

TRAUMATIC BRAIN INJURY IN ADULTS: AGE, ETIOLOGY, AND TREATMENT OUTCOME

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Abstract

Traumatic brain injury had been described as silent epidemic. The etiology is said to be changing and older adults have been said to have worse outcome compared to the younger ones. We prospectively studied adults managed for traumatic brain injury in our center over a four and half year period.

Objectives: To determine the effects of age and etiological factors on the outcome of adults managed for traumatic brain injury.

Methods: It was a prospective study on adults managed for traumatic brain injury in our center from August 2010 to January 2015. Patients were resuscitated in accident and emergency using Advanced Trauma Life Support (ATLS) protocols. Biodata, history and physical examinations were done in accident and emergency. Severity of the injury was assessed using Glasgow Coma Scale. Comatose patients were admitted in intensive care unit (ICU), while orders were admitted in the wards.

Data were collected using structured Performa which was component of our prospective data bank that was approved by our ethics and research committee. Data were analyzed with Environmental Performance Index (EPI) info 7 software.

Results: Two hundred and eighty seven patients were studied. There were 244 males. Their ages ranged from 20 – 76 years. The most common etiology in all age groups was road traffic accident (RTA). The outcome was worse in older patients.

Conclusion: We found that more males had traumatic brain injury. The most common etiology in all ages was RTA. The outcome was significantly affected by age. Older patients had worse outcome.

Keywords: age, etiology, outcome, traumatic brain injury

Introduction

Traumatic brain injury (TBI) is heterogeneous in terms of pathophysiology, clinical presentation, and outcome with case fatality rates ranging between <1% in mild traumatic brain injury up to 40% in severe traumatic brain injury.^[1, 2] Traumatic brain injury was reported to have an estimated annual incidence of up to 500/100,000 population and more than 200 hospital admission/100,000 admissions in Europe each year.^[3, 4] Age had been reported to play important role in determining the outcome and many series indicated age as an independent predictor of worse outcome after adjusting for co-variables including co-existing diseases.^[5-7] Older adults were said to have worse outcome compared to the younger adults but the threshold value from many studies ranged from 30 years to 60 years of age.^[8-13] Few studies had analyzed the association between age and outcome in a continuous way and reported a change around age 30-40 years, above which outcome became increasingly poorer, and a fairly continuous relation across all ages, which might be approximated by a linear function.^[6, 14] Some authors identified two ages as thresholds associated with worse outcomes, older than 26 years and older than 60 years.^[15] The etiology of traumatic brain injury among older adult was reported to be changing with falls rising in many studies.^[16-18]

We prospectively studied the effects of etiological factors and age on outcome among adult patients managed for traumatic brain injury in our center over a four and half year period.

Materials and Methods

It was a prospective and observational study among adult patients managed for traumatic brain injury in our center from August 1st 2010 to January 31st 2015.

Inclusion Criteria: Patients aged 20 years and above managed for traumatic brain

injuries in our center within the period whose data were complete to three months after the injury.

Exclusion criteria:

All patients below 20 years of age. Patients who left the hospital against medical advice or ran away from the hospital. Patients we could not ascertain their outcome status three months after injury; those who failed to attend the clinic at three months post-injury and we could not get them on phone.

Methods:

Patients were resuscitated in accident and emergency using ATLS protocols. We aimed at normotension and euolemia, using Normal saline. We gave oxygen via face mask or nasal catheter/prongs or endotracheal tube at 4-7l/minute to ensure oxygen saturation of 95% and above. For analgesia, we used intramuscular (i.m.) Paracetamol 1gm every 8 hours. We added i. m. Diclofenac 75mg 12 hourly in some cases. For patients with open wounds we gave i. m. Tetanus toxoid 0.5ml and intravenous (i. v.) Ceftriaxone 1gm once daily. Unconscious patients and those in shock were catheterized and urine output monitored. Full history and physical examinations, including Glasgow Coma Scale (GCS) scores were documented. Investigations, including cranial Computerized Tomography (CT) scan (those who afforded), were done. Those whose GCS scores were ≤ 8 were admitted in intensive care unit (ICU). Those with higher GCS scores were admitted in the wards. Patients requiring surgery were operated and sent to ICU or wards depending on the status of the patient. Unconscious patients were given high energy/protein diet from the third day post-injury. The diet was constituted thus: pap 500ml, powdered milk 2 tablespoonful, soya bean powder 2 tablespoonful, crayfish powder 1 tablespoonful, and red oil 1 tablespoonful. The diet was given 5-6 times daily via

nasogastric tube. Daily fluid requirements of the patients were factored into the diet. Intravenous fluids were stopped once we achieved daily fluid requirement via nasogastric feeding. Their oral drugs were given via the nasogastric tube. We used multivitamin, Encephabol, Vitamin C, and B-Complex tablets, one each three times daily. On discharge, patients were followed up in out-patients' clinic.

