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Diffuse Axonal Injury: An institute Experience

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Abstract

Introduction: Traumatic Brain Injuries are among the leading cause of mortality and morbidity world over. They are the leading cause of death among the younger age groups accounting for several indirect social and economic problems. This becomes more important in developing nations like India.

Methodology: 30 pediatric and 30 adult patients that were admitted and diagnosed to have Diffuse axonal Injury were included in our study. The duration of the study was 6 months between June and November 2018 in the Institute of Neurosurgery, Madras Medical College.

Discussion: Diffuse axonal injury is caused from widespread tearing of axons and small vessels by shearing forces and is defined as prolonged post-traumatic coma over 6 hours following injury without demonstrable mass lesion.

Results: Of the 21 patients with Grade 1 DAI, all 21 had favorable outcome as defined by GOS 4 or 5. 13 of the 20 in Grade 2 DAI had favourable outcomes while only 5 of the 19 with Grade 3 DAI had favourable outcomes.

Conclusion: In our study we were able to find out that while there was a statisticaly significant correlation between grade of DAI and the outcome (p=0.002), there was no statistically significant correlation between gender and the outcomes (p=0.3).

Keywords: Diffuse Axonal Injury; Traumatic Brain Injury; Mortality; Pediatrics.

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Introduction

Traumatic Brain Injuries are among the leading cause of mortality and morbidity world over. They are the leading cause of death among the younger age groups accounting for several indirect social and economic problems. This becomes more important in developing nations like India. As per report by the ministry of road transport, Government of India (2007) 1.4 lakhs road accident happened in 2007 with 40,612 people killed and 1.5 lakhs people injured [1,2]. Hence, India is leading the world in fatalities due to road accidents. With the increasing use of MRI, Diffuse Axonal Injury is reported far more frequently. While recovery from DAI is dependent on the grade of the Injury, in our study we attempt to establish a correlation between age and outcomes.

TBI in children often occur in the same manner as in adults but differ in both pathophysiology and management. The differences are attributable to age-related structural change, mechanism of injuries based on physical ability of the child, and

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the difficulty in neurological evaluation of pediatric populations [3]. Several small case series of DAI in children, using differing imaging methods, found varying outcomes and were unable to correlate injury with prognosis [4,5,6,7]. Advances in diagnostic imaging have improved the quality of care by assisting healthcare providers to evaluate and diagnose children with TBI.

Materials and Methods

Thirty (30) pediatric and 30 adult patients that were admitted and diagnosed to have Diffuse axonal Injury were included in our study. The duration of the study was 6 months between June and November 2018 in the Institute of Neurosurgery, Madras Medical College. Patients were assessed based on their admitting GCS, associated injuries, duration of coma and these were correlated with outcome at the time of discharge as defined by Glasgow Outcome Score as favourable (GOS-4 and 5) or unfavourable (GOS 1,2 and 3). Patients with hypotension and significant injury to other systems which required intervention were excluded from the study. DAI was defined based on the description by Gennarelli et al. An MRI finding of scattered small hemorrhagic lesions on hemispheric white matter was classified as grade I, a finding of additional focal lesions on the corpus callosum was classified as grade II, and a finding of additional focal lesions on the brain stem was classified as grade III. The correlation between the MRI grade and the mean time interval to recovery of consciousness was evaluated by one-way analysis of variance (ANOVA). Statistical significance was defined as $p < 0.05$.

Results

Of the 60 patients in the study, 43 were men and 17 were women.

Among the pediatric patients, 12 had Grade 1 DAI, 11 had Grade 2 DAI and the remaining 7 had Grade 3 DAI. Among the adults, 9 patients had Grade 1 DAI, 9 more had Grade 2 DAI and 12 had Grade 3 DAI.

The gender distribution among the pediatric patients was 8 boys and 4 girls had Grade 1 DAI, 7 boys and 4 girls had Grade 2 DAI awhile 4 boys and 3 girls had DAI of Grade 3. Among the adults, 8 men and a woman had Grade 1 DAI. Grade 2 DAI was noted in 7 men and 2 women while 10 men and 2 women had grade 3 DAI.

