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Original Article

Outcome in Moderate and Severe Traumatic Brain Injury among Elderly Individuals Analytical Study in Government Rajaji Hospital Madurai

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Abstract

Background: Head injuries are among the most common types of trauma encountered in emergency department. Many patients with severe brain injuries die before reaching a hospital; in fact, nearly 90% of pre hospital trauma related deaths involve brain injury.

Objectives: Aim of the study was to study the age as an important prognostic factor in the prediction of outcome in Head Injury patients, to find out the incidence and type of traumatic intracranial lesions in the elderly, to study and analyze the cause of head injury among elderly patients, and to compare conservative, medical, emergency surgical treatment, and outcome.

Results: The poor outcome at all levels in the elderly patients, even with good Glasgow coma scale (GCS) on admission. Although they have lower impact injuries and multiple injuries, their computed tomography scan show more number of intracranial mass lesions, sub arachnoid hemorrhage, and midline shift. Age has been cited as a significant risk factor for the poor outcome in this study.

Conclusion: The mortality rate increases with age mainly from the medical complications to prolonged coma. GCS is a good reliable predictor of prognosis even in the 1st day after admission. If the doctors are persuaded to elicit and record them carefully, predictions can be made reasonably accurate at the bed side.

Key words: Diffuse axonal injury, Extradural hemorrhage, Glasgow coma scale, Intracerebral hemorrhage, Intracranial pressure, Sub arachnoid hemorrhage, Sub dural hemorrhage, Traumatic brain injury

INTRODUCTION

Head injury with brain injury another wise is called traumatic brain injury (TBI). Head injuries are among the most common types of trauma encountered in emergency department (ED). Many patients with severe brain injuries die before reaching a hospital; in fact, nearly 90% of pre hospital trauma related deaths involve brain injury. Approximately 75% of patients with brain injuries who receive medical attention can be categorized as having mild injuries, 15% as moderate, and 10% as severe.

TBI survivors and often left with neuropsychological impairments that result in disabilities affecting work and social activity. Every year, an estimated 80,000–90,000 people in the India experience long-term disability from brain injury.

Given these statistics, it is clear that even a small reduction in the mortality and morbidity resulting from brain injury can have a major impact on public health.

The primary goal of treatment of patients with suspected TBI is to prevent secondary brain injury. The most important ways to limit secondary brain damage and hereby improve a patient's outcome are to ensure adequate oxygenation

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and maintain blood pressure as a level that is sufficient to perfuse the brain. After managing the ABCDEs, patients who are determined by clinical examination to have head trauma and require care at a trauma center should be transferred without delay. If neurosurgical capabilities exist, it is critical to identify any mass lesson that requires surgical evacuation, and this objective is best achieved by rapidly obtaining a computed tomography (CT) scan of the head. CT scanning should not delay patient transfer to a trauma center that is capable of immediate and definitive neurosurgical intervention.

The management of severe and moderate TBI in critical care unit is well advanced and expensive nowadays. Those who survive have protracted hospital stay requiring multi-disciplinary rehabilitative and supportive care, thus resulting in heavy financial and emotional burden to the families. This is especially true for the elderly with TBI.

Many studies have revealed the poor outcome at all levels in the elderly patients, even with good glasgow coma scale (GCS) on admission. Although they have lower impact injuries and multiple injuries, their C.T scan show more number of intracranial mass lesions, sub arachnoid hemorrhage, and midline shift. Age has been cited as a significant risk factor for the poor outcome in several studies.

Aim of the Study

The aim is as follows:

1. To study age as an important prognostic factor in the prediction of outcome in Head Injury patients.
2. To find out the incidence and type of traumatic intracranial lesions in the elderly.
3. To study and analyze the cause of head injury among elderly patients.
4. To compare conservative, medical, emergency surgical treatment, and outcome.
5. Associate with comorbid condition and other injuries.[1-59]

MATERIALS AND METHODS

Elderly patients with their comorbidities and increasing physical frailties are at significant risk from TBI. Aiming at maintaining adequate cerebral perfusion pressure comparable to existing guidelines for the management in moderate and severe TBI a standardized protocol is followed in our neurosurgical intensive care unit.

To evaluate this protocol, we decided to review elderly patients (Group A) with moderate and severe TBI and their outcomes as compared to younger (Group B) patients managed on a standardized protocol.

According to the various standard neurosurgical text books, the severity of head injury is divided, based on GCS at the time of admission of head injury patients.

Head injury severity scale

Severity grade	GCS
Minimal	15 (LOC or amnesia)
Mild	14 (Amnesia/LOC/impaired alertness)
Moderate	9–13 (LOC for 5 min or FND)
Severe	5–8
Critical	3–4

In this study, the moderate and severe grade patients were taken into study based on the above said severity scale. We reviewed the patients with moderate and severe TBI retrospectively over the period of January 2019 to December 2019 admitted in head injury ward, Government Rajaji Hospital (GRH), Madurai Medical College, Madurai, Tamil Nadu. Some of the patients required intubations and artificial ventilation or close neurological monitoring. They were managed in the Neurosurgical Intensive care unit.

In this study, elderly patients are named as Group A and other control group is named as Group B. Of the 250 patients selected based on above mention criteria, 101 (40%) were aged >60 years and 149 (60%) were aged between 20 and 40 years old. They were admitted to Neurosurgical Intensive care unit according to established criteria for moderate and severe head injury and were managed on a standardized protocol with the available facilities in our hospital. Early detection and treatment of secondary insults include hypotension, hypoxia, hyperpyrexia, and hypoglycemia.

For the selection of patients; in this study, a uniform criteria were followed the minimum criteria for that one followed by Becker *et al*.[5]

1. The patients failed to obey commands, failed to speak and have no verbal response to painful stimuli.
2. The motor response ranged from failure to obey commands to no motor response at all.

Data collected, included patients demographics, clinical findings on admission (including admission and post-resuscitative GCS, Pupillary signs, presence of poly trauma and spine injuries, ingestion of alcohol, and mechanism of injury) and C.T scan findings graded using the classification suggested by Marshall *et al*.[42]

A six-category scheme to classify head injury based on Initial CT Scan brain,classified into six categories. Lawrence Marshall and colleagues published it in the Journal of Neurosurgery. It was based on their experience in the pilot phase of the Traumatic Coma Data Bank (TCDB).

An increase in mortality was noted with increasing grade of diffuse injury.

