

ORIGINAL ARTICLE

Controversies in Traumatic Brain Injury: Neurotrauma Experience in Bandung Indonesia

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ABSTRACT

Objective: Despite progress in the management TBI, several controversies and questions such as the best use of technological advances and the data obtained from multimodality monitoring, the use of mannitol and hypertonic saline, decompressive craniectomy and barbiturate coma, therapeutic hypothermia, anemia and the role of blood transfusion, and venous thromboembolism prophylaxis in TBI. Our Hospital implementing the multimodal treatment for TBI patients.

Materials and Methods: This is a multicenter study, conducted at Spine Research Working Group, Division of Neurospine, Pain and Peripheral Nerve Department of Neurosurgery, Faculty of Medicine Universitas Padjadjaran – Dr. Hasan Sadikin Hospital, Bandung, Division of Neurotrauma, Department of Neurosurgery, Faculty of Medicine Universitas Padjadjaran – Dr. Hasan Sadikin Hospital, Bandung, Indonesia. The duration of study 1 year from Jan. 2011 to Dec. 2012.

Results: In our hospital, within 1 year, the total number of cases of TBI were 2069, or 173 cases / month. The caused of TBI is dominated by traffic accident 1498 (73%) from total cases. Male were more dominant 1537 (74%) compare to female 532 (26%). There were 1488 (72%) cases of mild head injury, 401 (19%) of moderate head injury and 180 (9%) cases of severe head injury. Among the traffic accidents motorbike accidents were predominant, 1132 (55%) cases, followed by pedestrian 322 (16%) cases, falling down 293 (14%) cases, cars 44 (0.2%) cases, crimes 27 (0.1%) cases and others like house family abuse and so on, in 251 (14.7%) cases respectively. Time interval from the occurrence site to the hospital was < 4 hours 1008 cases and the rest are > 4 hours. Clinically, we found 1953 (94%) with pupil equal. The mortality rate caused by Traumatic Brain Injury (TBI) in Indonesia on 2011 is 6.2 – 11.2% which is two times compare to the international literature (3 – 8%). Most of them are young adult. However there are still controversies in term of management of TBI.

Conclusion: There have been many controversies in the management of TBI. It is depend on the resource and experience of the trauma centre itself. Nevertheless it has to be focus on patient priority and outcomes.

Keyword: Controversies TBI, multimodal treatment, prehospital systems, head injury, road traffic accidents.

Abbreviations: TBI: Traumatic Brain Injury, CT: Computed Tomography, ICP: Intracranial Pressure, DC: Decompressive Craniectomy.

INTRODUCTION

Traumatic Brain Injury (TBI) is the leading cause of death in the world, a major killer of young men in the

developed world with enormous emotional, social and financial costs to society. Classification of TBI is based on the GCS (Glasgow coma scale) with severe

