Post Traumatic Epilepsy: A review of triggers and potential treatments after brain injury

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Introduction

In 2010, the Center for Disease Control and Prevention reported that nearly 2.5 million traumatic brain injuries occurred either in isolation or in conjunction with another injury. The results and long-term effects of such injuries span a multitude of outcomes, with research acknowledging the increased likelihood of seizures following TBI. Additionally, there is an increased potential for the development of epilepsy, also referred to as posttraumatic epilepsy (PTE).

PTE is defined as two or more seizures, thought to be related to the injury itself, that follow a traumatic injury occurring later than one week following the injury. Patients with TBI are at higher risk for the development of epilepsy relative to the general population. PTE is a significant and debilitating issue that complicates recovery after brain injury.

Age, previous seizures, smoking, genetics, timing of seizures and types of injury, all play a role in the potential for the development of PTE. Furthermore, these factors create a wide range of outcomes which further complicate risks and treatment.

With risk identification, clinicians and researchers alike may be able to more effectively identify treatment and rehabilitation options. As more research is conducted, a greater understanding of new technology and potential for the improvement of treatment is established.

In developing and perfecting these treatments, hopefully a more widespread understanding and protocol may be instituted for individuals at high risk for the development of PTE.

Objectives

1. Overview of PTE following traumatic brain injury (TBI).
2. Classifications and risks associated with the development of PTE after TBI.
3. Treatment options
4. Future research directions will be suggested in order to improve upon the dearth of knowledge in this area and further build and establish well utilized treatment options.

Epidemiology

- Statistical likelihood ranges anywhere from 3-20% depending on severity of injury—If severe- may increase risk to 40% (1, 2, 3)
- Contributing factors: age, genetics, previous seizures, type of injury and timing of seizures all play a role in the development of PTE (1)

Immediate Onset

- Occurs within 24 hours
- Usually result of immediate repercussions as opposed to long term neural alterations
- Often excluded from further analysis(4, 6, 7)

Early Onset

- Less than a week after injury
- Research has found a link to late seizures (6, 8, 9)
- Risk factors for early onset seizures comprise: acute intracerebral hematoma, acute subdural hematoma, younger age, increased injury severity, and chronic alcoholism (1)

Late Onset

- More than a week—highest indicator for PTE
- Risk factors include early posttraumatic seizures, brain contusion, subdural hematoma, prolonged posttraumatic amnesia (> 3 days), increased injury severity, and older than 65 years of age at the time of injury, along with many of the factors listed for early onset seizures (1, 2, 4, 10)

Biological Basis

Potential Indicators:
- mossy fiber sprouting (12-14)
- increased electroencephalogram activity (6)
- damage to the hippocampus or cortex (7, 12)
- neuronal cell loss (12)
- blood brain barrier disruption (8,15)
- inflammation (14)

Treatment Options

Stepwise process that begins with:
- Anti-Epileptic Drugs (ineffective for late onset) (3, 5, 6, 8)
- Often followed by (if resistant to drug treatment):
  - Resection surgery of the anterior temporal lobe on the non-dominant side (5)
  - Deep Brain Stimulation (5)
  - Vagus Nerve Stimulation (17)
  - Hypothermia Therapy (16)

Future Directions

- Additional imaging and EEG observation
- Monitoring within the following years— watch for predictive indicators (11, 13)
- Additional research regarding potential treatments— both what is currently available and those in preliminary stages
- Transitions to clinical trials are vital in order to make further strides (5)

References: