TRAUMATIC BRAIN INJURY: A COMMON NEUROSURGICAL ENTITY

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ABSTRACT

Traumatic brain injury (TBI) is leading cause of death and disability worldwide. Every year about 1.5 million affected people die and several millions receive emergency treatment. TBI with current best practice, results in only about one third of patients being able to live independently in the long term. Although people from all age groups may be affected; the TBI is more common in young age because of the exposure of this age to more chances of accidents on roads, work place and during leisure activities. Decompressive craniectomy is a surgical operation to decrease intracranial pressure, has been used with increasing frequency. Neurological surgeons however do not know whether the operation achieves its goal of improving patient outcomes. The question is considered of great importance internationally. In Pakistan and especially in the province of Khyber Pukhtunkhwa, firearm injuries are more common and children are more prone to fall. The incidence of TBI is on the rise in developing countries. Anything that can improve the outcome from TBI has the potential of improving the lives of many head injured patients.

Key Words: *Traumatic Brain Injury (TBI), Disability, Intracranial Pressure, Outcome.*

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INTRODUCTION

In United States about 500000 patients with Traumatic brain injury (TBI) are severe enough to require hospitalization leading to 9 deaths per 100000 populations[1]. In United Kingdom about 1 million people attend hospital each year following head injury [2]. Head injury is recognized as a major public health problem that is frequent cause of death and disability in young people and makes considerable burden on health services[3].

Outcome assessments are usually based on the integrity of neurological function[4]. Since 1970's Glasgow coma scale (GSC) and computed tomography (CT) scanning has been used in evaluating head injury patients[5]. Trauma manifests with variety of injuries and problems that demand rapid evaluation, improvisation and intervention to save life and prevent permanent disability[6,7].

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Level of Evidence:

Human beings and particularly clinicians basically provide services on different types of evidences in medical field. Evidences are of three levels:

Level I: Level I evidence is measurable and visible. It is based on facts, proven in the past and is effective. Effective means that it works. Level I evidence is recommended (recommended evidence), proven evidence and are both ethical and legal.

Level II: This may be measurable but not visible. This is actually based on clinical trials, clinical studies conducted and summaries. It is also justifiable and effective which means that it can/will work. Level II evidence can be considered (considered evidence). It is study based evidence. (Ethical and may or may not be legal).

Level III: This is neither measurable nor visible evidence. It is purely based on consensus made by clinicians. It is not based on clinical trial or studies. Level III evidence is hypothetical and not justifiable. It may work but is not recommended (consensus based evidence). It is not a legal evidence, can be ethical but experimental.

These evidences may be a strong base for making guidelines. Guidelines are based on level I and II evidences and have advantages and dis-

advantages. Guidelines are check lists and can be effective. These are never fixed and can be revised according to its outcome. Clinical guidelines either remain the same for longer duration or can be revised. The revision always depends on patient's specific factors and treatment specific factors for better outcome[8].

Guidelines are made for implementation in the clinical practice, in the society. For implementation a proper system is needed. Any system can be implemented when people related with this system are educated. For education symposiums and literature should be published and publicized which will create awareness. Once awareness is created the magnitude of the problem is calculated and understood. The significance is shown and thus guidelines are implemented.

Guidelines for TBI can be made based on level of evidence (I and II) which should be implemented via proper systematic approach for better out come by educating doctors, paramedical staff in a flourishing system after creating awareness in the field by publications and arranging seminars (Flow chart 1).

Flow Chart 1

Guideline
↓
Implementation
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System
↓
Awareness
↓
Education
↓
Mass mobilization
↓
Work shops
↓
Publications
↓
Clinical Trials

Guidelines are not final words, can be revised because they are applied for prognosis. Prognosis can be better explained on prognostic models but human beings are individual with different specific characteristics which may be different from others due to biological constituents, different sero-typing and different demographic factors like age, sex and other physiologic status of the body[9].

Guidelines are good and necessary in a sense that they have a place to start because it defines the problems. It has certain disadvantages like it stops one from further research and improvement, and one size does not fit all. Its legal implications in clinical practice become difficult as measuring intracranial pressure (ICP) for decompressive craniectomy is Level I evidence and may not be applicable everywhere[10].

Guidelines helps in showing prognosis and proper guidelines generally have acceptable prognosis. Randomly but case to case variation happens. Prognosis is like building blockings and blocks help in building a clinical structure. Likewise, guidelines are mandatory for management of TBI, which helps in treatment of patients with TBI[11].

Traumatic brain injury is a disease from beach to bed, irrespective of age, sex, race and geographical distribution. There are different causes but common one are road traffic accident (RTA), fall from height, sports injuries, violence, assaults and miscellaneous happenings. Which are different in different societies based on culture and civilization.

