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Clinical Study

Clinical significance of skull base fracture in patients after traumatic brain injury

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ABSTRACT

About 4% of all head injuries include skull base fractures. Most of these fractures (90%) are secondary to closed head trauma; the remainder are due to penetrating trauma. We reviewed the records from January 2006 through December 2008 of all patients older than 18 years of age who arrived at Soroka Medical Center in Be'er-Sheva, Israel, with skull base fractures following a traumatic brain injury (TBI). We identified 107 patients with a mean age of 42 years at the time of TBI. Glasgow Coma score on arrival predicted the clinical outcome. We observed temporal fractures in 30% of these patients, occipital fractures in 20%, pyramidal fractures in 19%, anterior skull base fractures in 17%, and multiple fractures in 14%. Cerebrospinal fluid (CSF) leak was observed in 16 patients (15%). Of the patients experiencing CSF leaks, otorrhea occurred in 10 (62%) and rhinorrhea occurred in six (37%). Three patients required surgical intervention to repair the leak. Meningitis occurred in four patients with clinically evident CSF leak. Multiple skull base fractures are associated with poor neurological outcome. The low rate of meningitis in this patient sample implies that there is no indication to administer prophylactic antibiotics to patients with skull base fractures.

1. Introduction

About 4% of all head injuries include skull base fractures [1,2]. Most of these fractures (90%) are secondary to closed head trauma; the remainder are the result of penetrating injury [1,3,4]. Due to the presence of blood vessels and nerves at the base of the skull, fractures in this area are often accompanied by complications arising from damage to these tissues [5].

Post-traumatic cerebrospinal fluid (CSF) leaks occur in 12% to 20% of patients with skull base fractures [6–8]. In the majority of these patients, the leak stops spontaneously within a few weeks. However, meningitis and/or encephalitis occur in 20% of patients with a CSF leak [9–11].

The ethmoid roof and the cribriform plate region are the sites most vulnerable to dural tears. The junction between the cribriform plate and the ethmoidal labyrinth is particularly susceptible to traumatic injury because the bone in this structure is delicate, and the adjacent dura is tightly adherent. Hence, patients with anterior skull base fractures are at high risk of developing CSF fistulae [6,8,12,13].

Skull base vascular injuries occur in about 50% of patients with skull base fractures. Most of these injuries are "silent" and have no clinical impact. However, vascular injury may lead to a devastating neurological outcome and death [2,14,15].

Diagnosis of skull base fractures is based on typical clinical presentation and verified by imaging studies. The imaging method of choice is the high-resolution CT scan. Although MRI can investigate soft tissue and blood vessels in more detail than CT scans can, it is time-consuming and unsuitable at the acute stage [12,16–18].

In this study, we will report our experience with patients who sustained skull base fractures. We will review the incidence of CSF leak in these patients, as well as the subsequent risk of neurological deficits and their overall outcome.

2. Materials and methods

We retrospectively reviewed the charts of patients older than 18 years of age who arrived at Soroka Medical Center in Be'er-Sheva, Israel, from January 2006 to December 2008 with a skull base fracture following a traumatic brain injury.

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We identified 107 patients with a mean age of 42 years at the time of injury. We reviewed their medical records for demographic data, age, mechanism of injury, neurological status, and neuroimaging studies upon arrival. Over the course of their hospitalization, patients were monitored for the development of CSF leaks and meningitis. The neurological outcome at the day of discharge was evaluated using the Glasgow Outcome Scale (GOS), a five-point scale that rates post-injury level of function (5 = normal activity with minor motor or mental disabilities; 1 = dead). Rates of rehospitalization in a neurology or neurosurgery department and duration of hospitalization were also evaluated.

Groups were compared using Pearson chi-square and Fisher exact tests. Further statistical analyses were performed on the following clinical variables: (1) age, (2) location of fracture, (3) Glasgow Coma Scale (GCS) score upon arrival, (4) mechanism of injury, (5) intracranial bleeding status, and (6) duration of hospitalization. Each of these variables was placed in a univariate logistic regression analysis model. The dependent variable was whether or not the patient had experienced a CSF leak. The level of statistical significance was set at $P < 0.01$.

3. Results

In the 3 year period from January 2006 through December 2008, 107 patients who had suffered traumatic skull base fractures were admitted to Soroka Medical Center in Be'er-Sheva, Israel. Of these patients, 86 were men and 21 were women. Patients ranged in age from 18 to 90 years (mean age 41.93 ± standard deviation [SD] 19.13). Of this group, 68 patients had suffered a fall or been hit by a blunt object, 27 patients had been injured in a motor vehicle accident (14 as pedestrians), and three had been injured by a penetrating object. Table 1 summarizes the demographic and clinical characteristics of these patients.

Upon arrival, 76 of the patients (71%) had a mild head injury (GCS score of 14 to 15), 17 of the patients (16%) had a moderate head injury (GCS score of 9 to 13), and 14 of the patients (13%) were comatose. The mean GCS score upon admission was 13 ± SD 3.38. The correlations between GOS scores and admission factors (GCS scores upon admission, duration of CSF leak, mechanism of injury and fracture location) are described in Table 2.

The average duration of hospitalization for patients in the mild head injury group was 5.8 days. All of the patients in this group were discharged from the hospital with a GOS score of 4 or 5. The mean score was 4.89.

The average duration of hospitalization for patients in the moderate head injury group was 10.3 days. In this group, the GOS scores at the time of discharge ranged from 1 to 5.

The GOS score was significantly worse in patients who arrived with severe head injuries ($P < 0.001$); their average duration of hospitalization was 20 days. Five patients died within 15 days after injury; two due to uncontrolled intracranial pressure, and three due to hemodynamic instability.

Upon arrival, 10 patients had focal neurological deficits: hemiplegia was seen in five patients, early peripheral facial nerve palsy was seen in three patients, and oculomotor and abducens nerve palsies were seen in one patient each. All three patients with early peripheral facial palsy were treated with steroids and improved, although at the time of writing they had not fully recovered (House Brackman grade 2, 4 and 5). The patient with oculomotor nerve palsy recovered fully, while the patient with abducens nerve paralysis did not.

The anatomical sites of the skull base fractures are shown in Figure 1. Most of the fractures (30%) were in the temporal bone; 20% were in the occipital bone, 20% were in the pyramidal bone, and 17% occurred in the frontal bone. Multiple skull base fractures

Table 1
Demographic and clinical characteristics of patients with skull base fractures

Variable	Category	Patients (%)
Age, years	18–40	53 (49)
	41–60	33 (31)
	61+	21 (20)
Sex	Male	86 (80)
	Female	21 (20)
Glasgow Coma score	3–8	14 (13)
	9–13	18 (17)
	14–15	75 (70)
Mechanism of injury	Penetrating	3 (3)
	Fall	68 (64)
	Car accident	13 (12)
	Pedestrian accident	14 (13)
	Other	10 (9)
Anatomical area of fracture	Frontal	18 (17)
	Occipital	21 (20)
	Pyramidal	21 (20)
	Temporal	32 (30)
	Multiple fractures	15 (14)
Type of fracture	Linear	80 (75)
	Depressed	27 (25)
Intracranial CT scan findings	Normal	36 (34)
	Small hemorrhages	44 (41)
	Subarachnoid hemorrhage	8 (7)
	Subdural hematoma	12 (11)
	Diffuse axonal injury	7 (7)
Duration of cerebrospinal fluid leak (days)	Nil	91 (85)
	1–3	7 (7)
	4+	9 (8)
Loss of consciousness	No	56 (52)
	Yes	51 (48)
Meningitis	No	103 (96)
	Yes	4 (4)
Glasgow Outcome score	Dead	5 (5)
	2	1 (1)
	3	3 (3)
	4	8 (7)
	Full recovery	90 (84)

Table 2
Correlations between admission factors and Glasgow Outcome (GOS) score

Mechanism of injury	Average GOS	
Fall	4.76	NS
Car accident	4.20	NS
Pedestrian accident	4.29	NS
Penetrating	4.67	NS
Glasgow Coma score		
3–8	3.21	P < 0.001
9–13	4.50	NS
14–15	4.91	NS
Cerebrospinal fluid leak		
No leak	4.74	NS
Early (0–1 day)	4.44	NS
Late (3–14 days)	3.29	P < 0.001
Anatomical area of fracture		
Frontal	4.67	NS
Temporal	4.81	NS
Pyramidal	4.81	NS
Occipital	4.81	NS
Multiple	3.60	P < 0.001

NS = not significant.