Mini Review Article

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Occurrence, Suggestions and the Executives of Seizures following Ischemic and Hemorrhagic stroke

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Abstract

Review objective: I have presented a synopsis of the most recent research on the incidence and management of seizures following ischemic and hemorrhagic strokes in this review. In addition, it is identified unanswered questions regarding standard clinical care and guidelines to assist future studies aiming to improve seizure management in stroke patients.

Recent results: Studies show that there are more seizures after strokes, probably because more people are using continuous EEG monitoring and better ways to treat strokes. Seizures following a stroke are linked to prolonged hospitalization and increased mortality; As a result, seizure prevention and prompt treatment are crucial. Regardless of the cause, Anti Epileptic Drugs (AEDs) are the treatment of choice for recurrent seizures. However, there are currently no established guidelines for using AEDs to prevent strokes.

Summary: Seizures after stroke are getting more common. Risk factors for recurrent seizures and epilepsy following strokes, as well as the most effective treatment options, require additional research.

Keywords: Ischemic Stroke • Hemorrhagic Stroke • Cerebrovascular Disease • Seizures • Epilepsy • Post Stroke Seizures

Introduction

At least one non febrile seizure will occur in the lifetime of about 5% of the global population. It is estimated that 150,000 adults in the United States present annually with an unprovoked first seizure. There is a bimodal distribution of seizures, Seizures occur most frequently in children and people over the age of 60. The main source of seizures in grown-ups past the age of 60 is cerebrovascular illness and it has been displayed to represent almost half of recently analyzed epilepsy in this age bunch. In addition, hemorrhagic and ischemic strokes are currently one of the most common reasons for this patient population to be admitted to the hospital. Seizures are temporary, abnormally high levels of synchronous brain neuronal activity that can be triggered or not. While many idiopathic epilepsies exhibit unprovoked seizures, provoked seizures are typically brought on by acute or chronic changes in the central nervous system. These frequently include cerebral venous sinus thrombosis, posterior reversible encephalopathy syndrome, intra cerebral and subdural hemorrhages, and cerebrovascular diseases like ischemic stroke. It is important to note that patients who present with strokes may also have other less common causes of seizures, such as CNS infections, inflammation, systemic metabolic abnormalities (such as hypo/hyperglycemia, hypo/hypernatremia, and hypokalemia),alcohol toxicity and withdrawal, benzodiazepines and barbiturates, traumatic brain injury, and intracranial surgeries [1].

Discussion

Ischemic Shock

After an ischemic stroke, many short-term and long term changes in brain tissue can cause

seizures. Due to their high metabolic rate and oxygen demand, neurons are more sensitive to hypoxia than other cell types. Due to increased glutamate, there is a lower threshold for depolarization from local excites toxicity during the acute stages of neuronal ischemia. Additionally, cytotoxic edema and acute metabolic dysfunction are brought on by rises in intracellular Ca2+ and Na+ as a result of malfunctioning sodium and potassium ion pumps. Hypoxia is especially harmful to the pyramidal neurons in the cortical layers that produce a measurable electrical signal over the scalp by generating postsynaptic excitatory and inhibitory potentials. Ischemia causes pyramidal dysfunction quickly, and Electroencephalographic (EEG) changes, such as slowing, typically appear within five minutes of acute ischemia. In addition, it is believed that the hippocampus is an epileptogenic region that is particularly vulnerable to ischemic insults during global hypoperfusion and is a common cause of status epilepticus [2, 3].

Hemorrhagic Shock

Seizures after Intracerebral Discharge (ICH) may emerge because of mechanical impacts of the extending drain as well as aggravation of the cortex because of results of blood digestion intensely and from hemosiderin testimonies and gliotic scarring persistently. Patients with ICH may be at risk from seizures because a head injury that occurs during a seizure may exacerbate the hemorrhage. Seizures can also cause additional damage to the brain's unprotected, therefore more vulnerable area if a craniotomy is performed during large hemorrhages. A new meta examination reasoned that there is an expanded gamble of post hemorrhagic stroke epilepsy following [4].

Management of Post Hemorrhagic shock

Despite their widespread use, there are currently no accepted guidelines that recommend the preventative use of AEDs following ischemic or hemorrhage strokes. However, a 2009 study showed that phenytoin was associated with more complications than the group without prophylaxis; including worse functional outcomes at three months post ICH and a higher frequency of fevers (longer duration of medication use was associated with more febrile days). Additionally, there is a possibility of a drug and drug interaction between nimodipine, which is commonly used to prevent vasospasm in SAH, and enzyme inducing agents like phenytoin and Carbamazepine. Studies have demonstrated a significant decrease in plasma Nimodipine concentrations, indicating that taking these medications concurrently in SAH patients could be harmful. In contrast, due to its inhibition of enzymes, valproic acid's concurrent use has been shown to increase Nimodipine concentrations by 50%. This should be taken into consideration when these medications are used together [5, 6].

Secondary prevention

Regardless of the cause, pharmacologic treatment with AEDs is the standard of care for recurrent seizures. Compared to other forms of focal epilepsy, such as hippocampal sclerosis or cortical malformations, clinical trials have shown that stroke related epilepsy tends to respond better to AED treatment and has a better prognosis. Without any known predictive factors, these trials suggested that between 5% and 46% of stroke related epilepsies exhibited drug resistance. Seizures following a stroke can often be controlled with monotherapy alone. Better side effect profiles, fewer drug and drug interactions, and better tolerability characterize more recent AEDs. The risk of AED adverse events in post stroke patients ranges from 7 to 31%, so when selecting an AED, concurrent medication use and comorbid conditions must be taken into account [7- 10].

Conclusion

In summary, cerebrovascular disease is the most common cause of seizures in adults over 60. The incidence of stroke related seizures is rising as a result of recent advancements in stroke treatment. Seizures may occur after both ischemic and hemorrhagic strokes, but the risk is greater after hemorrhagic strokes than after ischemic strokes. A higher risk can be predicted by the location of the stroke, such as anterior circulation or temporal ischemic strokes, which have been linked to an increased risk of seizures; it is thought that subcortical and cortical hemorrhages are more likely to cause seizures than hemorrhages in deeper structures. In addition, a significant factor in determining the likelihood of developing epilepsy is the timing of the seizures in relation to the onset

of the stroke. Early seizures following a stroke are associated with a lower risk of subsequent epilepsy than late onset seizures because early seizures are thought to be the result of transient derangements and may not be related to the development of a seizure focus as seen in later seizures.

Acknowledgement

None

Conflict of Interest

None

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