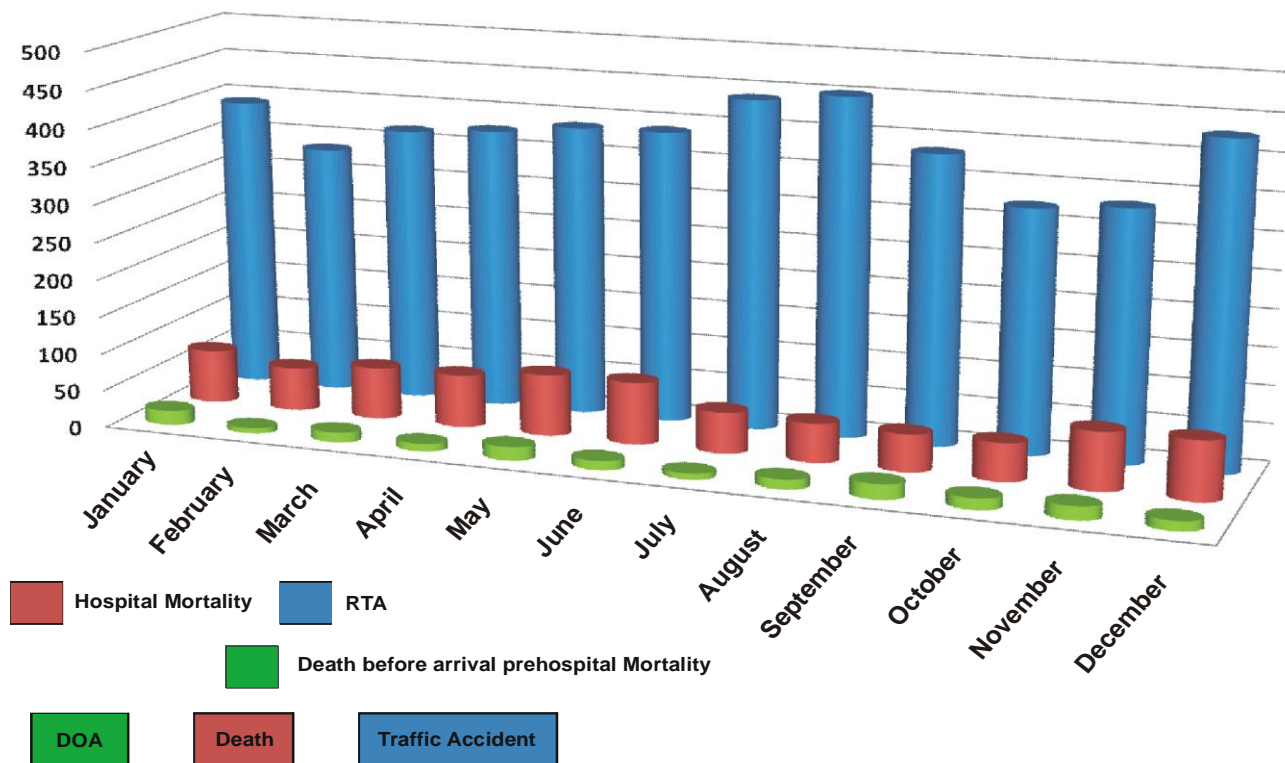


Figure 1: The pattern TBI in 2011 at Hasan Sadikin Hospital Bandung Indonesia.

TBI defined as a GCS < 9 for more than six hours. Mortality is closely related to the initial GCS (plus age and type of lesion). The incidence estimates for TBI range from 100 – 200 / 100.000 population. In USA 30.000 people / week suffer a TBI and 1000 / week will die, with costs of 60 Billion USD / year. So the TBI is now a major focus of US public health and law programmes.¹⁻³ Although computed tomography (CT) scans are the diagnostic test of choice to evaluate head trauma, clinicians vary widely in their use, ranging from less than 5% to upwards of 50% of patients presenting to the Emergency Department.⁴⁻⁸

MATERIALS AND METHODS

This is a multicenter study, conducted at Spine Research Working Group, Division of Neurospine, Pain and Peripheral Nerve Department of Neurosurgery, Faculty of Medicine Universitas Padjadjaran – Dr. Hasan Sadikin Hospital, Bandung, Division of Neurotrauma, Department of Neurosurgery, Faculty of Medicine Universitas Padjadjaran – Dr. Hasan Sadikin Hospital, Bandung, Indonesia. The duration of study 1 year from Jan. 2011 to Dec. 2012.

RESULTS

Epidemiology TBI in Hasan Sadikin Hospital Bandung

In 2011, the pattern of TBI patients in our hospital is shown in figure 1.

The green color represents as death on arrival (DOA), blue is for the traffic accident as the cause of TBI and red is death before arrival in our hospital. The pattern of TBI was high in January and increasingly again from July to August. It might be cause of the weather condition in December – January and also during Ramadhan followed by Idul Mubarak day in around July – August. This pattern is showing us for preparation in these seasons.

Sex Incidence

Based on gender, it is still dominated by male in our hospital, figure 2. The male were 74% and female was 26% from the total patients 2069. It is possible due to the males more having mobilization in their activities.

Severity of Head Injury

Refer to the percentage graph No. 3 in the level of TBI based on GCS classification, we found that mild head

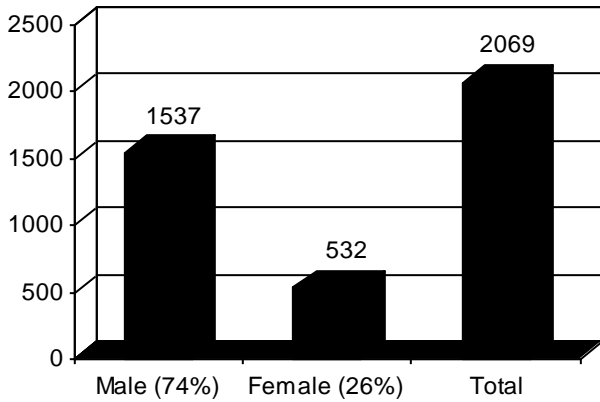


Figure 2: Gender in TBI at Hasan Sadikin Hospital.

