Cerebral vasospasm and primary Raynaud’s phenomenon

Editor—A 45-yr-old woman presented for coiling of a right middle cerebral artery aneurysm 16 days after developing a sudden occipital headache associated with neck stiffness and nausea. Although initial scans showed no evidence of a bleed, oxyhaemoglobin and bilirubin were present in the cerebrospinal fluid and CT angiography confirmed the presence of aneurysms of the right middle cerebral and left ophthalmic arteries. Past medical history included primary Raynaud’s phenomenon (PRP), migraine, osteoarthritis, and a 30 pack-year smoking history. Physical examination was unremarkable.

We sited a radial arterial line before induction with propofol and remifentanil. Anaesthesia was maintained with sevoflurane and remifentanil and a metaraminol infusion was used to preserve preoperative mean arterial pressure (MAP). The patient was warmed throughout the 3 h procedure with a forced warm air blower. Angiography showed marked spasm of the right internal carotid artery (Fig. 1), which was treated with 0.5 mg intra-arterial nimodipine. After relief of the spasm, the aneurysm was packed with three coils and a further 0.5 mg of nimodipine was given at the end of the procedure. On recovery, the patient was found to be GCS 15/15 with no focal neurological deficits. She was monitored in Neurosurgical HDU for 2 days before transfer to the ward and remained well throughout.

Although arterial spasm is a well-recognized complication of subarachnoid haemorrhage, with a peak incidence at 7–10 days post-bleed, we wondered whether the patient’s history of PRP had contributed to the severity of her vasospasm.

PRP is characterized by attacks of vasospasm precipitated by cold or emotional stress without clinical or serological evidence of a secondary cause. Attacks most frequently involve both hands and do not result in tissue necrosis. PRP results from an imbalance between vasoconstriction and vasodilatation and evidence suggests that abnormalities in the smooth muscle and endothelium of the blood vessels, central sympathetic control of vascular tone, and circulating mediators may all be involved in its pathogenesis.1 Controversially, it has also been suggested that PRP may be one part of a diffuse vasospastic disorder also affecting cerebral, coronary, and mesenteric vessels2 following observations that patients with PRP have a higher prevalence of chest pain, migraine,3 and an almost three-fold increase in the incidence of strokes.4 Further evidence suggests that peripheral manifestations of PRP may be accompanied by cerebral vasospasticity. A reduction in cerebral blood flow after cooling of the hands has been demonstrated using single positron emission computed tomography in seven patients with systemic lupus erythematosus (SLE) and secondary Raynaud’s phenomenon.5 SLE patients without Raynaud’s phenomenon did not show such a response to the same peripheral cold stimulus. More recently, an incident of cerebral vasospasm has been reported in a patient with a 40 yr history of PRP, occurring after separation from cardiopulmonary bypass.6 Spasm was evidenced by a precipitous decrease in cerebral oxygen saturation (measured with a near-infrared cerebral oximeter) in the presence of adequate $SpO_2$, MAP, and cardiac index and a constant $SvO_2$. Cerebral oxygen saturation in this case improved with a
bolus of glycerol trinitrate (GTN) and the patient was neurologically normal on waking.

Although the spasm in our case responded to nimodipine, simple strategies such as patient warming and avoiding the excessive use of vasoconstricting agents may be particularly important in Raynaud’s sufferers.

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Fig 1 Cerebral angiogram showing right internal carotid artery spasm.

1 Herrick AL. Pathogenesis of Raynaud’s phenomenon. Rheumatology 2005; 44: 587–96

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