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Original Research Article

To study the association of CSF leak in fronotbasal skull fractures classified with the Burstein's classification

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ABSTRACT

Background: To study the association of CSF leak in Fronotbasal skull Fractures classified with the Burstein's Classification.
Methods: A prospective study was conducted from November 2014 to May 2016 in patients admitted with head injuries to KIMSDU, Karad, Maharashtra. All data was retrieved using a standardized data collection form.
Results: Out of the total 55 patients of frontobasal fracture, 39 (70.9%) were found to have CSF leak. Out of 39 patients with CSF leak 34 (61.8%) had Type I head injury, 3 (5.5%) had Type II head injury, and 2 (3.6%) had Type III head injury. Statistical analysis showed significant association between CSF leak and Burstein's classes of head injury patients ($p< 0.05$).
Conclusions: It was found that patients who had Burstein Type I injuries had a higher chance of CSF leak and most post traumatic leaks could be managed conservatively.

Keywords: Burstein's classification, CSF leak, Frontobasal fractures

INTRODUCTION

Frontalbasal skull fractures are a relatively common in neurosurgical practice. It results from high energy impact injuries to the forehead and often are associated with other injuries.

Burstein et al suggested the following classification system for frontobasal skull fractures according to the fracture patterns seen by CT of the region involved.[1]

Type I (central) fractures confined to the upper naso-ethmoid complex, central frontal squama and medial third superior orbital rim.

Type II (Unilateral) fractures involving the entire superior rim and upper lateral orbit wall.

Type III (bilateral) fractures involves fractures of the upper nasal ethmoid complex, bilateral supraorbital and upper lateral orbital wall as well as bilateral frontal squama fractures.

CSF leaks are common in these fractures and has an estimated incidence of 1,50,000 per year in the united states.[2] The development of a persistent CSF fistula exposes the patient to a risk of meningitis and brain abscess development. The appropriate plan of treatment for these injuries is a subject of debate and controversy.[3] No data exists regarding the degree of frontalbasal skull fractures and association post traumatic CSF leak with it. The purpose of this study is to evaluate the incidence of CSF leak in different types of Frontal Basal skull fractures and establish guidelines for treatment of frontobasal skull fractures.

METHODS

This prospective study was conducted in the Department of Neurosurgery, KIMSDU, Karad from November 2014 to May 2016. All patients with Frontbasal skull fractures admitted to the Neurosurgery ward and intensive care unit were included in the study. All cases had been subjected to proper history taking. Patient information such as name, age, sex, occupation, income, socio-economic class, total family members and any medical past history of relevant importance are obtained from questionnaire method. Details about place and time of accident, time of arrival at hospital, cause of accident, type of vehicle used, influence of alcohol and Glasgow coma score, Type of head injury, CSF leak, CT head were noted on a standardized form.

Statistics

Statistical software SPSS version 20 was used for data analysis. Proportions and percentages were calculated. Variables were compared by performing chi-square test for small numbers, Fishers exact test was applied whenever applicable. P-value <0.05 was considered as statistically significant.

RESULTS

Study included 55 patients of head injury. All the patients were screened for the presence of frontbasal fracture and CSF leak. 39 (70.9%) patients had CSF leak whereas 16 (29.1%) patients had no CSF leak. Statistical analysis done for different variables among patients.

Patients were classified according to Burstein's classification into three groups as Type I, Type II and Type 3. Out of 55 patients, 43 patients (78.2%) classified as Type I, 8 patients (14.5%) as Type II and 4 patients as Type III.

Table 1: Distribution of patients according to Burstein's classification.

Burstein classification	No. of patients
Type 1	43 (78.2%)
Type 2	8 (14.5%)
Type 3	4 (7.3%)
Total	55 (100%)

Out of 55 patients of head injury, 39 patients (70.9%) had evidence of CSF leak. Out of 39 patients with CSF leak 34 (61.8%) had Type I head injury, 3 (5.5%) had Type II head injury, and 2 (3.6%) had Type III head injury. Statistical analysis showed significant association between CSF leak and Burstein's classes of head injury patients ($p< 0.05$).

