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Aetiology and Treatment Outcome of Severe Traumatic Brain Injuries in Neurosurgical Center with Inadequate Facilities

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Background: ***Severe traumatic brain injury (TBI) is a major challenge to the patient, the relatives, the care givers, and the society in general. The primary and secondary injuries, and the high metabolism are formidable stages of the injury, each capable of taking the life of the patient. The objectives were to determine the etiology and outcome in severe traumatic brain injuries patients.***
Methods: ***This was a prospective study on patients with severe traumatic brain injury patients managed in our center from August 2010 to December 2014. Patients were resuscitated in accident and emergency unit using Advanced Trauma Life Support protocols. Those with Glasgow Coma scale scores ≤8 were included in the study.Data were collected with structured proforma which was component of our prospective data bank that was approved by our ethics committee, and were analyzed with Environmental Performance Index info 7 software***
Results: ***One hundred and ten patients were studied. Males were 86. The mean age Was 31.43 years. Ninety six were involved in road traffic accident. Seventy seven patients had favorable outcome.***
Conclusion: ***The commonest etiology was road traffic accident.Seventy seven patients had favorable outcome.***

Keywords: etiology, outcome, severe traumatic brain injury

Introduction

Severe traumatic brain injury (TBI) is a big challenge to the patientsand the society [1, 2]. It leaves patients with physical, mental and cognitive deficits with direct and indirect consequences [3, 4], besides reducingtheir live expectancy [5]. Mortality had fallen by 50% since1885 but had been static since 1990 [6]. Successes gained from new investigative modalities, improved resuscitation and prompt surgery werecountered by increased mortality from high velocity vehicle crashes,[7]raising mortality among elderly [8], and poor outcomes from pre-hospital intubation[9] and intracranial pressure monitoring [10,11].

We studied the aetiology and treatment outcome in severe TBI patients managed in our center.

Patients and Methods

It was a prospective, descriptive and cross-sectional study on severe traumatic brain injury (TBI) patients managed in our center from 1st August, 2010 to 31st December, 2014. Our neurosurgical center started in July 2010 with no functional intensive care unit. The intensive care unit became functional in February 2012 with only five beds serving the whole Teaching Hospital. Few months later, the intensive care unit started having problems with cooling system, monitors and ventilators, and eventually became epileptic functionally which persisted till the time of writing this manuscript. Because of these many patients requiring intensive care were managed in the wards as our nearest neurosurgical center, about 400km away, was suffering from the same problems. The effect of this was many severe traumatic brain injury patients not having the services of intensive care unit (ICU).

We used Advanced Trauma Life Support protocols to resuscitate our patients in accident and emergency unit. We used Normal Saline for adult and 4.3% Dextrose in 1/5Saline for children. We gave intramuscular (i. M.)Paracetamol 15mg/kg every eight hours for pain. We augment this with intramuscular (i.m.) Diclofenac 75mg every twelve hours in adult. They were giving oxygen via face mask, nasal prongs or endotracheal tube at 4-7liters/minute. Seizures were controlled using intramuscular (i.m) Paraldehyde at 0.1ml/kg/dose and Phenytoin infusion at 14mg/kg for adult and 21mg/kg in children as loading doses, and 300mg/day for adult and 5m/kg/day for children (max 300mg/day) daily. They were given in 100ml normal saline infusion over one hour. Intravenous (i.v.) Ceftriazone 1gm daily for adult and 50-100mg/kg/day for children were given to those with open wounds.Theirscores on the Glasgow Coma scale (GCS) were calculated after their resuscitation or prior to surgery.The scale measures three parameters: best motor response (1-6), best verbal response (1-5), and best eye opening (1-4).[12] The highest score is 15, while the lowest score is 3. Traumatic brain injury patients are classified into severe (3-8), moderate (9-12) and mild (13-15).Patients with GCS 3-8 after resuscitation (severe TBI) qualified for the study. Referred patients were also classified as severe TBI if they had Glasgow Coma Scores ≤8 after resuscitation at the referring centers, and were included in the study. They were investigated, including brain computerized tomography (CT) scan, and those having surgical lesions were operated.