Categories

- Diffuse Injury Type I
  No CT visible intracranial pathology,
- Diffuse Injury Type II
  Cisterns present with middle shift 0–5 mm
  No high – or mixed-density lesion >25 cc.
- Diffuse Injury Type III
  Cisterns compressed or absent with middle shift 0–5 mm
  No high-or mixed-density lesion >25 cc
- Diffuse Injury Type IV
  Midline shift >5 mm
  No high- or mixed-density lesion >25 cc.
- Evacuated Mass Lesion
  Any lesion surgically evacuated.
- Non-Evacuated Mass Lesion
  High- or mixed-density lesion >25 cc.

CT-scans are helpful in assessing the degree of intracranial injury, in predicting outcome, and, if finding is normal, in avoiding unnecessary hospitalization.[43,44]

CT scans are very sensitive to acute hemorrhage or skull fractures. CT scans aid in evaluating.[1] intracranial hemorrhage,[2] skull fractures,[3] mass effect and midline shift,[4] obliteration of the basal cisterns, and[5] evidence of herniation (subfalcine, tonsillar, or uncal) CT scan cannot diagnose a concussion (which is a clinical diagnosis) and is poor for diagnosing diffuse axonal injury (DAI). If DAI has occurred, CT scans may show small hemorrhages in the corpus callosum and cerebral peduncles. In this case, on magnetic resonance imaging (MRI) of the brain should be obtained on an elective basis when the patient is clinically stable because no effective treatment of DAI is currently available. MRI is more sensitive for detecting brainstem injuries, posterior fossa lesions, and brain edema.

As a general rule, a repeat head CT scan is recommended within 4–8 h of the initial scan in patients with intracranial hemorrhages and/or coagulopathies.[33] A repeat brain CT scan is recommended sooner in patients who are deteriorating neurologically or lowering GCS

Treatment Protocol

Few specific treatment options for intracranial hypertension have been subjected to randomized trials, however, and most management recommendations are based on clinical experience.[11,27,32]

In the average adult, the skull encloses a total volume of 1450 Ml: 1300 mL of brain, 65 mL of cerebrospinal fluid (CSF), and 110 mL of blood. The Monroe-Kellie hypothesis states that the sum of the intracranial volumes of blood, brain, CSF, and other components is constant and that an increase in any one of these must be offset by an equal decrease in another.

Medical Management

Hyperventilation became a popular means of reducing intracranial pressure (ICP) in the past. However, recent studies raise concern that aggressive hyperventilation exacerbates cerebral ischemia.[11,27] At present, hyperventilation (PCO_2 of 30–35 mmHg) is recommended to reduce ICP for only a short period, as a temporary measure while other methods of ICP control are initiated. Hyperventilation reduces ICP only temporarily, progressively losing effectiveness after 16 h of continuous use.

Mannitol probably has several mechanisms of action. One obvious mechanism is through osmotic diuresis through drawing edema fluid from the cerebral parenchyma. This usually takes 15–30 min, and the effect usually lasts 1.5–6 h. Serial serum osmolarity levels must be checked to maintain an osmolarity of no >315–320 mOsm/kg H_2O to avoid acute renal failure. For this reason, patients treated with Mannitol must be kept euvolemic with isotonic fluid resuscitation as required.

A barbiturate-induced coma with EEG burst suppression is often a "last ditch effort" to reduce the ICP and should be reserved only for patients with refractory ICP who are unresponsive to other measures. One may even consider decompressive craniotomy before the use of barbiturates.

One must give special attention to preventing hypotension.[30] Data from the TCDB reveal that hypotension in patients with severe TBI increases the mortality rate from 27% to 50%. Traditional management has included fluid restriction to minimize cerebral edema, but this practice may be dangerous in patients who already have intravascular volume depletion. Cerebral edema may occur regardless of the amount of intravenous fluid administered.

The ultimate goal of the management of patients with closed head injuries is to maintain a state of euvolemia. In a euvolemic patient who is hemodynamically stable, two-thirds maintenance of isotonic solution is recommended. Avoid hypotonic fluids because they may decrease serum osmolarity and increase brain swelling. Hypertonic saline may use with caution.

In addition patients with closed head injuries are more prone to acute coagulopathies.[33,40] In patients with acute intracranial hemorrhages, these coagulopathies must be addressed and corrected promptly.

Fresh frozen plasma transfusions until the coagulopathy is corrected are the preferred method. This is especially true for individuals who are taking anticoagulants (e.g., Warfarin) and who are at high risk of continued bleeding.[65]

Pyrexia commonly occurs in patients with head injuries, possibly because of post-traumatic inflammation, direct damage to the hypothalamus, or secondary infection. The most common cause is fever secondary to an underlying infection. Less common is an unexplained fever or "neurogenic" fever estimated to occur to approximately 8% of patients who have head injuries with pyrexia. The source of the fever must be identified and corrected.

Hyperglycemia has also been shown to have a determined effect on induced brain ischemia. Clinical trials support the correlation between hyperglycemia and poor overall outcome in patients with head injuries and recommended that euglycemia be maintained at all times.

Some patients with severe head injuries may develop hypertension, either from an exacerbation of a chronic process or as a result of the head injury. We have to keep systolic blood pressure <180 mm Hg, particularly in patients who have an intracranial hemorrhage if possible, avoid nitroprusside

Because it is a cerebral vasodilator and my actually increase ICP. A Labetolol drip is preferred in patients who blood pressure is difficult to control. Corticosteroids have no proven benefit for patients with severe head injuries.[10,11]

Effective treatment of intracranial hypertension involves the meticulous avoidance of factors that precipitate or aggravate increased ICP. When ICP becomes elevate, ruling out new mass lesions that should be surgically evacuated is important.[15] Medical management of increased ICP should include sedation and paralysis, drainage of CSF, and osmotherapy with either Mannitol or hypertonic saline.[32] For intracranial hypertension refractory to initial medical management, barbiturate coma, hypothermia, or decompressive craniotomy should be considered.

Surgical Management

As a general rule, indications for surgery include any intracranial mass lesion (Extradural hemorrhage [EDH], sub dural hemorrhage [SDH], and intracerebral hemorrhage [ICH]) that causes significant or progressive neurological compromise, particularly a decreased level of consciousness.[41] and death. The overall outcome of individuals with an intracranial lesion that causes significant mass effect is improved with rapid decompression; therefore, operating on these patients as soon as possible is advisable.[63,64]

Before operating, one must always consider the patient's condition and refrain from relying solely on radiographic evidence. For example, elderly patients with severe cerebral atrophy may accommodate a large intracranial hemorrhage,[48] whereas most young individuals may experience neurological deficits with relatively smaller intracranial hemorrhages.

