CASE REPORT

Convexity subarachnoid haemorrhage secondary to internal carotid stenosis: an indication for revascularisation

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SUMMARY

With increasing prevalence due to an ageing population, carotid artery stenosis is a significant cause of stroke morbidity and mortality. The indication for revascularisation treatment in symptomatic carotid stenosis is widely documented and accepted in the scientific community. However, treatment of asymptomatic carotid stenosis remains controversial. We report a case of a 78-year-old woman who was admitted with a convexity subarachnoid haemorrhage (cSAH) secondary to an asymptomatic high-grade carotid artery stenosis. Two months later, she suffered an atherothrombotic ischaemic stroke and was referred to surgery. Transcranial Doppler studies showed impaired cerebral vasoreactivity and, after endarterectomy, the patient developed a reperfusion syndrome; both findings consisting of exhausted collaterals as the underlying mechanism. We propose that cSAH secondary to a high-grade internal carotid artery stenosis is a high risk marker for stroke, and revascularisation therapy should be considered.

BACKGROUND

Significant carotid stenosis affects 7% of women and >12% of men by their eighth decade and causes 10% of all strokes.¹ However, in clinical practice, there is no consensus on how best to manage patients with asymptomatic carotid artery disease. For many years, based on randomised controlled trials for surgery—such as Asymptomatic Carotid Atherosclerosis Study (ACAS) and Asymptomatic Carotid Surgery Trial (ACST)—procedural management has been commonly recommended for stroke prevention.^{2–3} However, recent papers suggest that the widespread implementation of intensive medical therapy has diminished stroke risk markedly, up to 70–80%, in recent years.⁴ In this context, strategies to identify patients at high risk for stroke who might benefit from preventive surgery are needed. Interestingly, convexity subarachnoid haemorrhage (cSAH) has been associated with significant arterial stenosis in the ipsilateral cervical internal carotid artery (cICA) and also in intracranial stenosis such as in Moyamoya disease.^{5–8}

CASE PRESENTATION AND INVESTIGATIONS

A 78-year-old woman with a history of diabetes mellitus attended the neurology outpatient clinic due to a left asymptomatic cICA stenosis. She was taking antiplatelet therapy, statins and biguanides. During a 7-year follow-up, the carotid stenosis had

progressed from plaques without haemodynamic stenosis (<50%) to a significant stenosis narrower than 80%. The patient experienced a sudden onset of headache and dysarthria secondary to right central facial paresis. Brain CT (figure 1A) and MRI (figure 1B) showed a left cSAH. Cervical ultrasonography and CT angiography showed a 90% stenosis of the left cICA (figure 1C). Transcranial Doppler (TCD) showed intracranial haemodynamic repercussion with minimal collateral support via the circle of Willis and impaired cerebral vasoreactivity in the left anterior territory. Owing to the cortical bleeding, antiplatelet therapy was stopped for 2 weeks and then reintroduced. Two months later, the patient was again admitted with acute right lower extremity weakness due to an atherothrombotic ischaemic stroke in the left middle cerebral artery territory.

DIFFERENTIAL DIAGNOSIS

The patient was first admitted with an atraumatic cSAH. Two main patterns have been described. Young patients usually present with clinical and imaging findings suggestive of reversible cerebral vasoconstriction syndrome while in older patients the most frequent aetiology is cerebral amyloid angiopathy.⁹ Less frequent aetiologies include cortical vein thrombosis or posterior reversible leucoencephalopathy syndrome. Brain MRI and CT angiography ruled out these entities as they showed no microbleeds, no signs of cortical vein thrombosis, no posterior white matter lesions and no intracranial arterial narrowing. Cerebrovascular ultrasound and CT angiography revealed a high-grade cICA stenosis, which, in turn, has been reported as a cause for cSAH.

TREATMENT

When the patient was admitted with the ischaemic stroke, the cICA stenosis was considered symptomatic. According to current guidelines on the management of carotid artery disease, she was referred for left cICA surgery.

OUTCOME AND FOLLOW-UP

One week after the procedure, despite blood pressure thresholds of 140/90 mm Hg, the patient developed a hyperfusion syndrome with delirium and partial seizures. A CT scan showed petechial haemorrhages compatible with hyperperfusion (figure 1D). The patient was treated with steroids, more aggressive antihypertensive therapy and

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Figure 1 (A) Axial cranial CT showing a hyperdense cortical lesion corresponding with a left convexity subarachnoid haemorrhage. (B) Axial T2-weighted gradient echo sequence image showing subacute left sulcal subarachnoid haemorrhage. (C) Sagittal CT angiography of the neck showing a high-grade left internal carotid artery stenosis. (D) Axial cranial CT showing small petechial haemorrhagic lesions compatible with hyperperfusion.

antiepileptic drugs, with good outcome, and since discharge remains asymptomatic.

DISCUSSION

Atraumatic cSAH has been associated with intracranial and extracranial artery stenosis.^{5–8} The precise aetiology for this relationship remains unknown. A progressive cICA occlusion usually allows the development of collateral circulation. When the compensatory flow provided via the circle of Willis is inadequate, collateral pathways are recruited through extracranial to intracranial anastomoses and via leptomeningeal collaterals. These pial arteries join the cortical branches of the middle cerebral artery (MCA), anterior cerebral artery and posterior cerebral artery (PCA). A weakening of the arterial wall could explain the cortical bleeding, as was suggested in a previous cSAH report where dilated pial collaterals were identified by angiography.⁷ Previously reported cSAH cases always occurred on the symptomatic side and mainly in the MCA-PCA watershed.^{7–8} In the setting of a high-grade c-ICA stenosis, especially when there is no collateral support via the circle of Willis, the compensation flow is provided via the PCA. If an acute increase in cerebral blood flow is required, the hyperflow through the PCA may favour the rupture of dilated fragile pial PCA-MCA leptomeningeal collaterals.^{6–7} These dilated collaterals reflect an exhausted vasoreactivity and therefore could increase the risk of cerebral hyperperfusion syndrome after carotid revascularisation, as happened in our case.

Before considering a causal association of cSAH and cICA stenosis, more common aetiologies should be investigated first, mainly reversible cerebral vasoconstriction syndrome in young patients and cerebral amyloid angiopathy in older individuals.⁹ Since an exhausted cerebral vasoreactivity is the proposed pathophysiology, a TCD study should be performed, as an impaired result would support the causal link.

After the publication of the first prospective randomised trials, carotid endarterectomy plus best medical treatment was defined as the treatment of choice for significant asymptomatic ICA stenosis.^{2–3–10} On the other hand, recent studies suggest that current best medical therapy has significantly dropped the average annual stroke to rates even lower than those achieved with surgery.⁴ However, further prospective, randomised trials are needed to assess the best management of asymptomatic ICA stenosis, such as the Carotid Revascularization Endarterectomy Versus Stenting Trial 2 (CREST 2), which is currently ongoing.¹¹

Since the association between cSAH and high-grade stenosis does not seem accidental, we propose that patients with asymptomatic carotid stenosis who have a spontaneous cSAH should be reclassified as ‘quasi-symptomatic’ or high-risk asymptomatic and revascularisation therapy should be considered.

Learning points

- ▶ Carotid artery disease (CAD) should be ruled out in convexity subarachnoid haemorrhage (cSAH).
- ▶ cSAH due to CAD reflects exhausted cerebral vasoreactivity.
- ▶ cSAH secondary to CAD should be considered a risk factor for ischaemic stroke.
- ▶ When cSAH occurs in the setting of CAD, revascularisation treatment should be considered.

Competing interests None declared.

Patient consent Obtained.

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