Those without surgical lesions and those who could not afford brain CT scan were managed conservatively. We commenced enteral feeding via nasogastric tube on third day post-injury using high energy/high protein diet constituted thus: 500ml pap, two tablespoonful soya bean powder, two tablespoonful powdered milk, one tablespoonful crayfish powder, and one tablespoonful red oil. The diet was given five to six times daily and their daily fluid requirements were factored in the diet. Tablets of multivitamin, Encephabol and vitamin B complex were given one tablet each three times daily via the nasogastric tube.

We monitored intracranial pressure (ICP) clinically using Cushing reflex (increasing blood pressure and reducing pulse rate), pupillary size/reactivity, and deteriorating level of consciousness. Increasing ICP was medically managed using mannitol and short positive pressure ventilation (PPV) as prolonged PPV use can cause cerebral

ischemia.[13] We didnot use PPV first day of injury due to reduced blood supply to the brain in first 24hours.[14]

When the ICU was not functioning, patients were monitored in the wards.The house officers in the unit share unit patients among themselves and compete among themselves on who would have best outcome, especially least mortality. They become extra vigilant whenever they have severe head injury patients, checking regularly for abnormal changes in physical signs and ensuring that the patients receive their diet and medications promptly.

Patients in ICU were transferred to the wards when their GCS improved to 10 and had good airway control. Associated major organ injuries were managed by respective specialist units. On discharge, the patients were followed up in surgical out-patient clinic.

Data were collected using structured proforma which was component of our prospective data bank approved by our hospital's ethics and research committee. In accident and emergency unit we collected biodata, time of injury, time-lag to presentation, direct or referred, etiology, pupillary sizes/reactivity, GCS after resuscitation in accident and emergency and at referring centers, and CT scan findings. In theater, GCS prior to induction, the type of surgery and findings were documented. The progress of the patients in the ICU and wards were documented. On discharge, the length of stay was calculated and documented.

Their functional outcomes were measured with Glasgow Outcome Score (GOS) [15]. It classifies patient into 5 categories:

- death (1),
- vegetative state (2),
- severe disability (3),
- moderate disability (4), and
- good recovery (5).

At the out-patient clinic, the GOS were documented at three months post-injury. Those who were still in the wards at three months post-injury,had their GOS documented there. For those who missed their appointment at three months post-injury, we contacted them or their relatives on phone to ascertain their GOS. Patients we did not know their GCS score after resuscitation (mainly referred ones) and those who did not attend clinic at three months and we could not access them or their relatives through phone were excluded from the study.

Data were analyzed using Environmental Performance Index (EPI) Info software (Center for Disease Control and Prevention, Atlanta, Georgia, USA, EPI info 7 version 7.0.8.0 of 2011). We used analysis gadgets of the visual dashboard method to analyze the data. Frequency component was used to determine the frequency of variables like etiology; the mean component for age and hospital stay. The MxN/2x2 component was used for univariate analysis, while its advanced component was used for multivariate analysis. At 95% confidence interval, P<.05 was considered significant.

Results

There were one hundred and ten patients in the study. Eighty six were males while 24 were females. Their ages ranged from nine months to 76 years with mean age of 31.43 years. The 20 - 30 and 30 - 40 years age groups had the highest frequency of 28 each (Table 1), and both formed 50.9% of the patients. The most common cause was road traffic accident (RTA), (Figure 1).

Table 1. Age Group Frequency

Group (years)	Number	Percent (%)
0 - <10	14	12.73
10 - <20	10	9.09
20 - <30	28	25.45
30 - <40	28	25.45
40 - <50	12	10.91
50 - <60	11	10.00
60 - <70	3	2.73
70 - <80	4	3.64
Total	110	100