Data were collected using structured Performa which was component of our prospective data bank that was approved by our ethics and research committee. The biodata, history and physical examination findings were documented in accident and emergency (A&E) unit. Investigation findings were documented in A&E or whenever available. Type of surgery and findings were documented in theater. Their progress were documented in wards and out-patient clinic. Their Glasgow Outcome Scores (GOS)^[19] were documented three months post-injury. Glasgow Outcome Score classifies patients into five groups: 1 dead, 2 vegetative state, 3 severe disability, 4 moderate disability, 5 good recovery.

Glasgow Outcome score at three months had been found to be predictor of long-term outcome.²⁰

The data were analyzed using Environmental Performance Index (EPI) info 7 software (Centre of Disease Control and Prevention, Atlanta, Georgia, USA). We used the analysis gadget of the visual dashboard to analyze the data. We used frequency component to analyze frequency of some variables. We used the mean component to analyze continuous variables like age. MXN/2X2 component was used for univariate analysis and its advanced component was used for multivariate analysis. At 95% confidence interval, $P < 0.05$ was considered significant.

Results

There were two hundred and eighty seven patients. Males were two hundred and forty four (85.02%), while females were forty three (14.98%). The ages ranged from 20 years to 76 years with mean age of 35.86 years. The most common age group was 20-29 years, 106 (36.93%), followed by 30 – 39 years, 90 (31.36%), table 1. The two groups formed 68.29%.

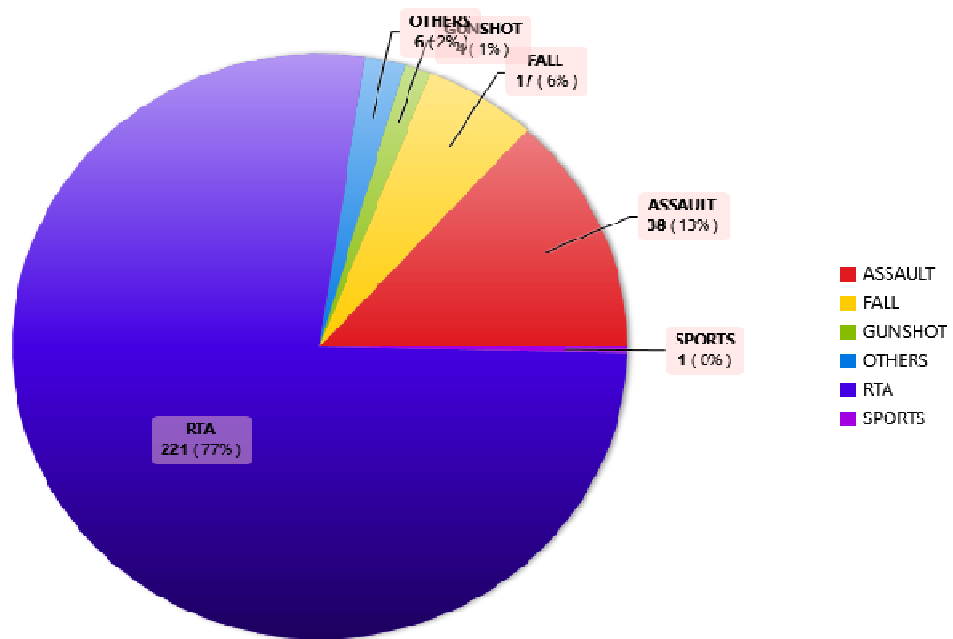
Table 1 age group frequency

Age group	Frequency	Percent (%)
20 - <30	106	36.93
30 - <40	90	31.36
40 - <50	51	17.77
50 - <60	22	7.76
60 - <70	11	3.83
70 - <80	7	2.44
Total	287	100

One hundred and six (36.93%) were 20-29 years, while those thirty years and above were 181 (63.07%) — **age group 2**. Two hundred and forty five patients (85.37%) were 20-49 years, while 42 (14.63%) were

50 years and above — **age group 3**. Two hundred and seventy four (95.47%) were 20-64 years, while 13 (4.53%) were 65 years and above — **age group 4** the most common etiology was RTA, 221 (77%), fig 1.