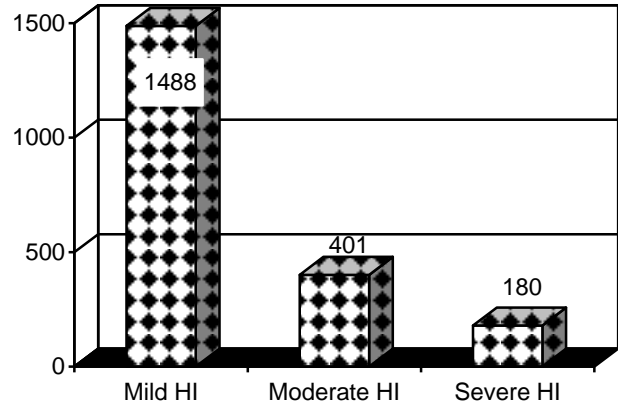


Figure 3: Level of TBI based on GCS.

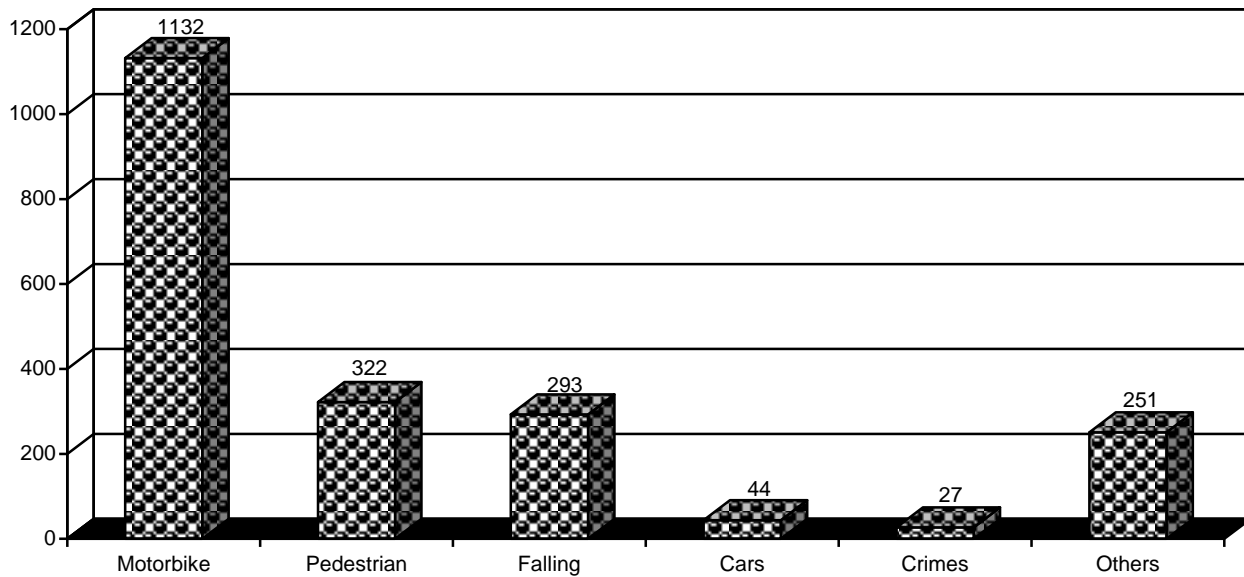


Figure 4: Cause of TBI in 2011.

injury is predominant than moderate and severe head injury (Figure 3.)

Mostly 1488 were having mild head injury and moderate in 401 followed by severe one in 180 cases. This commonly occurred worldwide. The etiology of the TBI is mainly caused by traffic accident as describe in the figure 4.

In Indonesia, most of people are riding motorbike when they go to their office, therefore the incidence of TBI is dominated by motorbikers. It is also being concerned that the police obligate bikers to use helmet, but sometimes they do not obey this regulation. As shown in figure 5.