Out of 39 patients of CSF leak, 20 patients (51.3%) had CSF leak for 1 to 5 days, 12 patients (30.8%) had CSF leak for 6-10 days, 7 patients (17.9%) had CSF leak for > 10 days.

Table 2: Distribution comparison with Khalid study.

Burstein classification	Present study	Khalid' study
Type 1	78.2%	60%
Type 2	14.5%	22%
Type 3	7.3%	9%

Out of 39 patients with CSF leak 21 (53.8%) were treated conservatively, 12 (30.8%) required only a craniotomy and exteriorization of the frontal sinus, 5 (12.8%) required a lumbar drain placement. Only 1 (2.6%) patient was first treated conservatively failing which an external lumbar drain and later a craniotomy was needed.

At the time of discharge out of 55 patients of head injury, 48 patients had Glasgow coma score of 15 (complete recovery). Out of 43 patients who had Glasgow coma score on admission between 12-15, maximum number of patients (95.3%) had complete recovery while 2 patients (4.7%) had mild neurological deficit. Out of 9 patients with Glasgow coma score on admission was between 7-11, 5 patients (55.6%) had complete recovery, 3 patients (33.3%) had mild neurological deficit and 1 patient had moderate neurological deficit on discharge. Out of 3 patients with Glasgow coma score on admission < 7, 2 patients (66.7%) improved completely and one patient (33.3%) had minimal neurological deficit. Statistical analysis showed that association between Glasgow coma score on admission and discharge was highly significant. ($p< 0.05$) Patient with low Glasgow coma score on admission showed significant improvement after treatment i.e. improved Glasgow coma score on discharge. No deaths occurred in the study.

Table 3: Outcome of head injury patients.

Outcome	CSF	Leak
	Yes	**No**
Complete cure	35 (63.7%)	13 (23.6%)
Incomplete cure	4 (7.2%)	3 (5.5%)
Total	39 (70.9%)	16 (29.1%)

Out of 39 patients of head injury with CSF leak, 35 patients (89.7%) completely recovered (GCS 15), 3 patients (7.7%) had mild neurological deficit (GCS 12-14), 1 patient (2.6%) had moderate neurological deficit (GCS 7-11).

DISCUSSION

Frontbasal skull fractures are a challenging neurosurgical problem. 80% of CSF fistulas result from head injuries with skull base fractures.[4] Treatment of these injuries is still subject to many controversies. To the best of our knowledge there are few Indian population studies based on the incidence of CSF leak in frontobasal skull

fractures and its relationship with the Burstein's classification system. In our study, 55 patients of frontobasal skull fracture were classified clinically and according to CT scan findings. Clinically patients were classified as open and close skull fractures. Burstein's classification system was used to classify patients according to CT scan findings.[1]

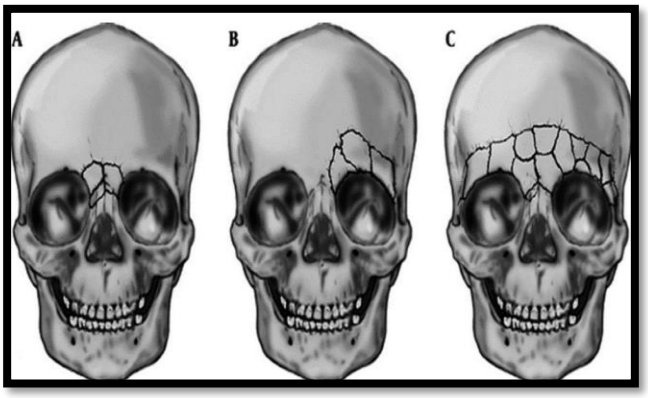

Figure 1. Diagrammatic representation of Bursteins classification; A- Type I, B- Type II, C- Type III.

