Evolution of epileptic encephalopathy in an infant with non-accidental head injury

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To the Editor

I have 2 comments on the interesting case report by Koul et al.[1]

First, Koul et al[1] stated in their study that even on detailed discussion with the parents of their studied patient, they could not identify the underlying reason for the child abuse (CA). I presume that the following might be the plausible explanation. It is well known that parents of children with epilepsy, like parents of children with many other chronic conditions, are faced with a constant psychological tension as childhood epilepsy has a severe impact on parental quality of life (QOL) and psychological health. This tension can lead to decreased ability to cope as evidenced by increased stress levels, negative mood states, and impaired family functioning.[2] Factors significantly correlated with altered parental QOL were noticed to include seizure control, visit status, anxiety, depression, employment, cost of epilepsy, status epilepticus, drug side effect and, age of parents.[3] As a result, it is not surprising to notice that CA can be the net result of long term parental failure to cope properly with caring for an epileptic child as in the studied patient by Koul et al.[1]

Second, obviously there is a link between infantile spasms (IS) and non-accidental head injury.[4] Koul et al[1] stated in their study that an EEG carried out at the age of 10 months and 20 days, 5 months after the first EEG revealed features of epileptic encephalopathy in the form of hypsarrhythmia (HA) and burst suppression. I presume that the modified EEG picture generally indicated IS. It is well known that IS typically reveals interictal EEG of HA. However, many EEG variants could be noticed in IS. In a Russian study,[5] an evaluation of the interictal EEG characteristics in a cohort of children with IS was carried out. The following EEG types were detected: typical HA (16.7%), different variants of modified HA (72.9%), a presence of focal epileptiform discharges, but not in the form of modified HA with focal component (6.2%), and an absence of epileptiform discharges (4.2%). The typical and modified HA cases ratio was estimated as 18.6:81.4%. Among the patients with modified HA, the following variants were detected: synchronized variant of modified HA (35.3%), asymmetric regional or unilateral HA (42.9%), HA with partial component (45.7%), and HA with persisting "suppressive-burst" pattern (20%). Unfortunately, it was not feasible to sequentially monitor the EEG pattern of the studied patient, as the parents did not attend regular follow-up.

Reply from the Author

Many thanks for the comments of Prof. Al-Mendalawi. We appreciate his keen interest in our paper. The main purpose of our paper was to report a child with epileptic encephalopathy (infantile spasms [IS]), following non-accidental head injury.[1]

Comment 1: Long term paternal failure to cope properly with an epileptic child. In our case report, it is highlighted that the seizures were seen in the child after the abuse. It is well known that abused children can present with seizures as a first manifestation.[4] After we noted subdural hematoma on his first presentation, the parents refused further work up and the child was discharged on antiepileptic medications. On several occasions, the parents stopped antiepileptic medications. Though the diagnosis of epilepsy could have had a severe impact on the parental QOL, their failure to administer medications was baffling to us. It is also a known fact that perpetuators of the abuse are not recognized most of the time.

Comment 2: Link between infantile spasms and non-accidental injury. There were only 2 citations before our paper. One of these was the conference abstract by the same authors,[6] which is now published.[4] Our article appeared soon after the paper Prof. Al-Mendalawi has quoted. Our article could have appeared earlier; however, it was refused by several journals before being accepted by Neurosciences. We appreciate the Neurosciences editorial team and their reviewer's for recognition of this rare association. We mentioned in our discussion that any injury to the brain during the wiring phase (unbalanced maturation during the critical brain development time) can result in IS.[7] Our article highlighted sequential evolution of the EEG changes from initial normal to the epileptic encephalopathic pattern (IS). Other reports only presume this.

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