

Delayed Diagnosis of Ruptured Aneurysms When Vasospasm is Present: Is Immediate Surgery Best?

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Cerebral vasospasm refers to delayed-onset cerebral arterial narrowing in response to blood clots left in the subarachnoid space after aneurysm rupture. It is rare in the first 72 hours from bleeding, peaks in severity about the end of the first week, and thereafter resolves within three to four weeks. When the arterial narrowing is severe or diffuse cerebral ischemia and infarction can occur. Although preventative measures and vasospasm treatments (such as induced hypertension) have reduced the incidence and clinical impact of vasospasm in the last decade, it continues to be an important cause of poor outcome. Some degree of vasospasm is common after subarachnoid hemorrhage (SAH), but severe and morbid vasospasm is most commonly seen in the setting of severe SAH and thick subarachnoid clots, usually correlating with poorer neurological condition.

In a recent study from a large urban hospital 12% of patients presenting with SAH were initially misdiagnosed and discharged home.¹ Persistent headache, aneurysm re-rupture and occasionally even symptomatic vasospasm bring these patients back for medical re-evaluation, and consequently aneurysms are sometimes detected at a time when vasospasm is already present. While the primary objective is always to repair the aneurysm as soon as possible, it has long been considered hazardous to perform surgery in the presence of vasospasm. This notion was supported in two separate studies on the timing of aneurysm surgery in which patients who underwent surgery toward the end of the first week from initial hemorrhage had significantly poorer outcomes than earlier or later surgery groups.^{2,3} Explanations included the possibility that surgical manipulation of vasospastic vessels exacerbates arterial narrowing, and the possibly increased susceptibility of injured brain with compromised blood supply to perioperative blood pressure instability or brain retraction.⁴

In this journal Drs. McLaughlin and Bojanowski review their experience with a uniform and uncompromising policy of as-early-as possible surgery for aneurysm ablation, vasospasm notwithstanding.⁵ From a consecutive series of 894 patients treated between 1990 and 2004 they retrieved the records of 40 patients that were considered to have clear angiographic vasospasm at the time of diagnosis and whose neurological condition was not complicated by repeat hemorrhages. Ten of these patients had symptomatic cerebral ischemia due to the vasospasm, and two had cerebral infarcts on CT scanning prior to surgery. Every patient except one proceeded to surgery within 48 hours of their angiographic diagnosis, albeit with care to avoid perioperative hypotension and with antiischemia treatment following surgery, namely calcium antagonists and various intensities of hypervolemic, hypertensive treatment in all patients, and angioplasty in two. As expected, symptomatic vasospasm was encountered postoperatively, but 10 of these 12 patients had ischemic symptoms prior to surgery. Cerebral infarcts occurred in six patients. In total, however, clinical

outcome associated with this strategy of operating despite vasospasm combined with attentive vasospasm treatment was "favourable" on the Glasgow Outcome Scale in 37 of the 40 patients assessed at three months. The one small subgroup that might stand out is the one with "early" vasospasm seen within the first several days of primary rupture. As suggested above these are uncommon patients, and the diagnosis of symptomatic vasospasm in this situation is difficult without baseline angiography and because of the possibility that neurological deficits are due to hemorrhagic brain injury rather than ischemia. Nevertheless this experience from the Centre Hospitalier de L'Universite de Montreal-Hopital Notre-Dame indicates that these patients are at very high risk of deterioration and death, and this finding is in agreement with recent reports (see authors' references 22 & 42) as well as my own personal experience in a handful of notable patients.

The authors demonstrate that in their excellent hands early surgery is feasible in many patients presenting with established vasospasm. If surgery is to be undertaken extra caution to minimize brain and vascular manipulation and to avoid perioperative hypotension is imperative, especially when vasospasm is symptomatic, severe, or diffuse. I fully expect that the new generation of neurovascular surgeons and interventionalists will continue to regard these patients as "high risk" for open surgical clipping, making the case for endovascular treatment even stronger in this small, particular group of patients, probably in combination with either therapeutic or prophylactic cerebral angioplasty. Clearly, every patient with his or her special features must be considered individually and carefully before such critical decisions are made. While management principles remain constant, individual patients are best approached with imagination and flexibility rather than policy.

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