According to Burstein's classification out of 55 patients, 43 patients (78.2%) had Type I fractures, 8 patients (14.5%) had Type II fractures, and 4 patients (7.3%) had Type III fractures. In Khalid et al study 60% classified as Type I injury, 22% classified as Type II injury and 9% classified as Type III injury.[5] Our observations were in agreement with study, suggesting that maximum number of patients were found to have Type I frontobasal fracture.

In our study total 39 patients (70.9%) had CSF leak in the form of either rhinorrhoea or otorrhoea and 5 patients (9.1%) had ENT bleeding. Out of 39 patients of CSF leak, 38 had CSF rhinorrhoea and 1 had CSF otorrhoea. In Khalid et al study 27 patients (54%) patients had CSF leak.[5] In Velho et al study, 32 patients had CSF leak at presentation.[6] In Shisoka et al study 13 patients (13.5%) had CSF rhinorrhoea and 11 patients (11.5%) had CSF otorrhoea.[7] In Bell et al study 34 patients were identified with CSF leak presenting as otorrhoea (25 patients) or rhinorrhoea (9 patients).[8]

Out of 39 patients with CSF leak 34 (61.8%) had Type I head injury, 3 (5.5%) had Type II head injury and 2 (3.6%) had Type III head injury. Statistical analysis showed significant association between CSF leak and Burstein's classes of head injury patients ($p< 0.05$). Type I head injury patient had maximum cases of CSF leak. The severity of the leak is also not always proportional to the size of the dural tear. Usually the leak is through a dural tear which is associated with a fracture of the anterior cranial fossa involving a paranasal air sinus or the cribriform plate of ethmoid. Samii and Draf are of the opinion that most leaks occur in the adjacent ethmoid roof.[9] The next common sites for the fistulas are fractures of the posterior wall of frontal sinus and those involving the sphenoid sinus (tuberculumsella and anterior wall of the sella rather than the planumsphenoidale).

Out of 39 patients 37 patients had CSF leak at presentation and 2 patients developed CSF leak on second day. In Velho et al study 32 patients (15%) had CSF leak on presentation.[6] G. Rocchiet al study rhinorrhoea occurred immediately after trauma in 30 cases and in following days (1-5) in 6 cases.[10] In 70% CSF leak occurs within 48hrs of the injury. The CSF leak will clinically be obvious in 98% within 3months. The CSF leak ceases spontaneously in 70% during the first 7days. In most cases CSF leak will stop within 6months.

Out of 39 patients of CSF leak 20 patients (51.3%) had CSF leak for 1 to 5 days, 12 patients (30.8%) had CSF leak for 6-10 days, 7 patients (17.9%) had CSF leak for > 10days. In Bell et al study twenty-eight patients (84.6%) experienced uncomplicated resolution of the leak without treatment in 2-10days.[8] Mincy reported that spontaneous closure occurs in 68% of posttraumatic CSF fistulas within 48hours of injury and 85% within 1week.[11]

Out of 39 patients with CSF leak 21 patients (53.8%) were managed conservatively, 5 patients (12.8%) managed by external lumbar drainage and 12 patients managed by surgical repair. 1 patient was managed by both external lumbar drainage and surgical repair. In Khalid et al study 22 (44%) patients managed conservatively while 28 patients (56%) managed surgically.[5] In Bell et al study 28 patients (84.6%) conservatively, 6 patients (0.8%) underwent CSF diversion via a lumbar drain for a period of 5 to 10 days.[8] Two of these patients were treated successfully; the remaining 4 patients required surgical procedures. In our study bed rest, elevating head 300, stool softeners and acetazolamide are used as conservative treatment. The patient was given antibiotics and is cautioned not to sniff or blow his nose.