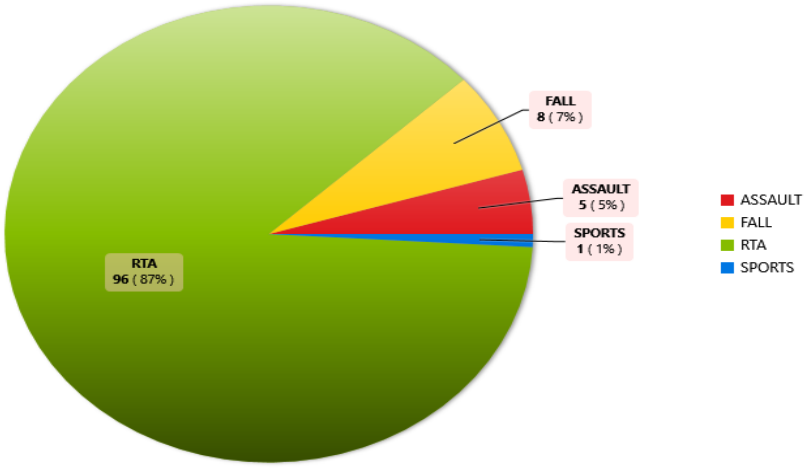

Figure 1. Distribution of the Causes of Severe TBI

A total of 84 (76.4%) of the patients were referred to us while 26 patients came direct from the trauma scene. Only 42 (38.2%) of the patients presented within 24 hours of the trauma (Table 2). In all, 56 (50.9%) of the patients had ICU care while 54 did not have ICU care. Patients with GCS 8 had the highest frequency (Table 3).

Sixty nine patients (62.7%) had brain CT scan, while 41 could not afford the investigation. The commonest lesion on the CT scans was diffuse axonal injury, Table 4.

Thirty patients had skull fractures (skull x-ray and CT scan combined). Ninety two patients were managed conservatively, while 18 patients had surgical care: craniotomy for extradural, subdural and intracerebral hematomas and craniectomy with primary bone replacement for depressed skull fractures. The hospital stay ranged from one day to 97 days with mean of 22.57 days and median of 16 days.

The overall favorable functional outcome (GOS ≥4) was 71.9% (77) with mortality of 27.27%(30). The GCS after resuscitation significantly affected the outcome, P =.01, (Table 5). The mortality among patients who were not admitted in ICU was 37.04% (20/54).The outcome among those admitted in ICU was significantly better than those who were not admitted in ICU,P=.04, table 6. The age of patients did not affect the outcome significantly, P= .26. Time lag at presentation did not have effect on the outcome P=.43. Intracranial lesions did not have effects on outcome P= .21.

Table 2. Period Presented Frequency

Presentation period (days)	Number	Percent (%)
0-1	42	38.18
>1-3	34	30.91
>3-7	20	18.18
>7	14	12.73
Total	110	100

Table 3. GCS after Resuscitation Frequency

GCS score	Number	Percent (%)
3	11	10
4	7	6.36
5	13	11.82
6	15	13.62
7	30	27.27
8	34	30.91
Total	110	100

Table 4. Intracranial CT Finding Frequency

CT Lesions	Number	Percent (%)
Contusions/ICH	16	23.19
DAI	22	31.88
Edema	6	8.70
EDH	5	7.25
None	1	1.45
Others	3	4.35
SDH	16	23.19
Total	69	100

Key: ICH = intracerebral hematoma, DAI = diffuse axonal injury, EDH = extradural hematoma, SDH = subdural hematoma

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Table 5. GCS vs GOS

GCS	Glasgow Outcome Score				
	1(%)	3(%)	4(%)	5(%)	Total (%)
3	6(54.55)	0(0)	0(0)	5(45.45)	11(100)
4	4(57.14)	1(14.29)	0(0)	2(28.57)	7(100)
5	7(53.85)	0(0)	2(15.38)	4(30.77)	13(100)
6	2(13.33)	1(6.67)	3(20.00)	9(60.00)	15(100)
7	8(26.67)	0(0)	1(3.33)	21(70.00)	30(100)
8	3(8.82)	1(2.94)	4(11.76)	26(76.47)	34(100)
Total	30(27.27)	3(2.73)	10(9.09)	67(60.91)	110(100)
P= 0.01					

Table 6. ICU admission VS GOS

Admitted	GOS				
	1(%)	3(%)	4(%)	5(%)	Total (%)
Yes	10(17.86)	3(5.36)	7(12.50)	36(64.29)	56(100)
No	20(37.04)	0(0)	3(5.56)	31(57.41)	54(100)
Total	30(27.27)	3(2.73)	10(9.09)	67(60.91)	110(100)
P= 0.0407					

