

Posttraumatic epilepsy: current and emerging treatment options

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Dear editor

We have read with great interest the recent paper by Szaflarski et al¹ published in *Neuropsychiatric Disease and Treatment*. Patients suffering from traumatic brain injury (TBI) have three times greater risk than the general population to have epilepsy. This makes the TBI one of the most important causes of secondary epilepsy.² In the paper by Szaflarski et al¹ the authors highlighted the epidemiological relevance of this topic and describe the therapeutic options in detail.

The treatment indicated for TBI may involve multiple medications and/or surgical procedures. However, none of these treatments has as its purpose to prevent the occurrence of late posttraumatic epilepsy. Antiepileptic drugs currently in clinical use are indicated for the control of seizures that may occur but not to prevent progression to epilepsy. A strong point of the paper refers to future directions in the treatment, including new insights into epileptogenesis. However, despite widespread discussion of treatments in this paper some considerations were not included. An important consideration should be the mechanisms involved in the genesis of posttraumatic epilepsy, including some evidence of a genetic component and posttraumatic epilepsy,³ important information about inflammatory mechanisms and blood–brain barrier dysfunction associated with this posttraumatic disorder,⁴ and their impacts in future directions of the treatment. It should also be considered that a number of compounds successfully tested at the preclinical level, despite lacking clinical relevance, might bring insights in terms of mechanisms of action for antiepileptogenesis or disease modification.⁵ Finally the authors failed to mention strong preclinical evidence indicating that neuroprotection, per se, does not influence antiepileptogenesis nor provide disease modification of posttraumatic epilepsies.⁶ These small caveats, however, do not take away the main relevant messages and discussion raised by Szaflarski et al.

Disclosure

The authors report no conflicts of interest in this communication.

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Authors' reply

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Dear editor

We would like to thank Drs Paiva and Mello for their interest in our recent review focusing on the current and emerging treatment options for individuals with traumatic brain injury.¹ We agree with the authors of the letter that in patients with traumatic brain injury, many factors including genetics,

blood–brain barrier dysfunction, mechanism of action, and the neuroprotective properties (or lack thereof) of the used compound, may be important. With that in mind, we need to recognize that the goal of our review was not to address these issues but rather to provide a detailed comparison of the clinical data on the two anticonvulsant drugs most commonly used for seizure prevention in the setting of acute traumatic brain injury – phenytoin and levetiracetam – with only a snapshot of other compounds. A discussion focusing on genetics, blood–brain barrier dysfunction, or other aspects mentioned previously was clearly outside of the scope of our review.

Disclosure

The authors report no conflicts of interest in this communication.

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