Letter to the Editor

Early post-traumatic epilepsy in a patient with initial normal CT scan

Madam, Despite several studies, no drug strategy has been identified to date, to curb the biochemical events leading to epileptogenesis in patients with post-traumatic epilepsy.[1] Current evidence is that routine preventive anticonvulsants are not indicated for patients with head injuries and the treatment of early post-traumatic seizures does not influence the incidence of post-traumatic epilepsy.[1-3] A 45 year gentleman presented with history of assault by an iron object on his head. He had history of loss of consciousness for about 30 minutes. There was no history of vomiting, ear or nasal bleed or seizures. At the time of admission his Glasgow coma scale was E4V4M6 and pupils were bilateral equal and reacting to light. There were no focal neurological deficits. Initial CT scan done about after 6 hours of injury was apparently normal. He had associated fracture neck of right humerus. He was managed conservatively and his GCS improved to E4V5M6 by next day morning. On third day of admission he developed three successive attacks of generalized tonic-clonic seizures that could be controlled with

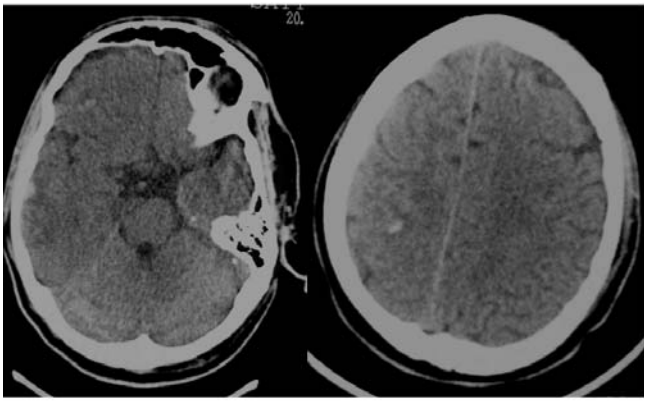

Figure 1. CT scan performed after the episode of seizures showing multiple contusions in right frontal, temporal and parietal lobe.

intravenous diazepam and at the same time a loading dose of phenytoin was administered. His immediate CT scan showed multiple specks of contusions with mild cerebral oedema (Figure-1). Serum electrolytes, X-ray chest and D-dimer study were normal. According to literature there is a well defined subgroup of patients with a significantly elevated risk to develop post-traumatic epilepsy. These include patients with evacuation of a subdural haematoma, surgery for an intracerebral haematoma, low Glasgow coma scale (3 to 8), early seizures, especially delayed early seizures, time to following commands of a week or more, untreated depressed skull fracture, dural penetrating injury, at least one non-reactive pupil and lesions involving the parietal cortex.[4,5] In these high risk patients the prophylactic use of anti-epileptic drugs is recommended for a short period, which prevent immediate and early seizures.[6] There is some evidence that very early treatment might be able to stop the neurochemical epileptogenic cascade at its origin, however in the absence of high risk factors the drug therapy should be instituted only after the first late unprovoked seizure.[1] In our patient the initial examination and normal CT findings suggested low risk of developing seizures and he was managed accordingly. However, it is a well known fact that the initial CT scan may be normal and patients can develop parenchymal lesions over a period of time; and can fall into the category of high risk group as in the present case. How to identify this group and anticipate the occurrence of post-traumatic seizures is still a challenge particularly in a patient who is recovering and has a normal initial CT scan.

Amit Agrawal[1], Rafael Cincu[2], S. R. Joharapurkar[3], Kaustubh Sarda[4]

Department of Surgery[1,3,4], Datta Meghe Institute of Medical Sciences, Sawangi (Meghe), Wardha (India), Department of Neurosurgery[2], Miguel Servet University Hospital, Zaragoza, Spain,

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