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Post traumatic epilepsy. An analysis of 12 cases

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Abstract: An analysis of the mechanism of epilepsy and epileptogenesis after traumatic brain injury will give us an insight into neural circuitry. In a retrospective analysis of 48 cases of moderate and severe traumatic brain injury, who reported for follow up to our centre over a period of two years. Of these, 12 patients with post traumatic epilepsy were identified. The risk factors, EEG patterns and the quality of control were analyzed. The pathophysiology and paradigms of management have been discussed.

Keywords: head injury, post traumatic epilepsy

INTRODUCTION

There is much ongoing controversy surrounding post traumatic epilepsy (PTE). What is the mechanism of seizure genesis? Will post traumatic seizures lead on to epilepsy? Is there a role for prophylactic antiepileptic drugs and if so for how long should we administer them? We attempted to address these issues in a retrospective study and a review of contemporary literature.

MATERIAL AND METHODS

We retrospectively evaluated 48 consecutive cases of severe traumatic brain injury (TBI) who reported to our institution for follow up between June 2006 and May 2008. We studied the mode of injury, the management protocol, the type and frequency of seizures, the EEG pattern, the antiepileptic drugs used and the degree of seizure control.

RESULTS

Age and Sex:

The ages varied between 28 years and 54 years. The median age was 44. All 48 patients studied were males.

Mode of Injury

Of the 48 cases studied, 32 patients had suffered TBI as a result of Road Traffic Accidents (RTA), 12 were battle casualties and 6had fallen from heights.

Severity of the Injury

18 patients had severe head injury (GCS<8). 30 patients had moderately severe injuries. 14 patients had undergone surgical procedures for evacuation of hematomas or contusions.

Post traumatic Seizures

Twelve patients had suffered post traumatic seizures. Six of these patients were from the battle casualty group and 6 from the road traffic accident group. All the war injury patients had undergone surgery. Three of the road traffic accident patients had undergone surgical procedures for hematoma evacuation.

EEG patterns

EEG showed focal onset epilepsy, with one or two zones showing spike patterns, in 6 patients while 2 patients showed a generalized seizure pattern. 4 patients had normal interictal EEG patterns. The two patients who showed a generalized seizure pattern had both suffered predominantly inertial injuries (high speed RTA-Non surgical lesions). Of the 6 patients with focal epileptic foci, 3 were from the RTA group and 3 from the war injury group. Clinically all the patients had generalized seizures.

Seizure control

8 of the 12 patients had satisfactory seizure control (< 2 overt seizures a year). 4 patients suffered more than 2 seizures a month. One patient suffered more than 4 seizures every month despite being on 4 drugs.

DISCUSSION

There is a surge of excitatory amino acids, mainly glutamate and aspartate at the moment of injury[1]. The NMDA receptor modulated channels are affected, resulting in an inflow of calcium[2]. This Calcium acts as

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a mitochondrial toxin. The levels of ATP are decreased. The sodium pump fails. This results in an influx of sodium into the cell. The levels of extracellular magnesium are also depleted, resulting in neuronal hyper excitability[3].

After a TBI, there is a field of excitable neurons around the injury site. The incidence of electrographic seizures after TBI is about 30% [4]. As the electrical activity spreads, clinically overt manifestations appear. Partial motor seizures can occur, with or without Jacksonian progression. Complex partial seizures occur when the limbic circuits get activated. The incidence of clinically overt seizures has been reported to be in the range of 12-20% . There is a higher incidence of seizures in patients with compound head injuries and in the pediatric age group.

Seizures after TBI are detrimental. They increase the cerebral metabolic requirement and increase the intracranial pressure, both phenomena resulting in neuronal hypoxic injury. Prophylactic phenytoin is therefore considered the standard treatment of care in moderate and severe TBI[5]. However, recent studies have refuted this assumption. A study by Young et al in the pediatric age group on the efficacy of phenytoin in the prevention of post traumatic seizures showed an incidence of 7% and 3%, in the test and control groups after TBI (no statistical significance)[5]. However the low incidence of seizures in both the control and test groups was probably due to the tight ICP control and the liberal use of diazepam for sedation in these patients.

Even more contentious is the issue of epileptogenesis. The phenomenon of epileptogenesis after injury is poorly understood. It is also probable that the mechanisms of epileptogenesis are different in impact injuries and inertial injuries. Impact injuries are predominantly cortical injuries, while inertial injuries involve dominantly axonal ruptures.

In cortical injuries, there is neosynaptogenesis probably mediated by the trophic influence of astrocytes .This neosynaptogenesis may result in the establishment of an epileptic focus. In inertial injuries, the mechanism of epileptogenesis is different. After axonal loss, there is an attempt at regeneration which involves the proliferation of neural stem cells in the CA3 region of the dentate gyrus of the hippocampus. Mossy fiber proliferation in this area, has been correlated with epileptogenesis . In the present study, there is a tendency towards generalized epilepsy patterns after inertial injury, although the numbers are too small for statistical significance.

CONCLUSION

The incidence of post traumatic epilepsy in this cohort is 25%. Being a retrospective analysis, there is possibly a bias, with more epilepsy patients reporting for follow up. The incidence of post traumatic epilepsy in our battle casualty group was 50%, again reflecting the same bias. The patients with EEG features of generalized seizures were from the inertial injury group, suggesting a different mechanism for epileptogenesis..

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