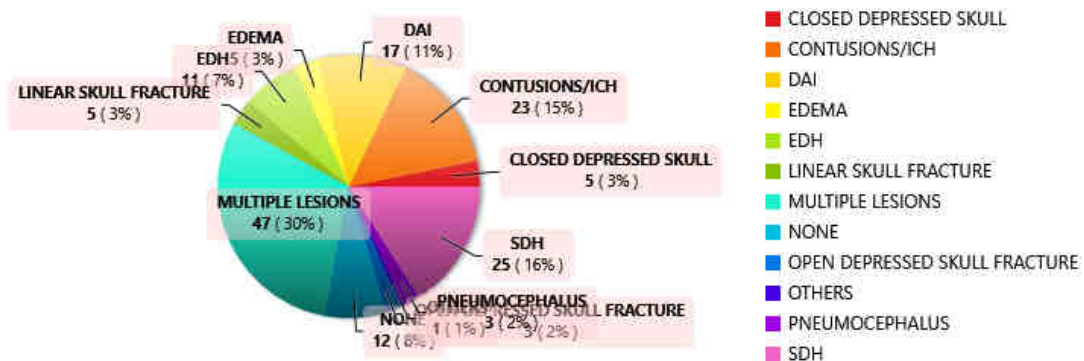
Fig 1 Etiology frequency.



The most common etiology among patients 50 years and above was RTA, 33 (78.57%), followed by fall with 5 patients (11.90%) and assault with 4 patients (9.52%). The most common etiology among patients 65 years and above was RTA, 13 (100%). Only two of the 13 patients 65 years and above had co-morbidity. There was no significant relationship between all the age groups (2, 3,

& 4) and etiology, $P = 0.2665, 0.3706, 0.5399$ respectively. One hundred and fifty eight patients had mild TBI, forty had moderate TBI, and eighty nine had severe traumatic brain injury. One hundred and fifty seven of the patients did cranial CT scan and patients with multiple lesions were most common, fig 2.

Fig 2 CT findings



Abbreviations: Intracerebral hematoma (ICH), diffuse axonal injury (DAI), extradural hematoma (EDH), subdural hematoma (SDH)

Among age group 2, sixty three of patients aged 20 – 29 years did CT scan. Lesions on their CT were multiple lesions 22 (34.92%), contusions/ICH 11 (17.46%), and the rest form the remaining 47.62%. Among those ≥ 30 years, ninety four did CT scan. The lesions seen were multiple lesions 25 (26.60), SDH 21 (22.34%), contusions/ICH 12 (12.77%) and the rest formed 38.29%. There was not significant statistical difference between the two groups, $P = 0.9768$. In age group 3, one hundred and thirty patients aged 20 – 49 years did CT scan. Lesions seen were multiple lesions 45 (34.62%), contusions/ICH 19(14.62%), DAI and SDH 14 (10.77%) each, and the rest formed 29.22%. Among patients that were ≥ 50 years, twenty seven did CT scan.

Lesions found were SDH 11 (40.74%), contusions/ICH 4 (14.81%), DAI 3 (11.11%) and the rest 33.34%. There was significant statistical difference between the two groups, $P = 0.0082$. In group 4, one hundred and forty seven patients aged 20-64 years did CT scan. Lesions found were multiple lesions 46 (31.29%), contusions/ICH 23 (15.65%), SDH 19 (12.93%), DAI 16 (10.88%), and the rest 29.25%. Among patients aged ≥ 65 years, ten did CT scan. Lesions found were SDH 6 (60%), EDH 2 (20%), and the rest 20%. There was also significant statistical difference between them, $P = 0.0344$.

The favorable functional outcome (GOS ≥ 4) was 88.85%, table 2.

Table 2 GOS frequency

GOS	Number	Percent (%)
1	30	10.45
3	2	0.70
4	22	7.67
5	233	81.18
≥ 4	255	88.85
Total	287	100

The etiology was not significantly related to outcome, $P = 0.9305$. Severity of injury was significantly related to outcome, $P = 0.000$, table 3.

