through the Emergency
Department where initial resuscitation was carried
out then shifted to neurosurgery service for man-
agement.

Initial neurological evaluation was done using
Glasgow coma scale and recorded in patient file,
patients were classified accordingly into mild (GCS
15-13), moderate (GCS 12-9), severe (8-3).

All patients had plain X-ray skull standard
views/plain CT brain with coronal and axial views
upon admission for assessment of the primary brain
insult and to plan for surgical treatment.

Patients were given broad spectrum antibiotic
to cover gram positive, gram negative and anaerobic
organisms, antiepileptic prophylaxis. Anti edema
measures to control increased intra cranial pressure.

Cerebral angiography/CT brain angiography
were done for patients with suspected vascular
injury.

All patients were operated via wide craniotomy,
removal of the foreign body, debridement of all
necrotic tissues, reconstruction of the dura, and
skin closure.

Patients were kept at Intensive Care Unit till
their clinical status permit to be discharged to the
ward.

All patients had follow-up CT brain next day
of surgery except those who had neurological
deterioration they had CT brain done immediately
and managed accordingly.

MRI brain was done for patients who had de-
layed neurological recovery to evaluate the brain
parenchyma and rule out presence of brain abscess
or diffuse axonal injury.

In this review we analyzed, the mechanism of
injury, the presence of secondary injury to the brain
(subarachnoid hemorrhage, brain hematoma, vessel
injury, etc.), clinical presentation according to the
Glasgow Coma Scale (GCS), operative technique,
peri-operative complications, neurosurgical man-
agement, and out-comes according to the Glasgow
Outcome Scale.

Results

A total of 32 patients were enrolled in this
retrospective study. The age ranged from 10 to 52
years with mean age (38 ±7.6 years), most of pa-
tients were between second to fifth decade of life,
regarding the gender 23 males and 9 females (Table
1). Industrial injury was the most common cause
followed by assault and self-inflicted, penetration
through the non-natural orifices was more common
than penetration through natural orifices (Table 2).

According to mode of injury, penetrating injury
where the foreign object penetrates skull and dura
and remains lodged within the intracranial cavity
this encountered in 25 patients, tangential where
the foreign object glances off the skull, often
driving bone fragments into the brain in 7 patients,
and perforating injury 'through-and-through' injury,
characterized by entry and exit wounds non of our
patient sustained such type of injury.

According Glasgow Coma Scale (GCS) done
at the initial neurological evaluation upon admission
after resuscitation, patients were classified into
three groups mild (GCS 15-13) 15 patients, mod-
erate (GCS 12-9) 9 patients, sever (8-3) 8 patients.

Plain skull X-ray standard views, plain CT
brain with axial and coronal views done for all
patients upon admission, it showed intra cerebral
hematoma in 8 cases, intra ventricular hemorrhage
in 4 cases, sub arachnoid hemorrhage in 3 patients,
brain edema in 18 patients CT brain angiography
done for 4 patients, where the foreign body was
suspected to be in the vicinity of major vessels, venous sinus or traversing two brain compartment.

The most frequent location of NMP injuries through natural orifice was through the roof of the orbit because of its fragility this occurs in 9 out of 12 patients, leading to injuries and contusion of the frontal lobe. The second most common site was through the mouth 2 followed by the nose in one case.

All patients had surgical exploration through wide craniotomy, with localized craniectomy at the entrance of the foreign body following the surgical exploration. Patients kept at ICU for care, we have 6 patients kept on mechanical ventilation for more than 10 days, 6 patients were successfully weaned of mechanical ventilation, while 2 patients were deeply comatose and were kept on mechanical ventilation both died later on due to septic shock.

Morbidity and mortality among 32 patients with NMP, 7 patients had post operative convulsions and kept on anti epileptic for 6 month post operative, subarachenoid hemorrhage in 3 patients two of them had post traumatic hydrocephalus which was treated by ventricluo-peritoneal shunt, 6 patients had Cerebro-Spinal Fluid (CSF) leak, 4 of them developed bacterial meningitis which was treated by antibiotic therapy according to the culture sensitivity result and two of them had to be taken to operating room to graft the dura and close the leak, among 4 patients with meningitis one patient had post-meningetic hydrocephalus which was treated by ventricluo-peritoneal shunt after clearance of infection, two patients had brain abscess due to reaind infected tissue which was tapped twice and 2 patients remained in vegetative state (Table 3).

Comparing the neurological outcome 15 patients admitted with mild degree, 22 patients discharged with mild degree, 9 with moderate GCS score 6 cases discharged in moderate, 8 cases with sever GCS on admission 2 cases died and two remained vegetative.

Table (1): Gender and age of 32 patient with non missile penetrating brain injury.