Note that some intracranial hemorrhages may be actively bleeding during the initial head CT scan; what may appear as relatively small on the initial scan may actually become quite significant in a short period of time. In this case, the patient's physical examination finding is more valuable in evaluating his or her intracranial status than the initial head CT scan findings.

Surgical interventions including craniotomy, simple decompression, and evacuation of clot for mass lesions performed according to the existing criteria, were also collected.[57,60] The multi-modality monitoring of physiological monitoring in these patients includes continuous electrocardiogram pulse oximetry, Hourly Urine output, and core temperature. Specific treatment targets for conventional intensive care unit management regarding nutrition, infection surveillance. Deep vein thrombosis- Prophylaxis and ventilator protocols were followed.

The outcome was assessed at post injury using the Glasgow outcome scale (GOS). For early analysis, se divided outcomes into three categories –

1. Death (GOS-1)
2. Unfavorable (GOS 2 and 3)
3. Favorable (GOS 4 and 5).

Clinical findings as well as C.T scans and radiological findings and surgical intervention were correlated to outcome in both age groups. Since the initial seriousness of the injury may be due to other extracranial complications all those patients selected in this study must be in this state for at least 6 h after the injury as recommended by Jennet *et al*.[28] Patients who had spoken after injury before development of coma that issued for 6 h or more are also included.

Exclusion Criteria

Patients in whom the depressed level of consciousness was due to alcohol, drug over dosage and metabolic coma were excluded from this study. Furthermore, all the patients who were apnoeic with dilated and fixed pupils all the time of admission were excluded from this study.

The information collected regarding all the selected cases were recorded in a Master Chart and data analysis was done.

Clinical Assessment

After ABCDE resuscitation, clinical examination and neurological examination were carried out. Of equal importance in the initial examination of patients with impaired consciousness is the vital signs, pupils, eye movements, GCS, and autonomic abnormalities. Of these pulse, respiratory rate, blood pressure, and ICP are monitored at regular interval for the general condition assessment, whereas GCS, oculocephalic response, oculo vestibular response (OVR), etc., are recorded for the prognostic point of view. These are the most powerful predictors of outcome. Apart from GCS, sex and age were taken into account for the analysis. In this study, more emphasis is given for the clinical factors in predicting the prognosis, since they are the factors, which can be assessed easily in any center irrespective of the availability of sophisticated diagnostic, and treatment facilities.

The following factors are assessed

1. Parameter of unconsciousness as in Glasgow Coma Scale. As soon as, the patient was admitted in the ER, patient was examined, and the neurological status was assessed. The eye opening response and verbal response are assessed and entered in the case sheet. It was reassessed every 4 h regularly, When the eyes were closed by edema, the eye opening response was marked as "C," if tracheostomy was done the verbal response was marked as "T."
2. Age and sex
3. Pupillary Reaction: It was tested with a good pinpoint torch light. It was recorded whether the reaction was present or absent. The category that is sluggishly reacting pupils to light was avoided because there might be variability between the observers. Care was taken to avoid errors namely temporary unreacting pupils after epileptic fit or non-reacting dilated pupils due to bilateral local damage to peripheral structures or due to instillation of local eye drops or after an episode of anoxia.

Oculocephalic Response (OCR)

a. OCR is tested by moving the patient's head on either side and observing the ocular mobility. Before eliciting this, cervical spine fractures should be ruled out. It consists of four defined levels.
b. Suppression of eye movements – Normal response in conscious patients.
c. Intact response: Bilateral conjugate righting movements.
d. Impaired response: Dysconjugate movements of the eyes.
e. Absent response.

In this study, the OCR or the doll's eye movement was recorded whether present or absent. Impaired response was taken as brain stem function.

OVR

For eliciting this response, the head should be rotated to 30° to one side and flexed about 30°. The drum must be intact and is not obscured by cerumen. Ice-cold water introduced in 20 ml amounts with the help of syringe.

For declaring this response as "Absent" at least 100 cc should have been used. All those patients with obvious bleeding through the ears were omitted.

The response has four defined levels.

a. Nystagmus to the same side in the normal conscious patients and in lethargic subjects.
b. Tonic conjugate deviation to the irrigated side.
c. Dysconjugate response
d. No response.

OVR is recorded as "Present" or "Absent". Among the oculocephalic and OVR, OVR is more reliable because of its more powerful stimuli. Hence, the eye movement reflexes cannot be declared as absent unless the ocular vestibular response is done.

Management

The protocol used was uniform in all cases. It consisted of preliminary assessment of cases, endotracheal tube intubation whenever necessary, pulse, oxygen monitoring, early diagnosis, and treatment of intracranial complications, routine respiratory and urinary bladder care, and the effective treatment of other associated injuries: Antibiotics and anti-epileptics were given almost to all the patients and Mannitol was used to reduce intracranial tension.

Assessment of Outcome

The physical and mental disabilities combine to produce a social and overall outcome. However, after analysis many of the problems associated with it, Jennett and Bond[26] proposed Glasgow outcome scale. Glasgow outcome scale consists of five categories:

- Gr 1 Death
- Gr 2 Vegetative state
- Gr 3 Severely Disabled (conscious but dependent)
- Gr 4 Moderately Disabled (Independent but disable)
- Gr 5 Good recovery (Minor sequelae).

Within the category death, every effort should be made to distinguish death due to primary (or) secondary brain damage and death due to other systemic complications such as pneumonia, fat embolism, and renal failure. The term persistent vegetative state was coined by Jennett and Plum.[25] This term describes the survivor of acute brain damage who is breathing spontaneously but who remain

unresponsive and speechless with no psychologically meaningful response but the cerebral cortex is functionally inactive.

The moderately disabled patients can be described as independent but disabled. They are able to travel by public transport and can undertake work of a sheltered or reduced kind. However, they are unable to return to their former occupational level. Those who have had good recovery have been able to return to their former occupational level through not necessarily to their former occupation. They may have mild neurological (or) mental deficit.

RESULTS

In Group A, mean age was 67.3 years and standard deviation as 6.7 years. In Group B, Mean age was 29.3 years and SD was 6.7 years ($P = 0.0001$).

