

Examining the correlation between cerebral arterial vasospasm, delayed cerebral ischemia, and response to hypertensive therapy

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Introduction

Delayed cerebral ischemia (DCI) is a major contributor to the morbidity of aneurysmal subarachnoid hemorrhage (aSAH). Cerebral arterial vasospasm (CVS) has traditionally been associated with DCI; however, recently mechanisms other than CVS have been linked to DCI.

Methods

All aSAH patients were prospectively collected and evaluated for DCI. CVS location and severity were evaluated with digital subtraction angiography (DSA), and infarcts detected on head CT. Induced hypertension as a treatment for DCI was analyzed in patients with and without CVS.

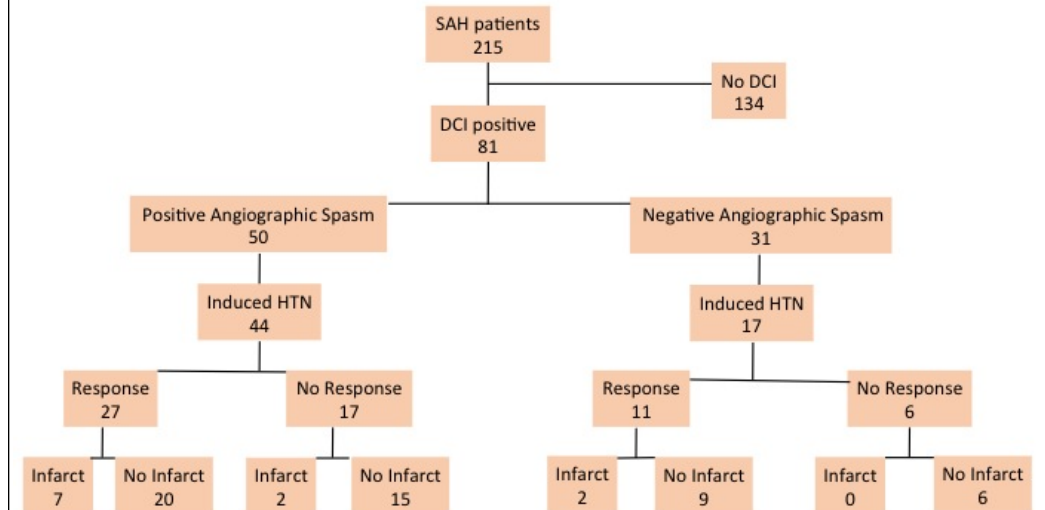
Results

215 aSAH patients were identified. Eight-one (38%) developed symptoms of DCI (60 with globally decreased mental status, 17 with focal motor deficit, 2 with cranial nerve palsy, and 2 with aphasia). CVS was absent in 31 of 81 DCI patients (38%). CVS location only correlated with expected anatomical location in 11 of 17 with focal motor deficit, 1 of 2 with cranial nerve palsy, and 1 of 2 with aphasia. Among patients with DCI, those with CVS were more likely to have an infarct (OR=3.2, 95% CI 0.64-15.8, P=0.19). Improvement with vasopressor treatment was not related with presence of CVS (OR=0.87, 95% CI 0.27-2.8, P=1.0). Among patients with DCI, those that improved with vasopressor treatment were more likely to have an infarct (OR=3.3, 95% CI 0.64-16.7, P=0.18).

Conclusions

A significant number of DCI cases either do not exhibit CVS (38%), or the location of CVS does not match the expected anatomic location of symptoms. Furthermore, response to hypertensive therapy does not correlate with presence of CVS. These findings suggest causes of DCI other than CVS. Non-CVS related DCI appears to respond to hypertensive therapy just as well as CVS-related DCI. Interestingly, patients who responded to hypertensive therapy had higher rates of infarction than those who did not, with all infarcts occurring in areas with CVS or adjacent to aneurysm treatment.

Table 1



Breakdown of aSAH patients, exhibiting symptoms DCI, presence of angiographic spasm, response to hypertensive therapy, and development of infarction.

Learning Objectives

By the conclusion of this session, participants should be able to: 1) characterize the relationship between cerebral arterial vasospasm and delayed cerebral ischemia, 2) Discuss, in small groups, whether the lack of vasospasm on digital subtraction angiography indicates other potential causes of delayed cerebral ischemia, and 3) Identify whether hypertensive therapy is a significant treatment for delayed cerebral ischemia.