Delayed cerebral ischemia after aneurysmal subarachnoid hemorrhage

Delayed cerebral ischemia (DCI) is the most consequential secondary insult after aneurysmal subarachnoid hemorrhage (SAH). It is a multifactorial process caused by a combination of the large artery vasospasm and microcirculatory dysregulation. Despite numerous efforts, no effective therapeutic strategies are available to prevent DCI. Follows some cases of SAH (usually), trauma, or other insults

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For decades, cerebral vasospasm triggered by the amount of blood in the basal cisterns was regarded as causal for delayed cerebral ischemia [1)]

Risk factors

Failure of cerebral autoregulation has been shown in patients with aSAH even before vasospasm sets in and contributes to delayed ischemic neurological deficits (DIND) along with vasospasm [2)].

Delayed ischemic neurological deficit (DIND) due to symptomatic vasospasm is a major cause of morbidity and mortality after aneurysmal subarachnoid hemorrhage (aSAH), most likely because of a decreased availability of nitric oxide (NO) in the cerebral microcirculation [3)].

The VASOGRADE: is a Simple Grading Scale for Prediction of Delayed Cerebral Ischemia After Subarachnoid Hemorrhage [4)].

Intracerebral hematoma (ICH) was associated with an increased risk of DCI. Furthermore, adding the presence or absence of ICH to the modified Fisher Scale (mFS) improved the identification of patients at the highest risk for the development of DCI. Thus, a simple adjustment of the mFS might help to identify patients at high risk for DCI [5)].

Pathogenesis

Delayed cerebral ischemia after aneurysmal subarachnoid hemorrhage pathogenesis.

Pathophysiology

see Delayed cerebral ischemia after aneurysmal subarachnoid hemorrhage pathophysiology.

Diagnosis

see Delayed cerebral ischemia diagnosis.

Prevention

Delayed cerebral ischemia after aneurysmal subarachnoid hemorrhage prevention

Treatment

Delayed cerebral ischemia after aneurysmal subarachnoid hemorrhage treatment

Outcome

Delayed cerebral ischemia after aneurysmal subarachnoid hemorrhage outcome.

Case series

Fifty-seven of 176 prospectively studied patients with aneurysmal subarachnoid hemorrhage (SAH) developed delayed cerebral ischemia. Clinical features included hemispheric focal signs (13), decrease in level of consciousness (14), or both (30), and mutism (15). Forty-seven patients showed hypodense lesions on CT in one (19) or multiple vascular territories (22), or diffusely in one or both hemispheres (6). Twenty-eight had symmetric decrease in ventricular size. Of 18 autopsied patients, only 1 had a purely univascular lesion. The clinical, CT, and pathologic features suggest that delayed cerebral ischemia after SAH is a multivascular or diffuse process in most patients [6].

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