The ratio of male and female was 4: 1. Of the 101 patients aged above 60 years old, 73 were male and 28 were female (Age range 60–86 years). In Group A, there were 72.3 male and 27.7% female and in Group B they were 91.9% and 8.1%, respectively ($P = 0.0001$). In the younger cohort, aged 20–49 years, 137 were males and 12 were females.

In Group A, GCS of 5–8 were 74.3% and GCS of 9–13 were 25.7% and in Group A it was 58.4% and 41.6%, respectively ($P = 0.0148$).

In Glasgow outcome scale among Group A patients deaths were 25.7%, poor outcomes in 11.9% and good outcome was 62.4%. In Group B, GOS showed deaths in 58.4% and poor outcome in 10.1% and good outcome in 31.5% ($P = 0.0001$).

Analyzing the mechanisms of injury, in Group a road traffic accident (RTA) cases were 63.4%, followed by 26.7 accidental fall and 9.9% assault cases. In Group B RTA, Fall, and Assault were 77.2%, 17.4%, and 5.4%, respectively.

On observation of eyes in Group A, pupils were normal in 40.6% cases and they were abnormal in 59.4% cases. In Group B, it was normal in 68.5% and abnormal in 31.5% cases ($P = 0.0001$)

In CT findings, Group A showed EDH – 23.8%, SDH – 13.9%, ICH- 11.9%, subarachnoid hemorrhage (SAH) – 24.8%, and DAI – 42.6%. In Group B, CT showed EDH – 31.5%, SDH – 10.1%, ICH – 10.7%, SAH – 3.4%, and DAI-41.6%.

In Group A, OCR was present in 44.6% and absent in 55.4% cases. In Group B, optical character recognition was present in 64.4% and absent in 35.6% cases ($P = 0.0029$).

Regarding OVR in Group A, it was present in 51.5% and absent in 48.9% cases. In Group B, OVR was present 69.8% and absent in 30.2% cases ($P = 0.0051$).

With intracranial traumatic mass lesion 33.3% cases in Group A and 66.7% cases in Group B operated with in 24 h. In Group A and Group B patients, 50% each were operated after 24 h.

Relationship between Outcome and Various Parameters [Tables 2-10].

DISCUSSION

The mechanism of injury between two groups was very different, which was also seen in many studies. The elderly group patients had lower impact injuries. In our study, mainly falls accounted for up to 27% of injuries, and 63 patients involved in RTA injuries, which is contrast to other studies. In RTA majority of injuries were drivers or motorcyclists and others were due to fall from heights and a significant number from Assault (10%).

Multiple injuries were more in younger age group (34%), compared to the elderly where it was only 14%. Co-existing cervical spine injury was also more in younger ones (9%) compared to the older ones where it was only 1% C.T. Scan findings, based on the classification system of Marshall *et al*., were more severe in the elderly group, with 76.4% exhibiting mass lesion compared to 55.6% in the young cohort. The elderly had a 29% incidence of SAH on C.T. Scan, which was comparable to the young, who had 30.2%. The mean GCS on admission in the elderly was (8.3) and it was (8.59) in the young. A GCS of <8 on admission was statistically significant in the elderly group as well as in young age group in predicting outcome and mortality. The distribution of patients, when grouped under GCS 5–8, and GCS 9–12 were also quite comparable in both the groups.

The mortality rate for the Group A was 62.3% compared to 32.2% in the younger age cohort. The outcome rate of the elderly was good about half that of the young (i.e.) 26% compared to 52.5% (Mean 66.8 in Group A and mean 33.2 Group B) [Table 11].

When the GCS on admission was taken into account, there was a trend of poor outcome at all levels in elderly patients compared to the young. The poor outcome was 23.4% in the elderly group compared to the younger age group of 13.5%. The poor outcome rate in those admitted with GCS <8 in the elderly group was lower than the younger group, because most of elderly patients had died due to co morbid conditions and its complications.

Table 1: Age

Age in years	Group A (Study cases)	Group B (Control cases)	Total
Range	60–86 years	20–59 years	20–86 years
Mean	67.3 years	29.3 years	44.3 years
S.D.	6.7 years	6.7 years	19.8 years
"*P*"		0.0001 Significant	

Table 2: Sex

Sex	Group A (Study cases)		Group B (Control cases)		Total	
	No.	%	No.	%	No.	%
Male	73	72.3	137	91.9	21084	
Female	28	27.7	12	8.1	40 16	
"P"			0.0001 significant			

Table 3: Glasgow coma scale

Glasgow coma scale	Group A (Study cases)		Group B (Control cases)		Total	
	No.	%	No.	%	No.	%
5–8	75	74.3	87	58.4	162	64.8
9–13	26	25.7	62	41.6	88	35.2
"*P*"			0.0148 Significant			

Table 4: Glasgow outcome scale

Glasgow Outcome Scale	Group A (Study cases)		Group B (Control cases)		Total	
	No.	%	No.	%	No.	%
Death	26	25.7	87	58.4	113	45.2
Vegetative and S.D.	12	11.9	15	10.1	27	10.8
M.D. and Good	63	62.4	47	31.5	110	44
"P"			0.0001 Significant			

Table 5: Mechanism of injury

Mechanism of injury	Group A (Study cases)		Group B (Control cases)		Total	
	No.	%	No.	%	No.	%
RTA	64	63.4	115	77.2	179	71.6
Accidental fall	27	26.7	26	17.4	53	21.2
Assault	9	9.9	8	5.4	18	7.2
Total	101	100	149	100	110	44

RTA: Road traffic accident

Table 6: Pupils

Pupils	Group A (Study cases)		Group B (Control cases)		Total	
	No.	%	No.	%	No.	%
Normal	41	40.6	102	68.5	143	57.2
Abnormal	60	59.4	47	31.5	107	42.8
"P"			0.0001 Significant			

Table 7: C.T. Findings

C.T. findings	Group A (Study cases)		Group B (Control cases)		Total	
	No.	%	No.	%	No.	%
Epidural hematoma	24	23.8	47	31.5	71	28.4
Subdural hematoma	14	13.9	15	10.1	29	11.6
Intracerebral hematoma	12	11.9	16	10.7	28	11.2
Sub arachnoid hemorrhage	25	24.8	5	3.4	30	12
Diffuse axonal injury	43	42.6	62	41.6	105	42
Total	100	100	149	100	250	100