Discussion

We had 110patients in our study and 78.18% were males. Their ages ranged from 9 months to 76 years (mean 31.34 years)with 50.91% between 20 and 40 years.The most common etiology was RTA, 87.27%. In their study of early indicators of prognosis in 846 cases of severe traumatic brain injury, Jiang et al.[16] found that males formed 78.96%. The ages of the patients ranged from 3months to 87 years with patients 16-45 years forming 68.09%. RTA formed 73.14% of the etiologies. Lyle et al [17] in their study of clinical course and outcome of 159 severe head injury patients in Australia found that majority were males with a ratio of 2.7:1 to females. Their mean age was 30 years and 74% were under 40 years with RTA constituting 65% of the etiological factors. These studies with ours are almost similar portraying the workforce of these nations on the move trying to provide for the nation, and getting involved in road traffic accident in the process.Adeolu et al [18] in their study of etiology of headinjuries in Southwest Nigeria also found that majority (39.8%) were between 20-40 years. Emejulu et al [19] in their study of traumatic brain injury in accident and emergency found that 79.2% were males and majority (40.2%) were 20-40 years old. They were similar to our findings.

Most of the patients, 76.36%, were referred to us and only 38.18% presented within 24 hours, rising to 69.09% by the third day. However, only 50.91% of the patients had ICU admission. Andriessen et al [20] in their multi-center study of moderate and severe traumatic brain injuries in trauma systemsetting, found that all their 508 patients were seen within 72hrs and 86% were referred. Among 339 patients with severe traumatic brain injury, 83% had ICU admission. We did not have trauma system and some of our patients came as far as 600km away, necessitating their being resuscitated in the local

hospitals before embarking on about six hour journey to our center on roads laden with potholes. Most of the time the relatives had to meet to raise money before embarking on the journey. Lack of functional ICU when we started, being few bedded when it became functional, and subsequently functionally epileptic depicts the harsh reality of starting neurosurgical center in rural areas of developing countries.

In our study, 69 patients (62.73%) did CT scan of the brain. The commonest lesion was diffuse axonal injury (31.88%). In Jiang et al.[16] study, all their 846 patients did CT scan. The commonest lesion in theirs was contusion (67.85%). In King et al [21] study, all their 159 patients did CT scan and the most common intracranial lesion was diffuse axonal injury (40%). The lower percentage of CT scan done among our patients showed both poverty and lack of organized health system as we had neither universal health insurance nor trauma system. The high percentage of diffuse axonal injuries (DAI) and contusions (features of DAI) in these studiesshowed the extent of parenchymal injuries in severe traumatic brain injuries.

The length of hospital stay in our study ranged from one day to 97 days with mean of 22.57 days. Tokutomi et al [22] in 797 patients they studied found that length of hospital stay ranged from seven to 92 days with mean of 43 ± 22days which was almost similar to ours. Lengthy days in hospital depicted the severity of injury and slow pace of recovery in severe traumatic brain injury patients.

The overall favorable functional outcome among our patients was 71.9% and mortality was 27.27%. GCS at presentation affected the outcome. Those who had ICU care had better outcome, 76.79% and mortality of 17.86%, while those without ICU care had worse outcome, 62.97% and 37.04% respectively. Tokutomi et al [22] in 797 severe traumatic brain injury they studied found favorable outcome in 31.1% and mortality of 49.7%. Jiang et al [16] in 846 patients they studied found 45.63% favorable outcome at one year and mortality of 29.43%. They also found that GCS at presentation significantly affected the outcome. Other authors found GCS significantly affected outcomes.[23]Lyle et al[17] in 159 severe head injury patients they studied, found favorable outcome of 42% at two years and mortality of 51%. Leijdesdorff et al [24] in their 1250 traumatic brain injury patients discovered that the mortality among 179 patients with severe brain injuries was 31.1%. The rest they lumped together as survived.