Of the 21 patients with Grade 1 DAI, all 21 had favorable outcome as defined by GOS 4 or 5. 13 of the 20 in Grade 2 DAI had favourable outcomes while only 5 of the 19 with Grade 3 DAI had favourable outcomes. There was a statistically significant correlation with p value of 0.002.

23 of the 30 pediatric patients had a favourable outcome. It included all 12 who had Grade 1 DAI, 8 of the 11 with Grade 2 DAI and 3 of the 7 with Grade 3 DAI.

Among the adults, 16 of the 30 had favorable outcomes. 9 belonged to Grade 1 DAI, 5 had Geade 2 DAI while only 2 of the 12 with Grade 3 DAI had favourable outcomes.

There was a statistically significant correlation between age and outcome with a p value of 0.03.

Discussion

Diffuse axonal injury is caused from widespread tearing of axons and small vessels by shearing forces and is defined as prolonged post-traumatic coma over 6 hours following injury without demonstrable mass lesion [8,9,10]. “Diffuse degeneration of the cerebral white matter” was first defined by Strich [11]. The time course of the pathological changes was established by Adams et al. [12]. Studies have suggested that children with deeper lesions are more likely to have poor outcomes, the so-called Ommaya-Gennarelli hypothesis [13,14,15].

The Ommaya-Gennarelli depth of lesion model was used to create the Adams classification for animal studies. Mild (grade 1) DAI includes microscopic changes in subcortical white matter, corpus callosum, brainstem, and cerebellum. Moderate (grade 2) DAI is defined by grossly evident focal lesions isolated to the corpus callosum. Severe DAI (grade 3) includes additional focal lesions in the dorsolateral rostral brainstem [16]. Although useful for animal studies, it is uncertain how well this classification translates to the clinical setting. In one study, patients with DAI lesions limited to subcortical white matter had better outcomes than patients with injury to the corpus callosum or brainstem [17]. Other investigators found a high incidence of brainstem hemorrhage in patients with more severe TBI and DAI.

There are unique biomechanical properties for pediatric brain injury due to a combination of higher plasticity and deformity, whereby external forces are absorbed in a different way compared to adults. The infant skull is less rigid, and open

sutures function as joints, allowing for a small degree of movement in response to a mechanical stress. The cerebral white matter contains little myelin, and its distribution is very different in newborns compared with that in adults. The neonatal brain is watery, while the myelinated brain has a much higher density due to the progressing myelination and progressively lowering of the water content. Temporal differences between myelination of various brain areas are pronounced during progressing development. Myelination follows programmed patterns with a caudo-cranial and posterior-anterior predominance. The degree of myelination results in different absorption of traumatic forces, with increased susceptibility to TBI in the unmyelinated regions [19].

Diffuse axonal injury can be diagnosed using clinical signs (level of consciousness and neurological deficits) and radiological findings. Zimmerman reported the first study of radiological diagnosis of diffuse axonal injury that includes small hemorrhagic lesions on the corpus callosum, upper brain stem, corticomedullary junction, parasagittal area, and basal ganglia. Brain computed tomographic (CT) findings lack accuracy in the prediction of a patient's outcome and do not correspond well to the patient's GCS score or neurological state [20].

Conclusion

In our study we were able to find out that while there was a statistically significant correlation between grade of DAI and the outcome (p=0.002), there was no statistically significant correlation between gender and the outcomes (p=0.3). However, we were able to establish a statistically significant correlation between age of the patient and the outcome (p=0.03). A greater percentage of pediatric patients had a better outcome compared to their adult counterparts. These findings of our study are in line with several studies that have concluded that pediatric populations tend to show comparatively better recovery than the adult populace.

Given the increasing incidence of DAI and the increasing number of pediatric patients being affected, further studies are needed to better prognosticate this condition which may have long lasting impact on the growing child and the plastic brain and ipso facto, be a significant factor in the development of the child and by extension, the community.