CT: Computed tomography

Table 8: OCR

OCR	Group A (Study cases)		Group B (Control cases)		Total	
	No.	%	No.	%	No.	%
Present	45	44.6	96	64.4	141	56.4
Absent	56	55.4	53	35.6	109	43.6
"P"			0.0029 Significant			

OCR: Oculocephalic response

Table 9: OVR

OVR	Group A (Study cases)		Group B (Control cases)		Total	
	No.	%	No.	%	No.	%
Yes	52	51.5	104	69.8	156	62.4
No	49	48.9	45	30.2	94	37.6
"P"			0.0051 Significant			

OVR: Oculovestibular response

Table 10: Treatment

Treatment	Group A (Study cases)		Group B (Control cases)		Total	
	No.	%	No.	%	No.	%
Within 24 h	24	33.3	48	66.7	72	100
Surgery (72)	47	39.8	71	60.2	118	100
Conservative (118)	71	37.4	119	62.6	190	100
After 24 h Surgery (60)	30	50	30	50	60	100
Total	101	40.4	149	100	250	100

Table 11: Age and outcome

Outcome	Group A (Study cases)		Group B (Control cases)		Total	
	Mean	S.D.	Mean	S.D.	Mean	S.D.
Good	66.8	7.1	33.2	4.8	52.4	17.8
Bad (Poor and Death)	68.1	6.1	27.5	6.7	38.5	19.2
"P"	0.185 Not significant		0.0001 Significant		0.0001 Significant	

In this study, among Group B, about 78 cases were operated for intracranial mass lesions with a mortality of 25 cases.

At the same time, in the elderly Group A, 54 cases were operated with a mortality of 26 cases. In the younger group, 48 cases were operated <24 h of admission whereas 30 cases were operated after 24 h of admission, with the mortality of 72% and 56%, respectively. In elderly group, 24 cases were operated in 24 h and 30 cases were operated after 24 h with the mortality of 50% and 20%, respectively. The outcomes as well as mortality following surgery for the traumatic intracranial mass lesions are showed in Table 19, in which 65% in the younger age group and 39% in the elderly group show good outcome.

More than 50% patients in the elderly group were not suitable for any neurosurgical intervention after re-assessment by the duty neurosurgeon; moreover, their families did not give consent for the proposed surgery. Nevertheless, they were given supportive treatment under the protocol aimed at minimizing secondary insults (such as preventing hypotension, hypoxia, hyperglycemia, and hyperpyrexia). The majority of 78% cases had a GCS of 5–7 on admission, with CT scan findings of skull fractures, Sub Dural Hematoma, Epidural Hematoma, intracerebral hematoma, and Sub Arachnoid Hemorrhage.

About 40% patients were aged >70 years old and 27.7% were females. Of the 101 patients in the elderly 62.3% died and 11.8% survived in a vegetative state. When these vegetative patients were excluded, the "true" mortality rate in the elderly group was still substantial at 70%, which was still nearly twice that of the younger group. The female mortality in the elderly age group was 42.8% compared to the male mortality rate of 69.8%. This gender difference was not seen in the young age group. Other factors including papillary signs incidence of hypoxia and hypotension did not reveal any significant difference between both age groups, which could account for the difference in outcome in the two age groups.

Injury and Outcome [Table 14]

In Group A following RTA 32.8%, after fall 51.9% and after assault 30% cases were in the poor outcome and deaths. In Group B, the poor outcome and deaths were altogether 67.8% in RTA, 80.8% in accidental fall, and 37.5% in assault cases.

Despite aggressive treatment out of 250 patients, only 38 survived among elderly giving a mortality of 62.3% and only 101 survived in younger cohort group giving mortality of 32.2%. They were called for neurological and functional examination periodically.

Factors Influencing the Outcome

Age and outcome [Table 11]

Most of the studies confirm that the age is the most important significant factor in deciding the outcome from severe head injury.[48] Mortality is very high in the elderly age group in many series.[17,29] In the age group of Group B mortality is low. In general patients of Group B age groups make a relatively better recovery after severe head injury. In this study, Group A shown mean value 68.1 (mean) in bad outcome with SD of 7.1 and 6.1, respectively, so *"P"* value was 0.185 that was not significant. However, in Group B, it was significant and his associates.[12] However, in older age group, the mortality is not directly due to head injury but mainly due to systemic complications such as chest infection, and other co-morbid conditions. About 45% of patients above the age of 60 had high blood urea level at the time of death and 22% had Florid Lung infection at the time of death. The same picture is reflected in the works of Caresson *et al*. and Becker *et al*.[12]

Sex and outcome [Table 12]

In Group A, among male 69.9% good and 30.1% poor and death outcome were seen in this study with significant *"P"* value (0.0227). In Group B in male, 32.1% were good outcome and 67.9% were poor and deaths. In female, 25% were good outcome and 75% were poor outcome with "*P*" value 0.4406 which was not significant.

Outcome related to GCS [Table 13]

Although G.C.S. is not intended to use as a prognostic indicator, the depth and duration coma is related to the outcome. In general, there was a strong correlation between decreasing G.C.S. and increasing mortality, whether the observation was made in the emergency room after resuscitation or after 24 h from the time of admission.

Table 12: Sex and outcome

Sex	Group A (Study cases)				Group B (Control cases)				Total			
	Good		Poor and death		Good		Poor and death		Good		Poor and death	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Male	51	69.9	22	30.1	44	32.1	93	67.9	95	45.2	115	54.8
Female	12	42.9	16	57.1	3	25	9	75	15	37.5	25	62.5
"P"	0.0227 Significant				0.4406 Not Significant				0.4655 Not Significant			

Table 13: GCS and outcome

GCS	Group A (Study cases)				Group B (Control cases)				Total			
	Good		Poor and death		Good		Poor and death		Good		Poor and death	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
5-8	51	69.9	22	30.1	44	32.1	93	67.9	95	45.2	115	54.8
9-13	12	42.9	16	57.1	3	25	9	75	15	37.5	25	62.5
"P"	0.0016 Significant				0.0705 Significant				0.0001 Not Significant			

Table 14: Injury and outcome

Injury	Group A (Study cases)				Group B (Control cases)				Total			
	Good		Poor and death		Good		Poor and death		Good		Poor and death	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
RTA	43	67.2	21	32.8	37	32.1	78	67.8	80	44.7	99	55.3
Accidental Fall	13	48.1	14	51.9	5	19.2	21	80.8	18	34	35	66
Assault	7	70	3	30	5	62.5	3	37.5	12	12	6	33.2

Group-A Patients showed good out come more than 50%. Group-B Patients showed poor of deaths more than 65%

Table 15: Pupillary reaction and outcome

Pupillary Reaction	Group A (Study cases)				Group B (Control cases)				Total			
	Good		Poor and death		Good		Poor and death		Good		Poor and death	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Normal	19	46.3	22	53.7	20	19.6	82	80.4	39	27.3	104	72.7
Abnormal	44	73.3	16	26.7	27	57.4	20	42.6	71	66.4	36	33.6
"P"	0.0111 Significant				0.0001 Significant				0.0001 Significant			

GCS if < 8 is associated with mortality of 28% in elderly (Group A) and 62.1% in younger age group (Group B): Likewise it was 65.4% when GCS is >8 in elderly (Group A). ("*P*" = 0.0016 – significant). In the Miller *et al*.[6] series, the mortality increases as GCS decrease.

