

The relationship between ruptured aneurysm location, subarachnoid hemorrhage clot thickness, and incidence of radiographic or symptomatic vasospasm in patients enrolled in a prospective randomized controlled trial. Abla AA, Wilson DA, Williamson RW, *et al.* *J Neurosurg* 2014;120:391-7.

Cerebral vasospasm following subarachnoid hemorrhage (SAH) is responsible for significant delayed morbidity. In the present study, the authors researched whether different aneurysm locations resulted in different SAH clot burdens and whether any concurrent differences in ruptured aneurysm location and maximum SAH clot burden affected incidence of vasospasm.

The hospital charts of 250 SAH patients enrolled in a larger prospective treatment trial (Barrow Ruptured Aneurysm Trial) were reviewed. Exclusion criteria for this retrospective analysis included lack of aneurysm visible on cerebralangiography and death before treatment. All enrolled patients were admitted with the diagnosis of SAH and had ruptured aneurysms treated via coiling or surgical clipping. Most outcome and demographic variables were included as part of the prospective randomized controlled trial. Additional variables collected at follow-up included vasospasm data and maximum clot thickness.

Aneurysms were categorized into 1 of 6 groups: Intradural internal carotid artery aneurysms, vertebral artery (VA) aneurysms (including the posterior inferior cerebellar artery), basilar trunk or basilar apex aneurysms, middle cerebral artery aneurysms, pericallosal aneurysms, and anterior communicating artery aneurysms. Twenty-nine patients with nonaneurysmal SAH were excluded. Patients with pericallosal aneurysms had the least average maximum clot burden (5.3 mm), compared with 6.4 mm for the

group overall, but had the highest rate of symptomatic vasospasm (56% Vs 22% overall, OR = 4.9, RR = 2.7, $P = 0.026$). There were no significant differences in maximum clot thickness between aneurysm sites. Middle cerebral artery aneurysms resulted in the thickest mean maximum clot (7.1 mm) but rates of symptomatic and radiographic vasospasm in this group were statistically no different compared with the overall group. It was seen that aneurysms of the pericallosal artery (albeit just 9 patients), which ruptured into the interhemispheric fissure, had a statistically significantly greater risk of symptomatic vasospasm despite having the lowest mean clot thickness. Patients with stroke from vasospasm had higher mean clot thickness (9.71 Vs 6.15 mm, $P = 0.004$).

Vertebral artery (including the PICA) aneurysms had the worst 1-year modified Rankin scale (mRS) scores (3.0 Vs 1.9 overall, respectively; $P = 0.0249$). Patients with posterior circulation or pericallosal artery aneurysms had a lower proportion of good outcomes (mRS score 0-2).^[1]

The authors recently published a new scale [the Barrow Neurological Institute (BNI) scale] that grades the maximum thickness of SAH on axial computed tomography (CT) and is predictive of vasospasm incidence. The BNI scale was based on a single measurement reflecting the point of maximal thickness of subarachnoid blood. To obtain this measurement, the reviewing neurosurgeon examined each axial head CT for the slice showing the thickest-appearing subarachnoid blood in any subarachnoid space. This thickness of the subarachnoid blood was measured across the cistern or fissure (perpendicular to the direction of the cistern or fissure on axial cut) in the thickest-appearing region. The proposed grading is as follows; 1, no blood; 2, SAH ≤ 5 mm thick; 3, SAH $> 5-10$ mm thick; 4, SAH $> 10-15$ mm thick; and 5, SAH > 15 mm thick. The proposed BNI grading scale for SAH addresses a major shortcoming of the Fisher scale by accounting for increasing degrees of SAH thickness. The method of grading SAH based on a single measurement of maximal SAH thickness is simple, quantitative, and practical to apply in clinical settings. In a cohort of 218 aneurysmal SAH patients, they found the BNI scale to be superior to the Fisher

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scale in predicting symptomatic vasospasm, especially identifying patients at highest risk, with higher inter- and intra-observer agreement.^[2] In the present study, larger aneurysms were no more likely to have a higher maximum SAH clot burden using the BNI scale. Using the BNI scale, the higher the grade, the more likely the risk for vasospasm. Comparing patients with a BNI scale grade of 5 patients with a grade of 1 produced an odds ratio greater than 11.

The authors concluded that the location of a ruptured aneurysm minimally affects the maximum thickness of the SAH clot but is predictive of symptomatic vasospasm or clinical deterioration from delayed cerebral ischemia in pericallosal aneurysms. The worst 1-year mRS outcomes in this cohort of patients were noted in those with posterior circulation aneurysms or pericallosal artery aneurysms. Patients experiencing stroke had higher mean clot burden.^[1]

Sheth *et al.*, found no significant influence of intraoperative rerupture during open surgical clip placement on the rate of angiographic or symptomatic vasospasm. These results did not justify early aggressive treatment with blood pressure augmentation and intravascular volume expansion to prevent delayed cerebral ischemia. Also, vasospasm rates were similar in patients treated with coil embolization vis-a-vis clipping, suggesting that drainage of the subarachnoid blood during open surgical treatment offsets vessel irritation by direct handling.^[3] Aneurysm coiling and increased clot clearance were independently associated with decreased severity of angiographic vasospasm in multivariate analysis, although there were no differences in clot clearance between coiled and clipped patients.^[4]

The randomized, double-blind, placebo-controlled, phase 3, CONSCIOUS-2 trial in patients with aneurysmal subarachnoid haemorrhage undergoing surgical clipping showed that clazosentan, an endothelin receptor antagonist, at 5 mg/h had no significant effect on mortality and vasospasm-related morbidity or functional outcome.^[5] Oral administration of cilostazol was found effective in preventing cerebral vasospasm with a low risk of severe adverse events in a multicenter, randomized open label blinded endpoint trial. Further studies are required to assess the impact of these interventions on a larger scale.^[6]

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Brar SS, Aharonian V, Mansukhani P, *et al.* Haemodynamic-guided fluid administration for the prevention of contrast-induced acute kidney injury: The POSEIDON randomised controlled trial. *Lancet* 2014;383:1814-23.

The administration of intravenous fluid remains the cornerstone treatment for the prevention of contrast-induced acute kidney injury (CI-AKI). Data from the Prevention of Radiocontrast-Induced Nephropathy Clinical Evaluation (PRINCE) study indicate that increasing the urine flow rate (>150 mL/h) reduces the toxic effect of contrast media (CM). Volume expansion with normal saline might reduce contrast-mediated injury by expanding plasma volume, reducing renin activation and loss of nitric oxide, reducing production of reactive oxygen species, and through dilution of contrast within the tubular lumen.^[1] However, no well-defined protocols exist to guide fluid administration in this treatment.

In a randomised, parallel-group, comparator-controlled, single-blind phase 3 trial published in the *Lancet* 2014, Brar *et al.*, studied the efficacy of a new fluid protocol based on the left ventricular end-diastolic pressure (LVEDP) for the prevention of CI-AKI in patients undergoing cardiac catheterization (the POSEIDON trial). LVEDP is a haemodynamic parameter and can be used to establish intravascular volume status. The primary outcome was the occurrence of CI-AKI, which was defined as a greater than 25% or greater than 0.5 mg/dL increase in serum creatinine concentration. Between Oct 10, 2010 and July 17, 2012,