

ANSWER TO PHOTO QUIZ

The brain has returned at ease, not suspecting another disaster

Keywords - subarachnoid haemorrhage, delayed cerebral ischaemia, induced hypertension, guidelines

DIAGNOSIS

Cerebral infarction due to delayed cerebral ischaemia after aneurysmal subarachnoid haemorrhage. Of note, the hypodensities involve both the medial cerebral artery and the posterior cerebral artery.

She was re-admitted to the ICU for hypertension induction with no clear effect on the neurological deficit. After two weeks she was discharged to the ward and four weeks after the haemorrhage she was discharged to a rehabilitation centre. At discharge she had neglect on the left side, an MRC grade 2 paresis of the fingers and cognitive challenges. Two years after the bleeding only a mild left hemiparesis remained.

Delayed cerebral ischaemia (DCI) is the most important cause of a poor outcome after aneurysmal subarachnoid haemorrhage (aSAH) in patients who survive the first 24 hours. DCI develops predominantly 4 to 10 days after SAH in ≈30% of survivors.^[1] To date, the L-type calcium channel antagonist nimodipine has been the only intervention proven to be effective in preventing DCI after aSAH.^[2] Several factors, such as angiographic vasospasm, microcirculatory constriction, microthrombosis, cortical spreading depression, and delayed cell apoptosis, are postulated to be associated with the occurrence of DCI. Loss of or abnormal autoregulation by conducting microvasculature, variations in collateral and anastomotic blood flow, and metabolic and (epi)genetic variations determine if angiographic vasospasm leads to tissue hypoxia and thus DCI. The final common pathway of the cause leading to DCI is a decrease in cerebral blood flow. Clinical signs of DCI may occur when the cerebral blood flow and thus the delivery of oxygen no longer meet the demand of the brain tissue in the setting of impaired cerebrovascular autoregulation.^[3]

After clinical signs of DCI occur, induced hypertension is a plausible but unproven therapeutic intervention.^[4-6] The primary rationale for the use of induced hypertension is that raising perfusion pressure may increase cerebral blood flow in high resistance vascular beds, increase collateral flow to ischaemic brain regions, or both and, therefore, elevate brain tissue oxygenation.^[4] Although the guidelines for the management of aSAH from the American Heart Association/American Stroke Association include a Class 1, level B

recommendation for induction of hypertension for patients with DCI unless the blood pressure is elevated at baseline or cardiac status precludes it, its presumed effectiveness is based on uncontrolled case series only and on general agreement that the treatment is useful and effective. This agreement is hugely based on opinion as at the writing of the guidelines there was not a single randomised controlled trial on this topic.^[5]

In the HIMALAIA study, aSAH patients with clinical symptoms of DCI were randomised to induced hypertension or no induced hypertension.^[7] The trial aiming to include 240 patients was ended, based on a lack of effect on cerebral perfusion and slow recruitment, when 21 patients had been randomised to induced hypertension, and 20 patients to no hypertension. With induced hypertension, the adjusted risk ratio for poor outcome was 1.0 (95% confidence interval 0.6-1.8) and the risk ratio for serious adverse events 2.1 (95% confidence interval 0.9-5.0). The study did not add any evidence to support induced hypertension, but due to lack of power also not against it.

The absence of any other comparative studies and the lack of effect on cerebral perfusion and a high rate of serious complications, the widespread use of induced hypertension in aSAH patients with DCI and the pertinent guideline recommendations may require reconsideration.

References

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