TRAUMATIC BRAIN INJURY GUIDELINE
Management of Seizures in the Patient with -Traumatic Brain Injury
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I. Definition, Assessment, Diagnosis

A. Definition:
1. "Epileptic Seizure is a clinical manifestation that is presumed to be the result of an abnormal and excessive discharge of a set of neurons in the brain. The clinical manifestation consists of sudden and transitory abnormal phenomena, which may include alterations of consciousness, motor, sensory, autonomic, or psychic events, perceived by the patient or an observer" (Guidelines for the Epidemiologic Studies on Epilepsy, International League Against Epilepsy, *Epilepsia, 1993*)
2. Traumatic Brain Injury is a common cause of epilepsy and is the leading cause of epilepsy in young adults (Annegers, 1996).
3. Post-traumatic seizure (PTS) refers to a single or recurrent seizure episode occurring after Traumatic Brain Injury (TBI) (Zasler, Katz, & Zafonte, 2007, p.443).
   a. Post-traumatic seizures can be further classified as:
      1) *Immediate* (within 24 hours of injury onset)
      2) *Early* (< 1 week after injury)
      3) *Late* (> 1 week after injury) (Teassell, Bayona, et al., 2007)
      4) Other potential seizure-inducing processes, such as but not limited to: premorbid epilepsy, electrolyte disturbances, alcohol withdrawal, medications, etc. must be excluded as etiologies.
   b. Frequency of occurrence amongst patients who have been hospitalized for TBI has been estimated to be 5-7%, with increased risk for severe TBI (11%) and marked increase for TBI from penetrating head injury (35-50%) (Yablon, 1993).
   c. Risk factors for predisposition of development of PTS include:
      1) Severe initial TBI
      2) Penetrating head injury
      3) Intracranial bleeds (epidural and subdural)
      4) Increasing age
      5) Premorbid alcohol abuse
      6) Family history of PTS (Zasler, Katz, & Zafonte, 2007, p.452-453)
   d. The risk of developing PTS is highest in the first 24 months following TBI (Yablon, 1993).

B. Assessment
1. Patients may exhibit any mix of classically associated seizure symptoms, depending on the area of epileptogenesis, such asa:
   a. Convulsions
   b. Repetitive motor activities
   c. Blank, staring spells
   d. Consciousness may or may not be preserved, depending on the subtype of seizure that is occurring
   e. Visual disturbances or hallucinations
   f. Auditory disturbances or hallucinations

g. Changes in mood or affect
h. Dysesthesias
i. Electrical sensations
j. Numbness (Zasler, Katz, & Zafonte, 2007, p. 446-447)

2. Presence of TBI can mask signs and symptoms that would have been more easily recognized prior to injury in the same patient. Thus, observation for subtle clues and symptoms is essential to diagnosis.
3. New observations of focal motor, sensory, or communication deficit should raise clinical suspicion for recently unwitnessed Post-Traumatic Seizure (Zasler, Katz, & Zafonte, 2007, p. 447).

C. Diagnosis
1. Diagnosis of seizure generally begins with observations. Symptomatic reports from the patient's family and observations by an experienced clinician can aid in potential classification of seizure activity.
2. Definitive diagnosis is made with an electroencephalogram (EEG).
a. Confirmation via EEG requires capture of the event to make diagnosis. Subsequently, absence of findings does not necessarily rule out the possibility of seizure activity (Binnie & Stefan, 1999).
b. To further evaluate seizure events, it may be necessary to refer patients to an epilepsy monitoring unit, where video electroencephalogram (VEEG) can be performed (Cascino, 2002). VEEG utilizes a longer period of observation and recording in attempt to capture events that may be missed on a standard EEG. Admissions for this process can typically span 24-72 hours.
c. VEEG can also be helpful in differentiating true epileptic events from psychogenic non-epileptic seizures (NES). Psychogenic NES can have a similar clinical presentation to epileptic events, making diagnosis otherwise difficult. Furthermore, TBI can have sequelae, such as behavior disorders and depression, that can further cloud the diagnostic picture (Hudak, Trivedi, et al., 2004).
3. Postictal prolactin measurement can also be helpful in clarifying the diagnosis. Several types of seizures exhibit increased prolactin levels in the hour following the event. Due to physiologic variation among patients, a baseline lab is recommended to be drawn within a few days of the event at approximately the same time of day as the event (Zasler, Katz, & Zafonte, 2007, p. 455,).