<table>
<thead>
<tr>
<th>Age group</th>
<th>Male</th>
<th>Female</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>10-20</td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>21-30</td>
<td>7</td>
<td>3</td>
<td>10</td>
</tr>
<tr>
<td>31-40</td>
<td>9</td>
<td>3</td>
<td>12</td>
</tr>
<tr>
<td>41-50</td>
<td>4</td>
<td>2</td>
<td>6</td>
</tr>
<tr>
<td>More than 51</td>
<td>2</td>
<td>–</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>23</td>
<td>9</td>
<td>32</td>
</tr>
</tbody>
</table>

Table (2): Mechanism of injury and GCS on admission of 32 patients with non-missile penetrating brain injury.

<table>
<thead>
<tr>
<th>GCS Grades</th>
<th>Mechanical of injury</th>
<th>Site of penetration</th>
<th>Mode of injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>15-13 (Mild)</td>
<td>Industrial truma</td>
<td>Natural orifice</td>
<td>Penetrating</td>
</tr>
<tr>
<td>12-9 (Moderate)</td>
<td>Assault</td>
<td>Non-natural orifice</td>
<td>Tangential</td>
</tr>
<tr>
<td>8-3 (Severe)</td>
<td>Self inflicted</td>
<td></td>
<td>Perforating</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Mechanism of injury</th>
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</tr>
<tr>
<td>Self inflicted</td>
<td></td>
<td>Perforating</td>
</tr>
</tbody>
</table>

Table (3): Morbidity in 32 patients with non-missile penetrating head trauma.

<table>
<thead>
<tr>
<th>Type</th>
<th>Morbidity Number of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Convulsions</td>
<td>7</td>
</tr>
<tr>
<td>CSF</td>
<td>6</td>
</tr>
<tr>
<td>Meningitis</td>
<td>4</td>
</tr>
<tr>
<td>Re-exploration</td>
<td>4</td>
</tr>
<tr>
<td>Vegetative state</td>
<td>2</td>
</tr>
<tr>
<td>Hydrocephalus</td>
<td>3</td>
</tr>
<tr>
<td>Brain abscess</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>28</td>
</tr>
</tbody>
</table>

Table (4): Relation between degree of injury and GCS on admission and discharge in 32 patients with non missile penetrating head trauma.

<table>
<thead>
<tr>
<th>Degree of injury</th>
<th>GCS on admission</th>
<th>GCS on discharge</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>15</td>
<td>22</td>
</tr>
<tr>
<td>Moderate</td>
<td>9</td>
<td>6</td>
</tr>
<tr>
<td>Severe</td>
<td>8</td>
<td>2</td>
</tr>
</tbody>
</table>

Table (5): Clinical outcome comparing the GCS on admission and discharge.

<table>
<thead>
<tr>
<th>Degree of injury</th>
<th>GCS on admission</th>
<th>GCS on discharge</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>15</td>
<td>22</td>
</tr>
<tr>
<td>Moderate</td>
<td>9</td>
<td>6</td>
</tr>
<tr>
<td>Severe</td>
<td>8</td>
<td>2</td>
</tr>
</tbody>
</table>
Fig. (1): A case of craniofacial trauma.

Fig. (2): Penetratin trauma by nail.
Discussion

Non missile Penetrating Brain Injury (NMPBI) with foreign bodies represents only 0.4% of all head trauma [3]. It is more common in wars or conflicts and uncommon in daily practice, especially if these objects are of low kinetic energy, defined as projectiles with a speed <100m/s [2-4]. The most frequent location of NMP injuries is the roof of the orbit because of its fragility, leading to injuries and contusion of the frontal lobe. The second most common site is the superior orbital fissure, which is in contiguity to the cavernous sinus, and may cause additional injury to the II, III, and IV cranial nerves [3,9,10].

In the present series, the most frequent location of NMP injuries through natural orifice was through the roof of the orbit because of its fragility this occurs in 9 out of 12 patients, leading to injuries and contusion of the frontal lobe. The second most common site was through the mouth 2 followed by the nose in one case seen in our series, most of our patients were in middle age group second and third decade most of them were young manual worker and hence the main cause of trauma was due to industrial injuries, aggression was the second most cause of trauma in this series, incidental injury was common in kids. We had a psychiatric patient who had self-inflicted injury, while in three patients it was suicidal attempt, we didn't have any victim of aggression precipitated by ethanol intake in this series compared to other series.

