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Seizing over the Conundrums of SAH Seizure Prophylaxis

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Abstract

Aneurysmal subarachnoid hemorrhage (SAH), a potentially catastrophic stroke, which occurs at a relatively younger age (median 55 years), afflicts over 30,000 patients a year in the USA. It accounts for 3% to 5% of all strokes and 4.4% of deaths from stroke belong to this group of patients. It carries high rate of morbidity (25%) and case fatality (35%) in most series.

Keywords: Sub-arachnoid hemorrhage; Seizure; Prophylaxis; Nimodipine; Phenytoin; Drug interaction

Introduction

Survivals of the first few days of this disease may suffer delayed cerebral ischemia (DCI) an important contributor to poor outcome [1]. Different modalities and care strategies have been studied to improve outcome, however this disease remains with unacceptably high rates of cognitive and functional disability in survivors [2].

Different guidelines have been designed to provide care based on best available evidence to the SAH patients [3] and seizure prophylaxis has been discussed as one of the components of care in these guidelines. Although seizure or seizure-like manifestations are common at the onset of aneurysmal rupture, in the absence of significant complications and re-bleeding the clinical seizure is less frequent later in the course of this disease.

During 1950s and the 1970s the risk of seizure was reported to be as high as 27% in these patients [4,5] which led to the current and generally accepted medical practice of prophylactic antiepileptic drugs (AED) use in patients with SAH. In these studies, approximately 40% of seizures occurred at the time of aneurysm rupture or following re-bleeding. Downward trends in the incidence of seizure in SAH have been noted in current times with the risk of only 5% to 8%.

This difference is possibly a reflection of more modern modalities of treatment, such as early aneurysm clip occlusion, endovascular coil treatment, and designated neurocritical care units [6,7].

Discussion

Although seizure prophylaxis, particularly with phenytoin, has been a common place in the care of SAH patients, there are areas of controversy such as more recent reports of poor longer term outcome in patients prophylaxed with phenytoin [8], use of other AEDs, the role of AED in prevention and management of non-convulsive seizure, significant AED adverse reactions and perhaps more concerning and less explored issues regarding AED drug interactions. One such interaction is between the two very important and concomitantly administered drugs, phenytoin and nimodipine, in SAH management.

Phenytoin along with other AEDs carbamazepine and phenobarbital can induce hepatic metabolism resulting in reduced serum concentration and effects of nimodipine up to staggering 80% and concomitant use of these agents with nimodipine is contraindicated by some reports [9,10]. Since the process of enzyme induction usually requires synthesis of new enzymes, it manifests with some delay after the exposure to the inducing agent [11,12]. This effect becomes brisker after a week when the enzyme cytochrome P450 induction by phenytoin is at its maximum. De-induction follows a longer time course about 2 weeks [13].

Conclusion

Along with the increasing incidence of adverse drug reaction by prolong use of phenytoin, particularly in the critically ill SAH patients; this important drug warning for nimodipine could well be another strong reason to limit the use of phenytoin for seizure prophylaxis to three days as proposed in recent SAH guidelines [3].

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