Table 3 Injury severity vs. GOS

Injury severity (GCS)	Glasgow Outcome Score (GOS)					Total (%)
	1 (%)	3 (%)	4 (%)	5 (%)	≥ 4 (%)	
Mild (13-15)	2 (1.27)	0 (0)	5 (3.16)	151 (95.57)	156 (98.73)	158 (100)
Moderate (9-12)	3 (7.50)	0 (0)	5 (12.50)	32 (80.00)	37(92.50)	40 (100)
Severe (3-8)	25 (28.09)	2 (2.25)	12 (13.48)	50 (56.18)	62 (69.66)	89 (100)
Total	30 (10.45)	2 (0.70)	22 (6.67)	233 (81.18)	255 (88.85)	287 (100)
$P = 0.0000$						

Co-morbidity did not affect the outcome significantly, $P = 0.1774$. The age significantly affected the outcome, $P = 0.0064$, table 4.

Table 4 Age group 1 VS GOS

Age groups	GOS					
	1 (%)	3 (%)	4 (%)	5 (%)	≥4 (%)	Total (%)
20 - <30	8 (7.55)	0 (0)	8 (7.55)	90 (84.91)	98 (92.46)	106 (100)
30 - <40	10 (11.11)	0 (0)	5 (5.56)	75 (83.33)	80 (88.89)	90 (100)
40 - <50	2 (3.92)	1 (1.96)	4 (7.84)	44 (86.27)	48 (93.75)	51 (100)
50 - <60	6 (27.27)	1 (4.55)	2 (9.09)	13 (59.09)	15 (68.18)	22 (100)
60 - <70	1 (9.09)	0 (0)	1 (9.09)	9 (81.82)	10 (90.91)	11 (100)
70 - <80	3 (42.86)	0 (0)	2 (28.56)	2 (28.56)	4 (57.12)	7 (100)
Total	30 (10.45)	2 (0.70)	22 (7.67)	233 (81.18)	255 (88.85)	287 (100)

$P = 0.0064$

There was no significant statistical difference between patients 20-29 years old and those 30 years and above in terms of outcome, $P = 0.3765$. There was significant statistical difference in outcome between patients 20-49 years and those 50 years and above, $P = 0.0005$, table 5.

Table 5 Age group 3 vs. GOS

Age (years)	GOS					
	1 (%)	3 (%)	4 (%)	5 (%)	≥4 (%)	Total (%)
20 - 49	19 (7.76)	1 (0.41)	17 (6.94)	208 (84.90)	225 (91.84)	245 (100)
≥50	11 (26.19)	1 (2.38)	5 (11.90)	25 (59.52)	30 (71.42)	42 (100)
Total	30 (10.45)	2 (0.70)	22 (7.67)	233 (81.18)	255 (88.85)	287 (100)

$P = 0.0005$

There was significant statistical difference in outcome between patients 20-64 years and those 65 years and above, $P = 0.0083$, table 6.

Table 6 Age group 4 vs. GOS

Age (years)	GOS					
	1 (%)	3 (%)	4 (%)	5 (%)	≥4 (%)	Total (%)
20 - 64	26 (9.49)	2 (0.73)	19 (6.93)	227 (82.85)	246 (89.78)	274 (100)
≥65	4 (30.77)	0 (0)	3 (23.08)	6 (46.15)	9 (69.23)	13 (100)
Total	30 (10.45)	2 (0.70)	22 (7.67)	233 (81.18)	255 (88.85)	287 (100)

$P = 0.0083$

Discussion

There was high percentage of males in our study, 85.02%. High percentage of males in traumatic brain injury had been attributed to their major roles in fending for their families. They are involved in occupations that expose them to danger of getting injured. Commercial vehicle, tricycle and motorcycle driving are populated by males.^[21, 22] Due to high rate of

unemployment, many young men have resorted to vehicle, motorcycle and tricycle commercial driving to make ends meet. Some occupation such as wine tapping is exclusive of men and falls from palm tree is common in our environment. Social life and night clubbing is daily in our city with clubbing 'joints' located in every nooks and crannies of the city. Fights break out from time to time with free use of bottles,

machetes, woods or iron rods. Males are usually involved. Impaired judgment from effects of alcohol on orbitolateral cortex of the frontal lobes plays a great role here.^[23] Andriessen et al^[24] in their study 'Epidemiology, severity classification and outcome of moderate and severe traumatic brain injury' found 70% male involvement. Myburgh et al^[25] in their study of epidemiology and 12-month outcomes from traumatic brain injury in Australia and New Zealand found 74.2% male involvement. These studies showed high male involvement like our study, and so did other series.^[26, 27]

The most common etiology in our study was road traffic accident. In patients 50 years and above, road traffic accident was the most common etiology just like their younger counterparts. All patients 65 years and above were involved in road traffic accident. Andriessen et al^[24] in their study of moderate and severe traumatic brain injuries in patients ≥ 16 found RTA in 50%, followed by falls in 38%. In meta-analysis of mortality among older adults after traumatic brain injury McIntyre et al^[28] found that among patients ≥ 60 years, 51-76% were due to falls and motor vehicle collisions in many studies.^[5, 6, 29-32] Maas et al^[3] noted that fall was increasing, while RTA was decreasing as causes of traumatic brain injuries. Many studies found that patients ≥ 75 years of age are more prone to falls.^[11, 33] Some of the above studies corroborated our study but others had falls as the leading etiology. The difference between some of these studies and our study was the percentage of older adults in our study. In our study the maximum age was 76 years and only 13 patients (4.53%) were 65 years and older. Among them, only two had co-morbidity. In the Western countries and USA the percentage of older patients are higher^[34, 35] with higher co-morbidity.^[36] It also reflected our lower life expectancy and inadequate

provision of health care compared to theirs. The aging process contributes to poor reflexes and poor co-ordination due to the effect of oxidative stress on the presynaptic mitochondria^[32, 37] which make elderly patients prone to falls. The percentage of falls reflects the percentage of elderly patients as seen in Western countries and America where some of the studies were done.^[38] It had also been noted that several aspects of aging might contribute to fall risks such as imbalance, frailty, joint disorders, chronic medical conditions and medical interactions.^[37]

Computerized Tomography scan findings showed that multiple lesions and contusions/ICH were common in younger age groups while SDH was more common in older age group. Cerebral atrophy in old age causes stretching of the bridging veins that pass through the subdural space to empty in the dural sinuses. Trivial force on the head can rupture these vessels and cause bleeding into the subdural space.^[40] Like our findings, other authors also found that the incidence of intracranial hematoma in older adults was higher than in younger adults.^[41]

The favorable outcome (GOS ≥ 4) in our study was 88.85%. Severity of injury affected the outcome in our study. This is in keeping with findings of many authors.^[42-46] Age significantly affected the outcome with transition to worsening outcome seen among the age group 50-59 years. The mortality among them was 27.27%, whereas 40-49 years group had mortality of 3.92%. Also there was significant difference in outcome between patients 20-49 years and those 50 years and above, which was more pronounced when compared to the difference between those 20-64 years and those ≥ 65 years. The transition to worsening outcome occurred in the sixth decade. Many studies reported a change around age 30-40 years, above which outcome became increasingly poorer.^[6, 14, 33, 45, 46] Some

authors used 60 years as the transition mark.^[33, 47-49] Many authors used 65 years as their transition age.^[18, 50-52] Due to many variations in human constitutions and environment, we believe that age range of transition is more realistic than a single age based on our result and others.^[18]

The aging process affects many organs with resultant changes in facial appearance, height and weight loss, lower metabolic rates, reduction in hearing, vision and olfaction, kidney, immune and pulmonary performance.^[53-55] Aging also affect the ability of the cardiovascular system to respond to shock in older adults and this cardiovascular effect affects autoregulation in the brain leading to decreased ability of older brain to maintain cerebrovascular reactivity after traumatic brain injury.^[56] The aging brain undergoes widespread atrophy, neuronal shrinkage, reduced synaptic density and decreased neural plasticity.^[57, 58] Some authors noted that aged brain might be more vulnerable to TBI, with less plasticity and repair after injury.^[59] This view was confirmed by Gilmer et al^[37] in their work 'Age-related mitochondrial changes after traumatic brain injury'. Many authors had documented that age was a strong outcome predictor.^[5, 6, 33, 46] Selassie et al^[60] found that older patients with TBI with three or more pre-existing comorbid diseases had mortality rates that were 4 times higher than patients without any pre-existing disease. All these pointed to the reason why older adults fared worse than the younger adults as we found in our study.

Conclusion

Our study showed that RTA was the most common etiology in all adult groups in our environment. The most intracranial findings in young adults were multiple lesions and contusions/ICH, while subdural hematoma was most common in older patients. We also found that the outcome was significantly related to age but not to etiology. The

transition to worsening outcome was in the sixth decade among our patients.

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