MANAGEMENT OF TBI

For better management of TBI, there should be pre-hospital care system which needs emergency medical networking means, standard ATLS protocol, which starts with; ABC (airway, breathing, circulation) should be maintained there and then safe transportation system to the hospital for admission and assessment. In the hospital (A / E) patients should be labeled as green way or the one who need monitoring. So inter hospital service with proper liaison with different departments is mandatory. Patients should be examined properly (thorough clinical examination). Airway (A), breathing (B), circulation (C), deformity (D), exposure (E), blood pressure, oxygenation, glucose level, fluids and associated injuries need stabilization[11-18].

Patients who need Neuro-Intensive care unit (NICU) care needs proper monitoring of the following important parameters: intracranial pressure (ICP) monitoring, cerebral perfusion pressure (CPP) monitoring, blood glucose level, blood pressure monitoring and Glasgow Coma Scale (GCS) monitoring[16].

Treatment of TBI includes the following measures:

1. Decrease energy use for neuronal cells by adopting hypothermia
2. Identification of neuropathic agent--- improve pharmocology
3. Neuroimaging CT scan / MRI / contrast imaging studies
4. ICP- CPP monitoring for better treatment option
5. Treatment planning

All these measures will lead to better outcome[11, 19].

No doubt brain use glucose but glucose has neurotoxic effects. Increase level of glucose exacerbates

neuronal ischemia leading to seizures. While hypoglycemia results in brain glycogen uptake leading to decrease synoptic activity and excitability which is responsible for brain dysfunction and necrosis. Strict glucose control is needed for better neuronal function. Sugar level below 80 mg/dl is hypoglycemia, 80-180 mg / dl is normoglycemia and level above 180 mg /dl is hyperglycemia. Level between 80-110 mg / dl is strict and ideal glycemic level. So conventional tight glycemic control is ideal which results in excellent improvement.

Hyperglycemia leads to impaired lactate / pyrolate ratio which disturb brain metabolism. Therefore, for brain euglycemia is fine while hypo and hyper glycemia both are harmful. Severe and prolong duration of hypoglycemia are responsible for killing neurons. Insulin therapy does play important role in head injured patients because under stress, hyperglycemia occurs. The corticosteroids should not be given in patients with traumatic brain injury not only due to its hyperglycemic and other side effects but also because it damages neurons through secondary effects. Therefore do not give steroids to patients with traumatic brain injuries[11,16].

Another important factor is oxygenation by properly maintaining cerebral perfusion and intracranial pressures, hyper ventilation, hypothermia and even barbiturate (sedative) use. Carbon dioxide (CO_2) level is also important[16]. Normocapnia is ideal. Both hypo and hypercapnia leads to PH change.

Intracranial pressure (ICP) above 20 mm Hg needs to be treated vigorously. Intracranial pressure (ICP) is measured by ICP monometer but indirectly by clinical examination, pupillary reaction, Glasgow coma scale (GCS) status, pulse, BP monitoring and observing changes in breathing pattern. ICP can be reduced by supportive measures like head up and oxygenation, symptomatic measures like controlling BP, glucose level, supplemented measures like giving osmotherapy, specific measures like hyperventilation, sedation with barbiturate and surgery if not responded to this 5-S therapy[10,11,16].

Surgery for TBI:

In TBI different type of surgeries can be performed like putting of ICP monometer by twist drill, CSF drainage by burr hole, decompressive craniectomy, decompressive craniotomy for removal of clots (mass lesion) and cranioplasty procedure later on or treatment of its complication[20-23].

In every patient with TBI oxygen, glucose, ICP, CPP and electrolytes and maintaining intake / output record is mandatory. Intracranial pressure above 25 mm Hg almost always need decompressive craciectomy, if remain refractory to medical treatment.

All the national and international data suggest that it is by keeping the intra cranial pressure (ICP) optimal that one could achieve a major improvement in the outcome of severe head injury[16,24]. The goal in this regard is the intra cranial pressure (ICP) of less than 20 mmHg and cerebral perfusion pressure (CPP) of more than 60mm Hg[16,25].

Cerebral perfusion pressure (CPP) should be maintained between 60-70 mm Hg. Below 60 and above 70 is harmful. Both normal ICP and optimum CPP (60-70 mm Hg) normalize the metabolic environment which affects, glucose, lactate, glycerol / glutamatae and pyruvate levels. Their measurement has therapeutic values. Thus inflammatory products which lead to patho-physiological events to the development of secondary brain injury can be stopped[26]. Great concern should be given to benefits and side effects of the metabolic system.