References

1. Samabasivan M. Epidemiology of Neurotrauma. Neurology and Prevention. Neurol India (Supl) 1991;43:915.
2. Ramamurthi B. Road accidents, Epidemiology and Prevention. Neurol India (Supl). 1991;43:915.
3. Ghajar J, Hariri RJ: Management of pediatric head injury. PediatrClin North Am. 1992;39:1093–1125.
4. Chiaretti A, Visocchi M, Viola L, et al. Diffuse axonal lesions in childhood. Pediatr Med Chir. 1998;20:393–397.
5. Vowles GH, Scholtz CL, Cameron JM. Diffuse axonal injury in early infancy. J Clin Pathol. 1987;40:185–89.
6. Kurihara M, Kumagai K, Nakae Y. Functional outcome after diffuse axonal injury in childhood traumatic brain injury. No To Hattatsu. 1999;31:408–14.
7. Gleckman AM, Bell MD, Evans RJ, Smith TW. Diffuse axonal injury in infants with nonaccidental craniocerebral trauma: enhanced detection by beta-amyloid precursor protein immunohistochemical staining. Arch Pathol Lab Med. 1999;123:146–51.
8. Alberico AM, Ward JD, Choi SC, Marmarou A, Young HF. Outcome after severe head injury : relationship to mass lesions, diffuse injury, and ICP course in pediatric and adult patients. J Neurosurg. 1987;67:648–56.
9. Eisenberg HM, Gary HE, Jr, Aldrich EF, Saydjari C, Turner B, Foulkes MA, et al. Initial CT findings in 753 patients with severe head injury. A report from the NIH Traumatic Coma Data Bank. J Neurosurg. 1990;73:688–98.
10. Eum SW, Lim DJ, Kim BR, Cho TH, Park JY, Suh JK, et al. Prognostic factors in patients with diffuse axonal injury. J Korean Neurosurg Soc. 1998;27:1668–74.
11. Strich SJ. Diffuse degeneration of the cerebral white matter in severe dementia following head injury. J NeurolNeurosurg Psychiatry. 1956;19:163–85.
12. Adams H, Mitchell DE, Graham DI, Doyle D. Diffuse brain damage of immediate impact type. Its relationship to "primary brain stem damage" in head injury. Brain. 1977;100:489–502.
13. Cordobes F, Lobato RD, Rivas JJ, et al. Post-traumatic diffuse axonal brain injury. Analysis of 78 patients studied with computed tomography. ActaNeurochir (Wien). 1986;81:27–35.
14. Ommaya AK, Gennarelli TA. Cerebral concussion and traumatic unconsciousness. Correlation of experimental and clinical observations of blunt head injuries. Brain 1974;97:633–54.
15. Grados MA, Slomine BS, Gerring JP, et al. Depth of lesion model in children and adolescents with moderate to severe traumatic brain injury: use

of SPGR MRI to predict severity and outcome. J NeurolNeurosurg Psychiatry. 2001;70:350–58.

16. Gentry LR. Imaging of closed head injury. Radiology 1994;191:1–17.
17. Kampfl A, Schmutzhard E, Franz G, et al. Prediction of recovery from post-traumatic vegetative state with cerebral magneticresonance imaging. Lancet. 1998;351:1763–67.
18. Cordobes F, Lobato RD, Rivas JJ, et al. Post-traumatic diffuse axonal brain injury. Analysis of 78 patients studied with computed tomography. ActaNeurochir (Wien). 1986;81:27–35.
19. Stark MJ, Hodyl NA, Belegar V KK, Andersen CC. Intrauterine inflammation, cerebral oxygen consumption and susceptibility to early brain injury in very preterm newborns. Arch Dis Child Fetal Neonatal Ed. 2016;101:F137– F142.
20. Duhaime AC, Christian CW, Rorke LB, Zimmerman RA. Nonaccidental head injury in infants—the "shaken-baby syndrome." N Engl J Med. 1998;338: 1822–29.