The nose or ear covered with a light dressing, but the orifice was not plugged tightly. Simple or minimally depressed fracture without clinical or radiological evidence of CSF leak were managed conservatively (n=16) with antibiotics and anticonvulsants depending on the extent of brain parenchymal damage. Profuse CSF leaks and rhinorrhoea which persisted for more than 5days were managed by CSF diversion via lumbar drainage for 5 to 7days. Early operation was performed in patients with compressive hematoma, open trauma, severe bone derangement and profuse CSF leak. Fractures associated with CSF leak or presence of significant contusion and significant edema or involvement of frontal sinus was promptly explored. The key step during surgery was preservation of pericranium, exposure of base to delineate fractures, removal of depressed fragments, thorough debridement of dead and devitalized tissue, generous wash followed by exteriorization of frontal sinus, and followed by brain

isolation using water-tight dural closure. A majority of patients in our study had frontal sinus injury that was dealt with exteriorization. This included removal of the mucosa of frontal sinus, rinsing it with bactericidal solution, packing it with fat from thigh, gelfoam mixed with chloramphenicol followed by covering it with vascularized pericranial graft that was harvested at the beginning of the surgery or tensor fascia lata from thigh. Decision regarding bony reconstruction was made depending on the extent of contamination, underlying brain damage and the extent of bony damage and loss. The management of basal skull fracture is usually determined by the presence or absence of a CSF leak. A patient with a basal skull fracture but no initially noted leak is managed by observation for 2 to 3days. During this time repeated checks for rhinorrhea or otorrhea are made to verify the absence of a CSF leak.

The selection of patients who do or do not require surgical treatment, timing of surgery and antibiotic prophylaxis are questions widely debated in the neurosurgical literature. Most neurosurgeons do not follow the advice of Loewet al and Cairns that all CSF fistulae should be treated surgically as soon as possible, but suggest initial conservative treatment.[12,13] The rationale of this suggestion is based on the observation that 50% to 85% of traumatic CSF fistulae occurring within 48hours after injury cease spontaneously.[14-16] However spontaneous cessation of CSF leakage does not guarantee that the dural tear is definitely sealed and recurrent rhinorrhoea or late intracranial infections may develop.[12,17,18] Delayed or recurrent CSF rhinorrhoea almost never stops without operative treatment and the risk of meningitis becomes high.[17,16]

Nevertheless reports which suggestive of spontaneous cessation of a CSF leak was possible led some to think that surgical intervention was unnecessary and unproven.[19,20] Calvert and Cairns in a discussion of war injuries to the frontal and ethmoidal sinuses, described a number of cases in which CSF rhinorrhoea healed spontaneously.[20,21] Only 3 (33%) of the 9 patients with posttraumatic CSF rhinorrhea did not respond to any of the nonsurgical measures and required surgical intervention to close the CSF leak. Mincy reported that spontaneous closure occurs in 68% of post-traumatic CSF fistulas within 48hours of injury and 85% within 1week.[11]

Out of 39 patients of head injury with CSF leak 35 patients (89.7%) were completely recovered (GCS 15). 3 patients (7.7%) had mild neurological deficit (GCS 12-14); 1 patient (2.6%) had moderate neurological deficit (GCS 7-11). 31 patients who had Glasgow coma score on admission between 12-15 maximum number of patients (96.8%) had complete recovery while 1 patient (3.2%) had mild neurological deficit. Out of 6 patients with Glasgow coma score on admission between 7-11, 3 patients (50%) had complete recovery, 2 patients (33.3%) had mild neurological deficit and 1 patient had moderate neurological deficit on discharge. 2 patients with Glasgow coma score on admission <7 improved completely. Statistical analysis showed that association between Glasgow coma score on admission and discharge was significant ($p<0.05$). Patient with low Glasgow coma score on admission showed significant improved after treatment (i.e. improved Glasgow coma score on discharge).

CONCLUSION

Frontobasal skull fractures is a complex pathology. They can be classified based on various classification systems. It was found that patients who had Burstein Type I injuries had a higher chance of CSF leak. Most acute post-traumatic CSF leaks stop spontaneously within 10days of injury. Bed rest, elevating head 300, stool softeners, acetazolamide, antibiotics and anticonvulsant are used as conservative management. Lumbar drainage used for profuse CSF leak without any other significant intracranial complications. Surgery is reserved for the treatment of CSF leaks that do not stop spontaneously or respond to conservative management with CSF diversion.

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Ethical approval: The study was approved by the institutional ethics committee

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