The non-contrast cranial CT, coronal reformations can improve detection and characterization of intra-cranial haemorrhage when compared with only using standard axial images. It was found that several foci of intra-cranial haemorrhage oriented transversely in the axial plane were found to be completely invisible on the axial images alone. CT is limited in the evaluation of the posterior fossa, middle cranial fossa and inferior frontal lobes, as Hounsfield artefacts can obscure these anatomical locations. Therefore, it is suggested that coronal and sagittal CT reconstructions also be performed to provide more detailed evaluation of these areas [11]. It is also valuable in the acute trauma setting due to its rapid scan times and compatibility with life support and monitoring devices, unlike the potential difficulties associated with MRI [5,10].

MRI is superior to CT in detecting brain parenchyma abnormalities, it is time-consuming, contraindicated if ferromagnetic objects are present and difficult to perform on unstable or ventilated patients, therefore is generally not recommended for acute imaging [3,11]. Monitoring equipment is often bulky and not compatible with this imaging modality, however it is more sensitive to detect wooden or non-metallic foreign bodies, for which CT is far less sensitive. This can be better achieved using gadolinium-enhanced MRI [12,13].

Plain films can still be useful in providing a general overview of fractures, as well as trajectory and location, however, the role today is limited, particularly when CT scout views can be equally valuable. If plain films are deemed necessary, they should only be obtained if delays to CT examination and further management will not be incurred [14].

Vascular injury to the head is a potentially life-threatening condition that results in 20% to 30% of all PBIs [15,16]. Vascular injuries following PBI fall into three main categories: Arterial dissection,
subarachnoid haemorrhage and traumatic intracranial aneurysms [5,17].

Rapid and accurate diagnosis of vascular injury is an integral stage of imaging, as emergency intervention may be needed to prevent potentially fatal neurological sequelae. 20 Confirmation of a fracture near the carotid canal signifies a 35% chance of dissection of the internal carotid artery. 53 other increased risk factors are the trajectory crossing dural compartments, sylvian fissure, supraclinoid carotid artery, the vertebrobasilar vessels, the cavernous sinus region or the major venous sinuses [3].

An initial negative angiogram may not always be conclusive as vascular injuries may have a delayed onset, manifesting weeks or months after the trauma [8]. If suspicion is maintained, or there is a development of an unexplained subarachnoid haemorrhage or delayed haematoma, further angiography would be recommended [3,7].

Diagnosis of vascular injury to the brain following PBI can be made using conventional CT or MR angiography. Conventional angiography is an invasive procedure with a potential risk of severe complications, including thrombosis of the femoral artery, arterial spasm and ischaemia in 0.16-2% of cases [13]. The procedure may take up to an hour, which is questionable for unstable trauma patients and has therefore been recently superseded by CT Angiography (CTA) [4,13]. CTA demonstrates the course of the foreign object in relationship to the cerebral structures of the brain and any possible vascular injury [13]. Added benefits to CTA involve the wide availability of CT scanners, it is minimally invasive and has a short acquisition time (<1min). Disadvantages compared to conventional angiography include potential degradation of image quality from artefacts, and no possibility of therapeutic intervention directly after diagnosis [18].

The complication rates in penetrating stab injuries are significantly higher than in closed head injuries. There is a higher risk of Cerebrospinal Fluid (CSF) leakage or brain abscess development because of the disruption of the dural barrier [14,19]. Also, the risk of posttraumatic epilepsy is higher probably due to direct traumatic injury to the cerebral cortex with subsequent scarring. It is reported that the more severe the injury to the brain, according to the Glasgow Coma Scale (GCS), the higher the risk for the development of posttraumatic epilepsy [1,14]. Vascular complications after PBI range from under 5% to 40% in various reports [9,16,18].

Vascular complications after PBI range from under 5% to 40% in various reports [13,20,21]. Traumatic aneurysms formation is the most commonly described vascular injury [7,13,20]. Pre-operative cerebral angiography is recommended when there is a suspicion of vascular injury. Features associated with higher risk of vascular injuries development in missile wounds include orbitofacial or pterional PBI, presence of intracranial hematoma and injuries with fragments crossing two or more dural compartments [4,13,20]. An angiography is also strongly recommended in case of delayed and/or unexplained subarachnoid hemorrhage or intracranial hematoma development [3,4,7,15,20]. The importance of angiography in the diagnosis of vascular complications lies in the bad outcome associated with this pathology when it is not aggressively.

Independent of NMP cranial injury type, whether this injury is through a natural or non-natural orifice, surgical management is usually recommended. Several aspects should be taken into account: Entry point (natural orifice or not), size of object, number of fragments, and secondary brain or vascular lesions. As shown in the preoperative radiological assessment [14].

Antibiotic prophylaxis is indicated for patients with NMP cranial injuries and should cover gram-positive, gram-negative, and anaerobic bacteria because the foreign bodies are highly contaminated. Using this antibiotic policy, we did not have infection among the patients included in this study [16].