Walder et al[25] in 921 patients with severe traumatic brain injuries in Switzerland found 14 day mortality of 30.2%. Stein et al [6] in their review of 150 years of treating severe traumatic brain injury extending from 1885 to this period, found that mortality had remained steady at about 35% from 1990 to date. They noted that the expected improvement due to modern modalities of care had been countered by the increasing population of elderly who had been associated with increase in mortality from traumatic brain injury. While the 35% might be average for above studies, only Jiang et al.[16] and ours were sub-thirty percent. This reflected on the demographics of both studies. Only 8.27% of the patients were above 66 years in Jiang et al [16] study and only 6.37% of our patients were above 60 years, unlike high percentage of elderly in others. In our study the mortality among those above 50 years was high but their effect was not much on overall mortality due to their low number, showing why age did not affect the outcome significantly in both studies. Low percentages, 3.3% [26], 3.8% [18], and 8.9% [19], of older people among traumatic brain injury patients in Nigeria had been documented.

The result of those who had intensive care was markedly better with mortality of 17.86%. This is where current modalities of care could have improved the outcome in many countries but for their aging population. In addition, our monitoring, especially our house officers, ensured that many factors that could cause secondary brain injuries were detected early and treated.

The ICU staff and our ward nurses played significant roles in this regard. The frequent review of our patients kept the nurses on their toes. A senior nurse told us one day that her nurses were quarrelling because of neurosurgical team; that a nurse took vital signs of our patients and failed to document them. Other nurses started quarrelling with her for that. They informed the nurse that the neurosurgical team was always around, checking their patients and the documentation of the nurses. We told her that reminding her should not cause quarrel since the nurses and us work together to achieve the same goal of better outcome; that quarrel disrupts unity in any team. Our care was in consonance with Haddad et al.[27] in their critical care management of severe traumatic brain injury in adults, where they stated that 'the management of severe traumatic brain injuries centers on meticulous and compressive intensive care that includes multi-model, protocolized approach involving careful hemodynamic support, respiratory care, aimed at preventing secondary brain insults, maintaining an adequate CPP, and optimizing cerebral oxygenation.

We avoided invasive ICP monitoring, relying on our clinical acumen as recent research on invasive ICP monitoring showed that it was associated with worsening of survival [10,11]. In the Cochrane database, systemic review found no randomized controlled trial that can classify the role of ICP monitoring in acute coma whether traumatic or non-traumatic [11]. We also avoided Decompressive craniectomy. Cooper et al [28]. in a prospective, randomized controlled trials found that it was associated with decreased intracranial pressure and length of stay in ICU compared with standard care, but it was also associated with more unfavorable outcomes. They also found thatthe rates of death at 6 months were similar, 19% for decompressive craniectomy and 18% for standard care. Probably that was why we did not have patient in vegetative state.

We commenced enteral high energy/high protein diet on the third day post-injury. The demolition phase of wound healing starts third day post-injury. It is a period of hypermetabolism, anabolism and catabolism, with catabolism more than anabolism. All these are triggered by cytokines released by body tissues in response to trauma and the magnitude is directly proportional to the severity of the trauma[29]. In severe traumatic brain injury hypermetabolic and catabolic state that increased systemic and cerebral energy requirement had been documented[30]. With glycogen store lasting less than 24 hours, the body would resort to gluconeogenesis from proteolysis and lipolysis orchestrated by interleukin-1, adrenaline, noradrenaline, growth hormones and other cytokines products, and growth factors. This is associated with suppression of humoral and cellular immune response. These could be averted by appropriate feeding.[28] Studies had shown that early feeding provided exogenous substrates, protected visceral protein and fat, improved immune competence, promoted neurological recovery, and decreased fatalities in surgical and injured patients[31, 32].

Chiang et al [33] in their comparative study of early enteral fed 145 patients and non-fed 152 patients with severe head injuries found that the outcome of early enteral fed

patients were significantly better than those not fed. In the study of the effect of early nutrition on deaths due to severe traumatic brain injury, Hârtl et al. [34] found that two-week mortality based on nutritional status was significantly higher among patients who were never fed within five days (P=0.0008) or seven days (P= 0.0001). Our early feeding of our patients, therefore, must have contributed to our outcome.

Conclusion

The study showed that the most common cause of severe traumatic brain injury was road traffic accident. The favorable functional outcome was 71.9% and overall mortality was 27.27%. The outcome of patients managed in ICU was significantly better than those without ICU care. The GCS score after resuscitation significantly affected the outcome. Adequate and early nutrition is essential in severe traumatic brain injury.

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