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Editorial

SAH-Induced Vasospasm Refractory to Medical Treatment: A Mini Review and Role of Intraventricular Agents

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Editorial

Aneurysmal Subarachnoid Hemorrhage (SAH) is a common cause of intracranial vasospasm with major disabilities and death due to delayed cerebral ischemia with an overall incidence of 33-67% and 10-13% incidence of cerebral infarct [1,2].

The pathogenesis of vasospasm in SAH appears to be multi-factorial with various theories being put forward to explain the occurrence of vasospasm. Oxyhemoglobin, a breakdown product of hemoglobin from lysed RBC in the cisternal CSF, is believed to be the spasmogen involved in the genesis of vasospasm [1]. Lately, vasoactive endothelium derived peptide, called Endothelin-1 (ET-1), was isolated and its effect on smooth muscle contraction demonstrated. Oxyhemoglobin liberated after SAH appears to be simultaneously capable of activating the gene for this potent vasoconstrictor, increasing levels of ET-1 mRNA in the CSF and of also removing the influence of the potent vasodilator NO, the physiological antagonist of ET-1, from the blood vessel walls by direct binding. ET-1 and NO are important factors in maintaining dynamic equilibrium in vasomotor tone. Disequilibria between the vasomotor effects of these molecules results in unmitigated vasoconstriction.

Factors significantly associated with vasospasm are age of 40 to 59 years, history of hypertension, worse neurological grade, thicker blood clot on cranial CT scan obtained on hospital admission, larger aneurysm size, presence of IVH, and prophylactic use of induced hypertension [3]. Transcranial Doppler (TCD) and angiography are the two main tools for the diagnosis of vasospasm.

Various treatment options for vasospasm are calcium channel blockers (nimodipine, 60mg q4hr PO or 2mg/hr IV), triple-H therapy (hypertension, hypervolemia, hemodilution), balloon angioplasty, and papaverine infusion. Although the effectiveness has still not been proven, these modalities are widely accepted as rescue therapies [4-6]. The goal is CPP levels greater than 120 mm Hg and a central venous pressure over 10 mm Hg. Other proposed but less certain modes of treatment are the non-glucocorticoid 21 aminosteroid, tirilazad, and Magnesium sulfate.

However, all medical therapies have their own limitations, including risk of rebleeding of the unclipped aneurysm after triple-H therapy, unavailability of balloon angioplasty in urgent conditions in all neurosurgical institutions, etc. Therefore, there is still the need for further last resort therapies.

Intraventricular agents has been showed as appealing options as many of SAH patients already have ventricular drains in place, they can be safely administered at the bedside, and can be used in patients for whom conventional therapies are either not effective or not tolerated. The following agents have been used: 1) Calcium channel blockers: nicardipine (4 mg bid x 5-17 days) and nimodipine (bolus of 0.4 mg, followed by Intrathecal (IT) infusion of 0.4 mg/h) has been shown to reduce mean cerebral blood flow velocity over 24 h with continued administration [7-10]. 2) Sodium Nitroprusside (SNP): Some promising efforts have been reported with intraventricular SNP (10-40 mg bolus followed by 2-8 mg/h infusion or 2-5 mg q3-12 h) with benefits shown clinically and by TCD [11-17]. It is possible that this agent exerts its effects through release of NO and playing a role in ET-1/NO equilibrium. 3) Magnesium sulfate.

As SAH is a life threatening and debilitating disease with a high personal, social, and economic burden, it really needs more research to better understand the complications and promote therapeutic options.

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