Angiographic vasospasm

Angiographic vasospasm as a complication of aneurysmal and other types of subarachnoid hemorrhage (SAH) that was identified about 1953.

Angiographic vasospasm develops between 5 and 15 days after the initial hemorrhage, and is associated with clinically apparent delayed ischemic neurological deficits (DID) in one-third of patients.

It is now hypothesized that angiographic vasospasm contributes to delayed cerebral ischemia (DCI) by multiple pathways, including reduced blood flow from angiographic vasospasm as well as microcirculatory constriction, microthrombosis, cortical spreading ischemia, and delayed effects of early brain injury. It is likely that other factors, such as systemic complications, effects of the subarachnoid blood, brain collateral and anastomotic blood flow, and the genetic and epigenetic makeup of the patient, contribute to the individual's response to SAH. Treatment of aneurysmal SAH and DCI includes neurocritical care management, early aneurysm repair, prophylactic administration of nimodipine, and rescue therapies (induced hypertension and balloon or pharmacologic angioplasty) if the patient develops DCI. Well-designed clinical trials of tirilazad, clasozentan, antiplatelet drugs, and magnesium have been conducted using more than a 1,000 patients each. Some of these drugs have almost purely vascular effects; other drugs are theoretically neuroprotective as well, but they share in common the ability to reduce angiographic vasospasm and, in many cases, DCI, but have no effect on clinical outcome. Experimental research in SAH continues to identify new targets for therapy. Challenges for the future will be to identify the most promising drugs to advance from preclinical studies and to understand why clinical trials have so frequently failed to show drug benefit on clinical outcome. Similar issues with treatment of ischemic stroke are being addressed by suggestions for improving the quality of experimental studies, collaborative preclinical trials, and multinational, multicenter clinical studies that can rapidly include many patients and be large enough to account for numerous factors that conspire to disrupt clinical trials [1)].

Pathophysiology

Is not fully understood but appears to involve structural changes and biochemical alterations at the levels of the vascular endothelium and smooth muscle cells. Blood in the subarachnoid space is believed to trigger these changes. In addition, cerebral perfusion may be concurrently impaired by hypovolemia and impaired cerebral autoregulatory function. The combined effects of these processes can lead to reduction in cerebral blood flow so severe as to cause ischemia leading to infarction.

Treatment

The strategy to treat asymptomatic angiographic cerebral vasospasm following subarachnoid hemorrhage (SAH) is controversial.

Shimamura et. al, treated 281 aneurysmal SAH cases, with postoperative angiography performed 9 ± 2 days after the onset of SAH. Four asymptomatic cases received intra-arterial (IA) injection of vasodilator due to angiographic vasospasm. All cases improved vasospasm immediately following intervention. But all cases turned symptomatic within 48 hours.

We retrospectively analyzed the time-density angiography curve and calculated the time to peak (TTP), mean transit time (MTT), and relative blood flow (rBF). Relative blood flow was calculated as follows. The integration of the value of the time-density curve for the artery was divided by the same value for the internal carotid artery multiplied by the MTT.

The decrease in TTP and MTT for the etiologic artery was similar to that of the nonetiologic artery. But the improvement in rBF for the etiologic artery and nonetiologic artery was 10% and 17%, respectively. Blood supply to the spastic artery decreased due to iatrogenic steal.

Prophylactic IA injection of vasodilator in cases of asymptomatic vasospasm can produce symptomatic vasospasm [2].

1)
Loch Macdonald R. Vasospasm: my first 25 years-what worked? what didn't? what next? Acta Neurochir Suppl. 2015;120:1-10. doi: 10.1007/978-3-319-04981-6_1. PubMed PMID: 25366591.
2)
Shimamura N, Naraoka M, Matsuda N, Kakuta K, Ohkuma H. Prophylactic Intra-Arterial Injection of Vasodilator for Asymptomatic Vasospasm Converts the Patient to Symptomatic Vasospasm due to Severe Microcirculatory Imbalance. Biomed Res Int. 2014;2014:382484. doi: 10.1155/2014/382484. Epub 2014 Apr 16. PubMed PMID: 24822199.