Diffuse Axonal Injury:

The physiological response after diffuse axonal injury (DAI) shows that these neurons are more hyper excitable and axon grows after mild and moderate injury. Neuronal death occurs due to cell membrane damage. If membrane is permeable, cell death ensues[9]. The exact mechanism of cell death is not known but proposed mechanism after mechanical trauma is due to necroptosis, apoptosis, autoptosis or due to pyroptosis. Delayed membrane opening leads to deranged metabolism of cells leading to cell death. Furthermore raised ICP is one of the cause of cell death.

If there is no CSF pressure but increase pressure in the brain exists, ICP should be decreased by opening skull bone[27]. The idea of decompressive craniotomy and expansible duroplasty is helpful in treating the increase ICP. The indication of decompressive craniectomy is limited particularly in DAI. If ICP is refractory and more than 20-25 mm Hg, GCS 3-8, diffuse TBI, no response with ventricular tap, more than 72 hours post injury, decompressive craniectomy can be an option. This is good modality for decreasing ICP, improve survival, decrease ICU and hospital stay on one side. But on the other side it increase dependent out come in the form of increase vegetative state and thus increase the socioeconomic burden on the society and on family due to prolong rehabilitation, and also have its potential complications like infection, sepsis, subdural haematoma, hydrocephalous and re-surgery for cranioplasty[28-31].

Thus decompressive craniectomy is not one stage surgery. It needs second stage after patient rehabilitation and improvement in the form of cranioplasty. Overall it results in generalized decrease in mortality but morbidity is prolonged. Decompressive craniectomy can be unilateral or bilateral. In case bi-frontal, expansile duroplasty and sectioning of falx is need-

ed in patient with TBI for prevention of herniation syndrome[20].

In DECRA trial[31] 60% of severe TBI patients die or remain with severe disability- never independent. Thus decompressive craniectomy should be an option, if first trial fails for decreasing ICP in the form of conservative treatment.

Short term outcome of decompressive craniectomy (within 6 months) is early improved survival but long term results (> 6 months) leads to severe disability. Therefore excluding criteria should be 1: fixed dilated pupils, 2: bleeding diathesis, 3: survival is difficult after 72 hours, 4: extra cranial trauma, 5: brainstem injury and 6: ICP > 40 mm Hg.

Certain authors are of the opinion that decompressive craniectomy is of no help as it increases complications or vegetative state. It is treatment for decreasing refractory ICP and cerebral edema. Hydrocephalous after brain injury is one of the complications and low pressure hydrocephalus does not need VP shunt and little improvement is seen with shunting procedure. Acute subdural haematoma more than 1 cm, midline shift > 5 mm, acute extradural haematoma and large contusion more than 5 cm should be evacuated surgically. These are not indication for decompressive craniectomy[20].

CONCLUSION

In conclusion one can say that do not treat TBI just for neuronal recovery only, but should consider recovery of neurovascular component, neuronal cell and glial cell, not only anatomical aspect but molecular behavior by adopting different approaches for better out come . Hypertonic saline 3% is not bad but in long term the results are same as with mannitol. Brain need glucose in disease state and only N/saline is not justified. Start oral feed on next day of admission by giving oral glucose not injectable and fluid as saline. Thus efficacy and effectiveness of every step should be considered.