Outcome and pupillary reaction [Table 15]

There was a strong correlation between bilateral absence of papillary light response and poor outcome following severe TBI. In Group a cases abnormal papillary reaction in 26.7% cases showed poor outcome. ("*P*" = 0.0111-significant). In Group B, 42.6% cases of abnormal papillary reaction showed poor outcome ("*P*" = 0.0001-significant).

Outcome and oculocephalic, oculovestibular response [Tables 16 and 17]

Both oculocephalic and oculovestibular response were found to be powerful predictors of outcome. In the elderly, Group A 73.3% patients with absent oculocephalic response were either dead or survived with severe disability and 26.7% had good recovery. However, in Group B, 94.8% patients with impair oculocephalic response were either dead or left with severe disabilities, but only 5.2% had good recovery; ("*P*" = 0.0001 – significant).

Likewise, 89.6% patients with impaired OVR had poor prognosis[30] when compared to 11% of patients who had good recovery in the elderly Group A, the outcome was same as in young Group B.

Outcome and mass lesion [Table 18]

Table 18 shows the outcome based on CT findings.

It has been found that intracranial mass lesion has an important significance to outcome. Miller *et al*.[6] had shown that mass lesion intracranial region had a worst outcome.

Table 16: OCR and outcome

OCR	Group A (Study cases)				Group B (Control cases)				Total			
	Good		Poor and death		Good		Poor and death		Good		Poor and death	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Yes	12	26.7	35	73.3	5	5.2	91	94.8	17	12.1	124	87.9
No	51	91.1	5	8.9	42	79.2	11	20.8	93	85.3	16	14.7
"P"	0.0001 Significant				0.0001 Significant				0.0001 Significant			

OCR: Oculocephalic response

Table 17: OVR and Outcome

OVR	Group A (Study cases)				Group B (Control cases)				Total			
	Good		Poor and Death		Good		Poor and Death		Good		Poor and Death	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Yes	21	40.4	31	89.6	17	16.3	87	83.7	38	24.4	118	75.6
No	42	85.7	7	14.3	30	66.7	15	33.3	72	76.6	22	23.4
"P"	0.0001 Significant				0.0001 Significant				0.0001 Significant			

OVR: Oculo vestibular response

In this series among elderly age, Group A 56.5% mortality among operated cases whereas among younger cohort only 31.6% mortality among operated cases irrespective of time of operation. The mortality rate in the elderly those who were operated within 24 h was 39.2% and after 24 h it was 83.3%. In the younger Group B, the mortality of operated cases (<24 h of admission) was 22.7% and after 24 h operated cases the morality was 42.8%.

Outcome and epidural hematoma [Table 18]

Epidural hematoma results from tearing of dural or skull vessels caused by fracture in most of the cases. Epidural hematomas can occur at all ages but are seen primarily

Table 18: CT findings and outcome

CT findings	Group A (Study cases)				Group B (Control cases)				Total			
	Good		Poor and Death		Good		Poor and Death		Good		Poor and Death	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Epidural Hematoma	17	70.8	7	29.2	12	25.5	35	74.5	29	40.8	42	59.2
Subdural Hematoma	10	71.4	4	28.6	7	46.7	8	55.3	17	58.6	12	41.4
Intracerebral Hematoma	7	58.3	5	41.7	7	43.8	9	56.2	14	50	14	50
Sub Arachnoid Hemorrhage	18	72	7	28	2	40	3	60	20	66.7	10	33.3
Diffuse axonal injury	25	58.1	18	41.9	22	35.5	40	64.5	47	44.8	58	55.2

CT: Computed tomography

Table 19: Treatment and outcome

Treatment	Group A (Study cases)				Group B (Control cases)				Total			
	Good		Poor and death		Good		Poor and death		Good		Poor and death	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
1. Within 24 h Surgery (72)	12	50	12	50	13	27.1	35	72.9	25	34.7	47	65.3
Conservative (118)	27	57.4	20	42.6	21	29.6	50	70.4	48	40.7	70	59.3
"P"	0.7505 Not significant				0.9294 Not significant				0.506 Not significant			
2. After 24 h Surgery (60)	24	80	6	20	13	43.3	17	56.7	37	61.7	23	38.3

in young patients (below 40 years).[35,37] Acute epidural hematoma with significant mass effect is not a common complication of head injury, and its incidence is about 2% of patients.

In adults, the incidence of epidural hematoma is lower than that of subdural and intracerebral hematoma. Most epidural hematoma occurs over the convexity of the cerebral hemisphere in the territory of middle meningeal artery. Separation of dura and bone is thought to occur at the time of injury, with subsequent bleeding in the preformed epidural space, rather than stripping of the dura of the inner table of the skull with progressive enlargement of the clot.

In epidural hematoma, there is an initial loss of consciousness after trauma followed by recovery (lucid interval), and then (as the blood clot enlarges) a rapid progression of neurological symptoms, obtundation, contralateral hemi paresis, ipsilateral oculomotor nerve paralysis, decerebrate rigidity, arterial hypertension cardiac arrhythmias, respiratory disturbances, and finally apnea and death. EDH any occur in bilateral location, coup, and contra coup or at vertex.[2,13,56]

Epidural hematoma is associated with a good recovery (70.8%) in the elderly Group A and 22.5% in younger age Group B. Death in this category is the result of other factors such as brain parenchymal damage and prolonged duration of coma. If the patients with extradural hematoma had bilateral dilated unresponsive pupils and decerebrate spasm, the prognosis was very poor.[21] In the elderly Group A with EDH, the mortality rate was 29.2% when compared to 75% among younger age group.