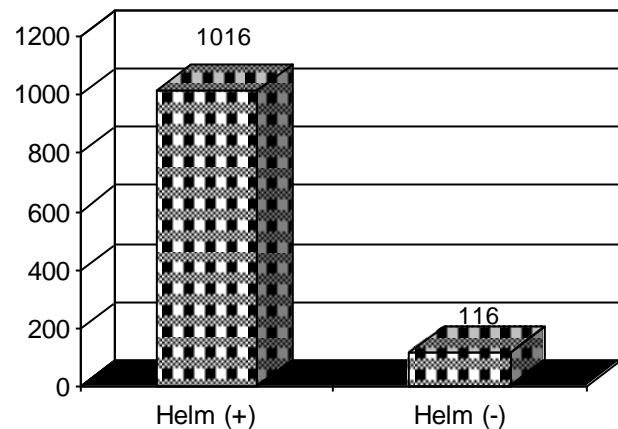


Figure 5: Bikers using helmet.

Interval before surgery was achieved in less than 4 hours mostly followed by more than 4 hours. The rest of them were more than 1 day (Figure 6).

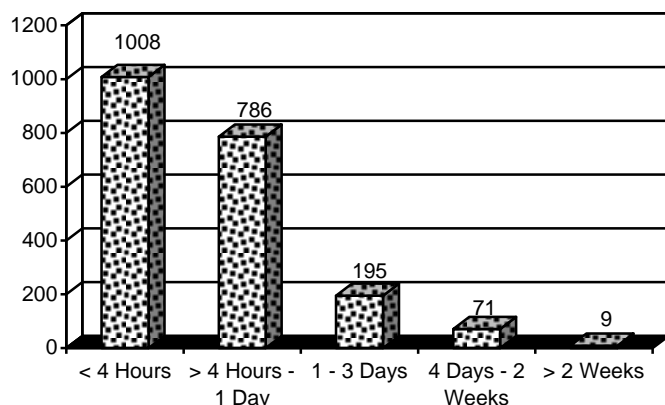


Figure 6: Interval surgery from the TBI occurrence.

DISCUSSION

Cervical Spine Clearance in Severe TBI

Cervical spine injury occurs in 5 – 8% of severe TBI patients. There have been controversies about appropriate clearance of the cervical spine prior to removal of the hard collar. Passive flexion, extension x-ray studies with dynamic fluoroscopy (DF) do not add any information to that obtain by plain radiographs and fine cut 1 mm CT and 3 mm CT plus reformatting.

Saline or Albumin for Fluid Resuscitation

While the SAFE study showed no significant difference in the risk of death in heterogeneous ICU patients resuscitated with either saline or albumin there was concern about a trend towards unfavorable outcome in those patients resuscitated with albumin who had TBI. Therefore a post hoc analysis was done (SAFE TBI Study). Outcome measures were mortality and functional neurological outcome 24 months post randomization. At 24 months 33.2% of the TBI / albumin group had died compared to 20.4% in the TBI / saline group. In those with severe TBI the mortality was 41.8% and 22.2% respectively. Most of the mortality occurred in the first 28 days. For favourable GOS score, the results were 47.3% (albumin) and 60.6% (saline).

Decompressive Craniectomy (DC)

While the need for neurosurgical evacuation of TBI related hematoma is not really controversial, surgical

management of diffuse brain injury with persistent swelling is still controversial. In the last 10 years there had been renewed enthusiasm for DC, with numerous anecdotal reports of favorable outcome. The DECRA study randomized 155 adults in Australia, NZ and Saudi Arabia between 2002 – 2010, with severe diffuse TBI and intracranial hypertension refractory to first tier therapy to either bifrontal DC and dural opening or continued standard care. DC produced dramatic and obvious short term improvement (decreased ICP, medical therapy, ventilation time and ICU stay) but eventual functional outcome measured at six months was the opposite.