II. Management and Treatment Recommendations

A. Immediate management should consist of measures to protect the patient from injury during the immediate seizure and postictal state. These include:
1. Placing the patient in a safe position from which they cannot fall. Remove any hazards, such as sharp objects from the immediate area.
2. Position patient on his or her side to help protect the airway if he or she is unconscious.
3. Record the time of onset and length of seizure.
4. Do NOT attempt to place anything in the mouth of the unconscious patient, including medications.
5. Do NOT attempt to hold the patient down if they exhibit convulsions.
6. Remain with the person until the seizure has ended. This may span a few seconds or several minutes.
7. If concern exists for the patient's breathing status or an injury is suspected,

immediately call emergency medical services (Shafer, 2014).

B. A patient that is in status epilepticus requires immediate emergency medical treatment. Status epilepticus be defined as a continuous, generalized, convulsive seizure lasting >5 min, or two or more seizures between which the patient does not return to baseline consciousness (Lowenstein & Alldredge, 1998).

C. Referral to a neurologist for further workup and appropriate selection of anti-epileptic drugs (AEDs) is recommended.

D. For a patient who has recalcitrant, late PTS, evaluation for surgical resection of the epileptic foci may be necessary for treatment.

E. The state of Arkansas does not currently require that physicians report epilepsy to the state Office of Driver Services; the onus to do so resides with the patient. The Office of Driver Services may impose restrictions to ensure the safe operation of the motor vehicle. A patient must be seizure-free for 12 months and pass a medical evaluation by a state-licensed physician that is reviewed and approved by the Office of Driver Services for reinstatement of driving privileges ("State driving laws database," 2014).

III. Prevention and Education

A. Anti-epileptic Drugs (AEDs) for prevention of PTS have been evaluated to varying degrees by randomized controlled trial. Currently, prophylactic treatment of PTS is not routinely recommended beyond one week following head injury ("Role of antiseizure prophylaxis," 2000).

   a. Phenytoin is the most rigorously tested AED for PTS
   b. Evidence supports that it is useful for prophylactic treatment of Early Post-Traumatic Seizures (those that occur during the first week following injury).
   c. Evidence does NOT support phenytoin's use for the prevention of Late Post-Traumatic Seizures, as it does not demonstrate any significant protective effect after the Early PTS period (Temkin et al., 1990).
   d. It is important to recognize that phenytoin displays significant drug-drug interactions, has a robust side-effect profile, and requires monitoring of serum drug levels, which must be taken into account when choosing this medication.

   2. Carbamazepine also demonstrates improvement in Early PTS prophylaxis but not in Late PTS prophylaxis (Glotzner, Haubitz, Miltner, Kapp, & Pflughaupt, 1983).
   2. Carbamazepine requires monitoring of serum drug levels and has a large interaction profile with other medications.
   3. Levitiracetam is anewer generation of AED that has several advantages over the older generation, including decreased drug interaction and side-effect profile. It requires no loading dose or typical serum monitoring. . The efficacy has not yet been fully evaluated in comparison to other agents such as phenytoin with EEG monitoring and further study is needed before this can be recommended as standard practice. (Jones et al., 2008).

B. Beyond the scope of Early PTS prophylaxis, for patients who have experienced two or more PTS events, it is generally agreed that these patients should be treated with an AED (Zasler, Katz, & Zafonte, 2007, p.456).

C. Trial of withdrawing from AED use may be considered after a two-year period with no seizure activity (Callaghan, Garrett, & Goggin, 1988).

D. It is also important to weigh the impact of AED medications on cognitive performance and safety profile against seizure risk. There is concern thatAED medications cause delay in

cognitive recovery following TBI and if this is a short-term or long-term effect (Zasler, Katz, & Zafonte, 2007).

This guideline was developed to improve health care access in Arkansas and to aid health care providers in making decisions about appropriate patient care. The needs of the individual patient, resources available, and limitations unique to the institution or type of practice may warrant variations.

Guideline Developers

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