Sub dural hematoma and outcome [Table 18]

Sub dural hematomas may be venous or arterial origin. Most frequently, these hematomas result from the tearing of bridging veins between the cerebral cortex and draining venous sinus. About 50%–60% of acute post-traumatic hematomas are sub dural hematomas. Acute sub dural hematomas are twice as common after injuries that involve a sudden movement of the head, such as fall or assault, than injuries caused by RTA.[16]

Most acute sub dural hematomas result from venous vascular injury that is produced by strain forces at the brain surface. Cerebral ischemia is an important component of brain injury caused by sub dural hematoma. Brain compression by the hematoma, resulting in impairment of the microcirculation, may be an important component.

A hemorrhagic mass should be considered as an intra-cerebral hematoma, when there is a homogenous collection of blood with relatively well-defined margins.[39] Multiple intra-cerebral hematomas are found in approximately 20% cases. Intra-cerebral hematomas are caused primarily by direct rupture of intrinsic cerebral vessels. The hematomas tend to form in the inferior frontal and temporal lobes. Intracerebral hematomas are commonly seen in head injuries in which force is applied to the head over a small area such as missile injuries, perforating wounds, and depressed fractures.[4,5]

Acute sub dural hematoma and intracerebral hematoma had been said to be associated with a very high mortality.[50] Various authors report the mortality ranges from 45% to 90%. In our series, among elderly age Group A, the

mortality for acute sub dural hematoma is 28.6% and with intracerebral hematoma, it is 41.7%: where as in Group B the mortality for acute SDH is 55.3% and with ICH it is 56.2%.

In general, patients with epidural hematoma fare better than patients with acute SDH and ICH. In this study, a slight change with good outcome in SDH and ICH but poor outcome in EDH cases.

Diffuse brain injury

Diffusion brain injury may be concussion or DAI. DAI results from more severe angular or rotational acceleration shear and tensile forces acting on the axons during acceleration and deceleration are thought to cause DAI. DAI is believed to be responsible for the severely impaired neurological function in patients without gross parenchymal contusions, lacerations, or hematomas.

DAI is most often associated with coronal or lateral acceleration injury, which produces the most severe DAI. Histological findings consist of axonal swelling, disruption of the axons, and "retraction balls" (swollen proximal ends of the severed axons). In this study, among elderly Group A the diffuse brain injury showed 41.9% mortality whereas it was 64.5% in the younger group.

Sub arachnoid hemorrhage

Sub arachnoid hemorrhage is common after severe head injury. The centri petal theory of Ommaya and Gennarelli holds that lesion depth is dependent on the force of injury.[45] Traumatic sub arachnoid hemorrhage results from relatively severe injury to the brain.[53] High angular acceleration of long duration is necessary to produce a strain that causes rupture of the superficial vessels in the sub arachnoid space, especially at the base of the skull. In this study, the incidence of the SAH with poor outcome and death was 28% in the elderly Group A and it was 60% in the young cohort (Group B) irrespective of either intracranial hematoma or diffuse brain injury.

Head injury is having significance proportion of neurosurgical condition afflicting the elderly.[4] The complications of a head injury are disproportionately more severe in the elderly. Due to the severity of head injury, patients who are elderly need admissions, prolonged neurosurgical intensive care and longer hospital stay. The epidemiological profile appears to be similar to those in the west.

Men appear to be more commonly affected, especially among younger patients when compared to the elderly individuals [Table 1]. The elderly group usually tends to have injuries of a lower impact, such as those sustained in falls. It may be due to frailties associated with advancing age, poor eyesight, impaired balance, postural hypotension, and cerebrovascular accidents. Volmer *et al*. concluded that multiple injuries do not seem to be a major determinant of death in head injury patients in any age group from the TCDB Study.[40,62]

As such, these elderly patients seem to have less associated multiple injuries or accompanying cervical injuries. This was echoed by Baltar *et al*.[4,6]

At the same time, elderly patients with moderate and severe head injury tend to have a higher incidence of traumatic ICH. In this study, if the patient presented with coma CGS <8 only 22.3% had a favorable outcome compared to 46.5% who had good outcome if the presenting CGS >8.

An increasing incidence of intracerebral hematoma with advancing age is associated with decreasing chances of survival[55] and it has been noted in other studies.[5,52] The clinical course of a patient with an epidural hematoma was first described by Jacobson in 1884.[58] Haselberger *et al*. demonstrated that patients with a pure epidural hematoma in whom the outcome fared significantly better than those with associated intradural lesions. (70% and 40%, respectively). The classic contralateral hemi paresis results from direct pressure on the underlying motor cortex and is seen only with epidural hematomas occurring in the frontotemporal region (70%–80 cases).[39,47]

Hemiparesis, however, may also be ipsilateral and occurs when the opposite cerebral peduncle is pressed against the tentorial edge, classically called Kernohan's notch. Epidural hematomas in the posterior fossa are rare finding. Of all post-traumatic intracranial mass lesions, only 5% are found in the posterior fossa.[21,66,69] Of these, epidural hematomas are most prevalent.[57] Pickard *et al*. showed that the surgical management of the post-traumatic epidural hematoma is one of the most cost effective of all surgical procedures in terms of quality of life and years preserved.[49]

In our study, among the intracranial lesions, epidural hematoma cases (47 cases) are more common in younger group with 74.5% mortality rate. However, in the elderly group, there is slight increase in epidural hematoma (24 cases) when compare to sub dural hematoma (14 cases) with mortality (29.2% and 28.6%, respectively). In a study of pure acute sub dural hematomas, it was found that in 72% of patients, head injury was produced by a fall or assault, in only 24% was the cause a motor vehicle collision.[58] Acute sub dural hematomas may also occur (after trivial Injury) in patients given anti-coagulants and in those with coagulopathies.

Even after operative decompression, the prognosis of subdural hematoma is still poor in many cases. It is though that the co-existing brain damage (DAI) is mainly responsible for the poor outcome. However, compression of the microcirculation and resulting low cerebral blood flow may explain the poor clinical condition and patients who develop acute sub dural hematoma have a worse prognosis than other head injury types with mortality between 50% and 60% in various series.[37]

Intracerebral hematoma account for approximately 20% of all hematomas.[41] Patients on long term anti-coagulant therapy are at increased risk of developing intracerebral hematoma, even after mild head injury.[33] Intracerebral hematoma generally does not require surgical intervention unless there is significant mass effect or increase in intracranial pressure. In this study, both young and elderly group show high mortality in the ICH (intracerebral hematoma) with 40% and 62%, respectively, which was reflected in other series also. In the sub dural hematoma cases, the mortality rate of elderly group after surgery doubles when compare to younger age group (86% and 39%, respectively).