REFERENCES

1. Lu J., Marmarou A., Choi S., et al: Mortality from traumatic brain injury. Acta Neurochir Suppl 2005;95:281-5.
2. Hermstad E, Adams B. Traumatic brain injury complicated by environmental hyperthermia. J Emerg Trauma Shock 2010;3:66-9.
3. Tabish SA, Shah S, Bhat AS, Bhat FA, Shoukat H, Mir MY. Clinical profile and mortality pattern in patients of ballistic trauma. J Int Med Sci Acad 2004;13:247-50.
4. MacLeod J, Lynn M, McKenney M, Jeroukhimov I, Cohn S. Predictors of mortality in trauma patients. Am Surg 2004;70:805-10.
5. Patel H, Bouamra O, Woodford M, King A, Yates D, et al. Trends in head injury outcome from 1989 to 2003 and the effect of neurosurgical care: an observational study. Lancet 2005;366:1538-44.
6. Lee A, Garner A, Fearnside M, Harrison K. Level of prehospital care and risk of mortality inpatients with and without severe blunt head injury. Injury 2003;34:815-9.
7. Jacobs G, Aaron-Thomas A, Astrop A. Estimating global road fatalities. London: Transport Research Laboratory; 2000.
8. Oxford Centre for Evidence-Based Medicine. Levels of evidence and grades of recommendation [Online]. 2009 [cited on 2012 Sep 10th]. Available from URL: http://www.cebm.net/levels_of_evidence.asp
9. Straus SE, Richardson WS, Glasziou P, Haynes RB. Evidence-based medicine: how to practice and teach EBM. 3rd ed. Philadelphia: Elsevier Churchill Livingstone; 2005.
10. Narayan RK, Kishore PRS, Becker DP, Ward JD, Enas GG, Greenberg RP, et al. Intracranial pressure: to monitor or not to monitor? A review of our experience with severe head injury. J Neurosurg 1982;56:650-9.
11. Bullock MR, Povlishock JT. Guidelines for the management of severe traumatic brain injury. J Neurotrauma 2007;24:1-106.
12. Bullock R, Chestnut RM, Clifton G, Ghajar J, Marion DW, Narayan RK, et al. Guidelines for the management of severe head injury. J Neurotrauma 2000;17:449-627.
13. Alderson P, Roberts I. Corticosteroids for acute traumatic brain injury. Cochrane Database Syst Rev 2000;(2):CD000196.
14. Schierhout G, Roberts I. Mannitol for acute traumatic brain injury. Cochrane Database Syst Rev 2000;(2):CD001049.
15. Guidelines for the management of penetrating brain injury. J Trauma 2001;51:3-15.
16. Bullock MR, Povlishock JT. Guidelines for the management of severe traumatic brain injury. J Neurotrauma 2007;24:1-106.
17. Bullock MR, Chestnut R, Ghajar J. Surgical management of TBI author group. Neurosurgery 2006;58:1-62.
18. Chestnut RM, Ghajar J, Maas A, Marion DW, Servadei F, Teasdale GM, et al. Early indicators of prognosis in severe traumatic brain injury. New York: Brain Trauma Foundation; 2000. p. 1-103.
19. Levine JE, Becker DP. Reversal of incipient brain death from head injury apnea at the scene

of accidents. N Engl J Med 1979;301:109.

20. Becker DP, Miller JD, Ward JD, Greenberg RP, Young HF, Sakalas R. The outcome from severe head injury with early diagnosis and intensive management. J Neurosurg 1977;47:491-502.
21. Galbraith S, Teasdale G. Predicting the need for operation in the patient with an occult traumatic intracranial hematoma. J Neurosurg 1981; 5:75-81.
22. Sheinberg MA, Kanter MJ, Robertson CS, Contant CF, Narayan RK, Grossman RG. Continuous monitoring of jugular venous oxygen saturation in head-injured patients. J Neurosurg 1992;76:212-7.
23. Andrews BT, Chiles BW, Olsen WL, Pitts LH. The effect of intracerebral hematoma location on the risk of brain-stem compression and on clinical outcome. J Neurosurg 1988;69:518-22.
24. Berger E, Leven F, Pirente N, Bouillon B, Neugebauer E. Quality of life after traumatic brain injury: a systemic review of the literature. Restor Neurol Neurosci 1999;14:93-102.
25. Diringer MN, Aiyagari V, Zazulia AR, Videen TO, Powers WJ. Effect of hyperoxia on cerebral metabolic rate for oxygen measured using positron emission tomography in patients with acute severe head injury. J Neurosurg 2007;106:526-9.
26. Narayan RK, Kishore PR, Becker DP, Ward JD, Enas GG, Greenberg RP, et al. Intracranial pressure: to monitor or not to monitor? A review of our experience with severe head injury. J Neurosurg 1982;56:650-9.
27. Gautschi OP, Hildebrandt G. Emil Theodor Kocher (25/8/1841-27/7/1917)--a Swiss neurosurgeon and nobel prize winner. Br J Neurosurg 2009;23:234-6.
28. Carter BS, Ogilvy CS, Candia GJ, Rosas HD, Buonanno F. One year outcome after decompressive surgery for massive nondominant hemisphere infarction. Neurosurgery 1997;40:1168-75.
29. Polin RS, Shaffey ME, Bugaev CA, isdale N, Germanson T, Bocchicchio B, et al. Decompressive bifrontal craniectomy in the treatment of severe refractory posttraumatic cerebral edema. Neurosurgery 1997;41:84-100.
30. Munch E, Horn P, Schurer L, Piepgras A, Paul T, Schmiedek P. Management of severe traumatic brain injury by decompressive craniectomy. Neurosurgery 2000;47:315-26.
31. Cooper JD, Rosenfeld JV, Murray L, Arabi YM, Davies AR, D'Urso P, et al. Decompressive craniectomy in diffuse traumatic brain injury. N Engl J Med 2011;364:1493-502.

CONTRIBUTORS

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