

The Role of Blood-Brain Barrier Breakdown Following Traumatic Brain in Post-Traumatic Epilepsy

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Abstract

Traumatic brain injury (TBI) refers to a brain injury caused by an external mechanical force such as an impact to the head, concussive forces, acceleration-deceleration forces, blast injury, and a projectile such as a bullet. Traumatic brain injury is recognized as a critical public health problem worldwide, TBI is accompanied with mortality and morbidity with an occurrence of approximately 200 cases per 100,000 people a year. It is also a known major risk factor for focal epilepsy. The incidence of post-traumatic epilepsy (PTE) ranges from 2–50% in different studies, accounting for approximately 20% of symptomatic epilepsies. Seizures may occur immediately following the trauma, though PTE usually develops several months and even years later. While immediate post-traumatic seizures may be successfully treated with antiepileptic drugs, the mechanisms underlying the development of PTE remain unknown with no means for preventing it. The central nervous system is protected by the function of the blood-brain barrier (BBB), which regulates the passage of blood constituents in and out of the brain extracellular space. It seems that an increase in BBB permeability may be associated with the pathogenesis of neurological disorders. However, only recent animal experiments directly showed that primary prolonged opening of the BBB leads to the development of delayed, long-lasting epileptiform activity. Furthermore, it has been suggested that the most common serum protein, albumin may underlie astrocytic activation and dysfunction, further leading to neuronal hyper synchrony and accumulated neuronal loss. On the other hand previous clinical studies showed that altered permeability is observed in neurological patients.

Keywords: Blood-Brain Barrier, Traumatic Brain, Post-Traumatic Epilepsy.

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