Some studies showed that the presence of traumatic SAH on initial C.T. Scan is also an adverse prognostic factor. It was postulated that the presence of subarachnoid bleed appeared to predict an abnormal ICP. A two-fold increase in mortality was noted in the age group with subarachnoid bleed in the United State trauma Coma Bank Study. In a study from the National institute of health TCDB, sub arachnoid hemorrhage was identified in 39% cases with severe head injury. The presence of subarachnoid bleed may represent major vessel injury with its attendant problems of vasospasm and tissue ischemia. This adverse prognostic effect was also noted in patients with acute subdural hematoma.

Some studies have even suggested that age can be considered an adverse risk factor in head injury. The poor outcome rates quoted in other studies on closed head injury in the elderly ranged from 46% to 98% and age has been identified as a strong prognostic indicator. The threshold has been suggested to be between 55 and 60 years of age. In this review, the poor outcome in the elderly with closed head injury is in line with that found in other studies. Although elderly patients have a much higher incidence of pre-existing systematic disease, age has been discounted as predictor of poor outcome in other studies.

In this study, nearly half of the patients showed diffuse brain injury with or without mass lesion, and therefore they were not subjected to any neurosurgical intervention, but were still managed as per the protocol. The factors which persuaded with not to perform invasive cranial procedures include poor anesthetic risk, from pre-existing co-morbid disease states, coagulopathy, or a very poor neurological condition at presentation (CGS – 3). While it may be argued that this group of patients may theoretically confound out results, we think that it would be highly unlikely as they represent patients with a very poor risk to benefit ratio for surgical intervention and or survival.

Nevertheless, even when this group of patients was dis-regarded, the mortality rate was still 56.5%; this is twice that of the younger group (31.6%). Overall, despite recent advances in the management of moderate and severe TBI, the mortality rate of the elderly remain high.[55] It still unclear that why the elderly have a greater propensity to develop a hematoma after an apparently trivial injury. Certainly, cerebral atrophy with a change in the visco elastic properties of the brain, alterations in the mechanical properties of the bridging veins and stress placed on the venous structures secondary to cerebral atrophy may all contribute.

The other systemic factors, including higher mean blood pressure, increased vascular rigidity and alterations in hemostatic mechanisms may result in the development of larger hematomas, mechanisms may result in the development of larger hematomas, as would the greater potential volume of the sub dural space following brain atrophy.[62] While current results compare favorably with the previous reports, the fact remains that only one in five is expected to have a favorable outcome, with most have a GCS more than 8 on admission. The poor prognosis for elderly patients with severe TBI (GCS <8) with traumatic intracranial hemorrhage on CT scan has important ethical consequences. How far should we pursue intensive and surgical management in this group? Perhaps an individualized approach may be more appropriate.

Obviously, patients who have a poor pre morbid condition, poor GCS, and massive traumatic intracranial hemorrhage may lead the surgeon towards a more conservative approach, while patients who have a good pre morbid state and GCS more than 8 may require a more aggressive approach. Several studies of severely head injured patients have confirmed the influence of clinical factor such as age and intracranial mass lesion, on the final outcome.[41] In our series a study of 160 severely head injured patients have confirmed the influence of age, intracranial mass lesion, papillary reaction, impaired eye movements, and motor response on the final outcome.

Our study shows that Glasgow Coma Scale is of value not only as a methods of quantifying the degree of neurological impairment but also a basis for making early, accurate

predictions of the likely outcome of head injured patients. Initial GCS score alone is enough to make reliable outcome prediction. The other factors such as papillary reaction, brain stem function vital signs, and age can also be used as a basis to predict outcome. Our study suggests that although individually these factors are song predictors of outcome, combination of these factors increased the accuracy.

The predictive power of these simple clinical parameters is undesirable and the amount of data required to make a prediction has to be quite small. Although various advanced investigations (MRI, SSEP, and Bio chemical values) help in prediction of outcome, they are not as useful as clinical parameters. Another aspect of these advanced and sophisticated laboratory data is the cost of their acquisition and their non-availability in the many different kinds of hospitals to which head injured patients are admitted.[60-67]

CONCLUSION

The elderly with head injury needs to be reassessed by neuro surgeons after initial resuscitation. In cases when the presenting GCS is poor and the patient has significant co-morbidities, counseling and discussions of the potential out comes with their families should be done before further therapies are instituted. This will help in the judicious use of limited resources available, as well as to reduce the conditions and financial burdens to the families concerned. The mortality rate increases with age mainly from the medical complications to prolonged coma. GCS is a good reliable predictor of prognosis even in the 1st day after admission. If the doctors are persuaded to elicit and record them carefully, predictions can be made reasonably accurate at the bed side.

Bilateral absence of papillary light reflex and impaired or absent OCR predicted a poor outcome. Abnormal or absent motor responses were also significantly correlated with poor outcome.

Age is probably the most significant factor in the prediction of outcome. If GCS, ocular vestibular response, Glasgow outcome score is included in the study.

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PROFORMA

NAME : I.P. No.:

AGE : D.O.A.:

SEX : D.O.O:

ADDRESS :

GCS ON ADMISSION :

AFTER 24 HOURS :

OCULOCEPHALIC RESPONSE :

OCULO VESTIBULAR RESPONSE :

MECHANISM OF INJURY : RTA/ASSULT/ACCIDENTAL FALL/ OTHERS

PUPILLARY ABNORMALITY :

PUPIL REACTION TO LIGHT:

ADMISSION PULSE :

ADMISSION BP :

RESPIRATORY RATE :

TEMPERATURE :

ANY SYSTEMATIC DISEASE : HYPERTENSION/DIABETES/CAD

BLOOD UREA:

(Serum Creatine and Electolotes)

BLOOD SUGAR :

BLOOD GROUPING :

BLEEDING TIME :

CLOTTING TIME :

CT SCAN FINDINGS :

VENTILATORY CARE:

TYPE OF SURGERY :

DURATION BETWEEN ADMISSION

AND SURGERY :

POST OP COMPLICATION :

GOS : GOOD/POOR/DEATH