Cerebral vasospasm (CV)

Cerebral vasospasm, is characterized by angiographic narrowing of arterial vessels, which can be symptomatic and asymptomatic [1].

Epidemiology

Cerebral vasospasm (CVS) is the most common neurological complication after aneurysmal subarachnoid hemorrhage (aSAH) and associated with poor functional outcome and mortality.

Angiographic vasospasm is detected in 30 to 70% of patients during the first 5 to 14 days after hemorrhage [2] [3].

Among these patients, 50% with detected vasospasm in angiography suffer from delayed cerebral ischemia, of whom 15 to 20% suffer from stroke or die [4] [5].

Although the development and prevalence of cerebral vasospasm (CV) has been extensively investigated in adults, little data exist on the development of CV in children.

Children have a relatively high incidence of angiographically detectable, moderate-to-severe CV. Children rarely develop symptomatic CV and have good long-term outcomes, perhaps due to robust cerebral collateral blood flow. Criteria developed for detecting CV with TCD ultrasonography in adults overestimate the prevalence of CV in children. Larger studies are needed to define TCD ultrasonography-based CV criteria for children [6].

Classification

Cerebral Vasospasm Classification

Etiology

Cerebral Vasospasm Etiology

Pathophysiology

A number of pathological processes have been identified in the pathogenesis of vasospasm including endothelial injury, smooth muscle cell contraction from spasmogenic substances produced by the subarachnoid blood clots, changes in vascular responsiveness and inflammatory response of the vascular endothelium.

The pathophysiology on cerebral vasospasm and delayed cerebral ischemia (DCI) remains poorly understood. Much research has been dedicated to finding genetic loci associated with vasospasm and ischemia.

In a study, endothelial nitric oxide (eNOS VNTR) and haptoglobin (Hp) polymorphisms appear to have the strongest associations with delayed ischemic neurologic deficit (DIND) and radiographic vasospasm, respectively [7].

The pathogenesis of vasospasm involves endogenous spasmogens including oxyhemoglobin and endothelin. These are believed to inhibit nitric oxide (NO) synthetase and subsequently reduce the level of endogenous vasodilators, thereby producing vasospasm [8] [9].

Diagnosis

see Vasospasm diagnosis.

Complications

Pulmonary edema, myocardial ischemia, rebleeding, rupture of a new aneurysm, vasogenic edema/hemorrhagic infarction insetting of compromised BBB.

In 30 to 40% of patients, CVS leads to cerebral infarction with neurologic deficit and death [10] [11] [12].

Treatment

see Cerebral Vasospasm Treatment.

Guidelines

see Cerebral Vasospasm Guidelines.

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