There were 19% more patients with poor functional outcomes and 23% more survivors with severe disability in the DC group. The cause for this poorer outcome is unclear but there is speculation that axonal stretch as the brain swelled out of the cranial vault through the DC defect produced an unexpected brain volu trauma. Based on the results, DC can not be recommended for the treatment of severe TBI (without hematoma) with refractory intracranial hypertension. Cost savings in Australia alone from avoiding DC are estimated to be 100M A\$. The RESCUE ICP trial is addressing a similar question in the UK and Europe, at higher threshold ICPs (25 mmHg) for longer periods of time (1 – 12 hours).

Intracranial Pressure (ICP)

Intracranial pressure monitors should be placed for GCS < 9 and abnormal CT (or two of age > 40, SBP < 90, motor posturing). Management of elevated ICP (> 20 mmHg) will generally follow a step wise algorithm and is aimed at reducing brain bulk and supporting arterial pressure. All of the steps can be initiated in the operating theatre, although induction of hypothermia must be done with extreme caution and with an understanding of the potential pitfalls. General CPP (MAP – ICP) should be maintained in the range of 50–70 mmHg with adequate fluid resuscitation and vasopressors. CPP is a major determinant of CBF which under ordinary circumstances with intact cerebral autoregulation, is relatively constant across a wide range of CPP (typically 50 – 150 mmHg). Outside these limits or when cerebral autoregulation is lost, CBF becomes directly dependent on CPP. CPP should not be augmented if ICP < 20 mmHg.

Hyperventilation

The cerebral vasculature is rapidly responsive to

PaCO₂ even after TBI and therefore hyperventilation will cause decreased CBV and ICP. However the concurrent reduced CBF will increase the volume of critically hypoperfused brain tissue and therefore worsen brain ischemia. In addition, the benefit of hypocapnia is short lived as cellular mechanisms compensate for the changes in CSF pH by 24 hours, with associated rebound ICP rises as CO₂ levels return to normal. Acute hyperventilation should only be used as life saving procedure for managing acute neurological deterioration until definitive imaging and neurosurgical intervention is undertaken.

Osmotherapy

Mannitol (0.5 – 1.5 gr/kg) over 15 – 30 minutes reduces ICP through a hemodynamic effect and an osmotic effect. The immediate plasma expanding effect of a bolus of mannitol alters blood rheology with reduced CVR and increased CBF. Autoregulatory vasoconstriction may then decrease CBV and ICP. There is then a delayed osmotic effect (brain shrinkage) that develops over 30 minutes, although this direct removal of water from the brain parenchyma may contribute less to the fall in ICP than the initial hemodynamic effect. Hypertonic saline (HTS) has similar immediate hemodynamic and delayed osmotic effects. The advantage of is that it is less likely to cross the BBB causing delayed rebound cerebral edema. It is also said to improve pulmonary gas exchange, decrease leukocyte adhesion and modulate the inflammatory response. Disadvantages are central pontine myelinolysis, CHF, RF and hyperchloraemic acidosis.

Glucose Control

Hyperglycemia (HG) (glucose > 7.8 mmol/L) is attributed to the stress response following the initial TBI, and is associated with poorer outcomes. The severity of HG probably reflects the severity of TBI and places the brain at risk of secondary injury via glucose driven oxidative stress. The Intraoperative period has been shown to be a time of particular risk and therefore a time for therapeutic intervention. The anesthetic or surgical process can drive up blood glucose level as can steroids. The exact blood glucose at which to intervene is unclear, although if insulin therapy is started, it is critical to avoid hypoglycemia.

Hypothermia

Mild hypothermia (32 – 35°C) induced in the first few hours after an ischemic event can prevent or mitigate

permanent neurological injury. Although the evidence is strongest for post cardiac arrest global ischemic encephalopathy and neonatal asphyxia, the ultimate mechanism of injury is fundamentally the same as that in TBI. There are a multitude of mechanisms by which hypothermia is thought to provide its protective effects of which reduced metabolism is only one. There have been numerous studies of hypothermia in TBI, without clear evidence of beneficial outcome, although the studies have been plagued by methodology and implementation issues, in particular controlling the known and expected side effects of cooling.