Most cases require craniotomy both to remove the object under direct vision, to treat secondary lesions, and to repair the dura mater and prevent CSF leakage [17].

Therefore, the goals of surgical treatment for NMP cranial injuries include local debridement of damage to the scalp, skull, dura mater, and brain parenchyma, followed by dural closure, cranioplasty, and cutaneous suture, which prevent CSF leak and infection [18].

For patients presenting with brain parenchymal secondary lesions or hemorrhage, Antiepileptic Drugs (AEDs) should be considered. If seizures are not evident in the acute phase, AEDs may be discontinued after 7 days, or as otherwise indicated [14]. In our study we used to keep patients on antiepileptic with no evidence of seizure after 6 months and their EEG didn't show any abnormal EEG activity, we withdraw the drug.
The most frequent location of NMP injuries through natural orifice was through the roof of the orbit because of its fragility this occurs in 8 out of 12 patients, leading to injuries and contusion of the frontal lobe. The second most common site is the superior orbital fissure, which is in contiguity to the cavernous sinus, and may cause additional injury to the II, III, and IV cranial nerves.

Approximately 30-50% of patients suffering a PBI will develop seizures [14]. It is estimated that up to 10% of them will appear early (first 7 days after the trauma). Although the initial studies did not confirm the beneficial effect of the prophylactic anticonvulsants administration, more recent ones recommend the prophylactic anticonvulsants use in the first week after injury. It is acceptable not to use prophylactic anticonvulsants at all in case of smaller, less serious trauma. Besides, the use of anticonvulsants beyond the first 7 days of injury is not recommended [14,17].

Outcome:

Low GCS is associated with an unfavorable outcome, patients with a GCS score <8 are described as having severe injuries, patients with GCS scores of 9-12 are classified as moderate and patients have a better GCS score, ranging from 13 to 15 [18].

Pupillary size and light reflex:

Bilaterally fixed and dilated pupils are highly predictive of mortality in PBI patients. An abnormal pupillary light reflex may be an indirect indicator of cerebral herniation or possible brain stem injury. Measurement of pupillary reactivity includes measurement of the light reflex and size of the pupils [21].

Predicting prognosis after TBI is complex, and existing prognostic models are not suitable for use at the level of the individual patient. In cases of NMP injuries, because of their low prevalence, this task is even more difficult because we do not have a broad review of the subject in the literature. This is because most of the articles, like ours, present results based on case with respect to the results in this article, the greatest statistically significant prognostic factor, similar to TBI in general [19], is the GCS score at admission. This was observed in those patients with GCS scores of 1-5 at admission, who all showed satisfactory developments, whereas among patients with GCS scores <8, 2 died and 2 patients remained vegetative.

Increasing age is correlated with a worse prognosis in PBI. Age greater than 50 is documented to be associated with an increased mortality in PBI patients, this also seen in our patients. Surgical exploration is warranted for PBI when deemed necessary. The procedure recommended in literature is debridement of necrotic brain tissue, removal of accessible bone or foreign body fragments only when the neurological risk is not increased, removal of intracranial hematomas with significant mass effect, and watertight closure of dural defects [22]. However, it remains debatable what technique, craniotomy or craniectomy, is best suited to achieve the best results. No statistically significant advantage of one technique over the other has been described in reports of morbidity and mortality rates associated with two procedures [15,23].

Infectious complications are not uncommon after PBI, and they are also associated with higher morbidity and mortality rates. They are more frequent when CSF leaks, air sinus wounds, transventricular injuries or those ones crossing the midline occur [3,16]. Staphylococcus aureus is the most frequently associated organism. Intravenous prophylactic broad spectrum antibiotic therapy is recommended in all cases and must be started as soon as possible. Currently, it is recommended to maintain it for at least 7-14 days [14,16].

The risk of posttraumatic epilepsy after PBI is high probably due to direct traumatic injury to the cerebral cortex with subsequent cerebral scarring. It is reported that the more severe the injury to the brain according to the Glasgow Outcome Scale (GOS) grade, the higher the risk for the development of posttraumatic epilepsy. About 30%-50% of patients suffering a PBI will develop seizures. It is estimated that up to 10% of them will appear early (first 7 days after the trauma), and 80% during the first 2 years, but about 18% may not have their first seizure until 5 or more years after injury [15,23].

Although the initial studies did not confirm the beneficial effect of the prophylactic anticonvulsants administration, more recent ones recommend the prophylactic anticonvulsants use (e.g., phenytoin, carbamazepine, valproate, or phenobarbital) in the first seizure until 5 or more years after injury [4,24]. It is acceptable not to use prophylactic anticonvulsants at all in case of smaller, less serious trauma. Besides, the use of anticonvulsants beyond the first 7 days of injury is generally not recommended [24,25].

References