Pain Management

Studies have shown that significant numbers of patients have severe post craniotomy pain. A number of pain management strategies have been published recently. Intravenous paracetamol has been shown increase patient satisfaction with pain relief following spinal surgery, without reducing the morphine requirement. Use of a COX₂ inhibitor in conjunction with narcotic analgesia has been shown to reduce pain, length of stay and hospitalization costs. Local anesthesia scalp blocks have also been shown to be an effective means of providing transitional analgesia with a duration of effect that far outlasts the pharmacology of the local anesthetic. The use of paracetamol (PCA) are now well established in neurosurgery with studies of paracetamol (PCA) morphine or fentanyl showing better analgesia, less vomiting and greater satisfaction levels compared to conventional IM / IV bolus regimens, without any differences in sedation or CO₂ levels.

The mortality rate has stuck at 30 – 35% since the 1980 while the mortality attributable to TBI for poly trauma victims who reach hospital late approximately 90%. There is now clear evidence that outcome from TBI is better although still some controversies in our hospital as compared to those with specialist neurosurgical and neuro ICU unit, that cannot simply be explained by good neurotriage. We found that young people in high speed (70 Km/h) using vehicle predominated and that in 25% of cases there was clear evidence of early secondary brain insult (specifically hypotension and hypoxia). Following head injury there is an initial primary insult resulting from the biomechanical effect forces applied to the skull and brain manifested in milliseconds. Although there is enormous heterogeneity in TBI, the common theme is the development of secondary injury occurring over minutes to days, as the result of the common mechanism of cerebral hypoxia

with ischemia setting up the vicious cycle of brain swelling and edema. This is a mixture of cellular energy failure, inflammation activation and blood brain barrier leak, sometimes termed vasogenic and cytotoxic edema. Because the cranial vault is a rigid structure (Monroe Kelly doctrine), as edema develops, ICP increases with limited ability for compensation (CSF or venous shunting and brain displacement / herniation). ICP / edema is aggravated by systemic hypoxia, hypercarbia, hypotension, anemia and hyperglycemia. These contributing factors are generally preventable (or at least manageable) and form some of the basis for guidelines for the management of TBI and represent some of the areas where management is controversial.⁶⁻⁸

The mortality rate caused by Traumatic Brain Injury (TBI) in Indonesia in 2011 was 6.2 – 11.2% which is two times compared to the international literature having 3 – 8% mortality. Most of them are young adults. The high mortality rate not only depends upon level severity, but also the prehospital care. Therefore immediate response should be to decrease morbidity and mortality rate in TBI patients. However there are still controversies in terms of management of TBI.

CONCLUSION

There have been many controversies in the management of TBI. It depends on the resource and experience of the trauma centre itself. Nevertheless it has to focus on patient priority and outcomes.

Consent

Informed consent was obtained from the patient for publication of this case report and any accompanying images. Her family was present at the time.

Competing Interests

The authors declare that they have no competing interests.

Authors' Contributions

RHD, FY, SEO, ABS, AF and MZA had examined, treated, observed, and followed up the subject of this case. FY, SEO, ABS, AF performed the operation on the patient. All authors participated in writing the manuscript. All authors have read and approved of the final manuscript.

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REFERENCES

1. Cooper DJ et al. Decompressive craniectomy in diffuse TBI. *NEJM* 2011; 364: 1493-1502.
2. Servadei F. Clinical value of decompressive craniectomy. *NEJM* 2011; 364: 1558-9.
3. Cooper DJ, Rosenfeld JV. Does decompressive craniectomy improve outcomes in patients with diffuse TBI. *MJA* 2011; 194: 437-8.
4. Langlois JA, Rutland – Brown W, Thomas KE. Traumatic brain injury in the United States. Atlanta, Georgia: CDC, National Center for Injury Prevention and Control; January 2006.
5. Quayle KS, Jaffe DM, Kuppermann N, et al. Diagnostic testing for acute head injury in children: when are head computed tomography and skull radiographs indicated? *Pediatrics* 1997; 99: E11.
6. Palchak MJ, Holmes JF, Vance CW, et al. A decision rule for identifying children at low risk for brain injuries after blunt head trauma. *Ann Emerg Med* 2003; 42: 492–506.
7. Dunning J, Batchelor J, Stratford – Smith P, et al. A meta-analysis of variables that predict significant intracranial injury in minor head trauma. *Arch Dis Child* 2004; 89: 653–659.
8. Dunning J, Daly JP, Lomas JP, et al. Derivation of the children's head injury algorithm for the prediction of important clinical events decision rule for head injury in children. *Arch Dis Child* 2006; 91: 885–891.