CONTROLLED HYPOTENSION FOR CEREBRAL ANEURYSM SURGERY IN THE PRESENCE OF SEVERE AORTIC COARCTATION

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SUMMARY

A patient is described with a bicuspid aortic valve and an undiagnosed aortic coarctation, presenting with a subarachnoid haemorrhage. A cerebral aneurysm was clipped under controlled hypotension. In view of the risk of inducing severe hypotension in the distal aorta in this patient, femoral arterial pressure was monitored. A marked reduction in the radial–femoral arterial pressure gradient during controlled hypotension was noted.

KEY WORDS


The association between coarctation of the aorta and cerebral aneurysm is rare, but well documented [1–4]. In such patients, the advantages of using controlled hypotension in clipping the aneurysm must be weighed against the risk of producing hypotension distal to coarctation, leading to spinal cord ischaemia.

A patient with this anomaly and a bicuspid aortic valve is described, in whom simultaneous radial and femoral arterial pressure monitoring was used to manage distal aortic perfusion pressure during clipping of a ruptured cerebral aneurysm.

CASE REPORT

A 33-yr-old female with a past history of hypertension, treated with metoprolol, and a congenital bicuspid aortic valve, was admitted to a provincial hospital with sudden onset of confusion and impaired consciousness, preceded by headaches for 1 week. Clinical examination revealed a drowsy but orientated patient, with neck stiffness and a dense left hemiplegia. Delayed femoral pulses were noted, as was rib notching on the chest radiograph, raising the suspicion of an aortic coarctation.

A lumbar puncture yielded uniformly blood-stained CSF and a CT scan confirmed a frontal midline subarachnoid haemorrhage. The patient was transferred to our institution for further investigation and management.

A cerebral angiogram attempted via the right femoral artery failed because of a complete aortic coarctation with large collaterals, just distal to the left subclavian artery. Angiography by the transaxillary approach demonstrated a 5-mm anterior communicating artery aneurysm, and a smaller aneurysm at the bifurcation of the right middle cerebral artery. An echocardiogram suggested peak systolic gradients across the bicuspid aortic valve and the coarctation of 44 mm Hg and 56 mm Hg, respectively. Mild left ventricular hypertrophy was noted.

The patient was scheduled to undergo clipping of the anterior communicating artery aneurysm via a left frontal craniotomy in the supine position. All laboratory values were within normal limits. Premedication consisted of dexamethasone 4 mg and atropine 600 µg i.m. After induction of anaesthesia with thiopentone 250 mg, fentanyl 300 µg and atracurium 30 mg, anaesthesia was maintained with 60% nitrous oxide and isoflurane in oxygen. Neuromuscular block was maintained with an infusion of atracurium. Mannitol 20 g and
The patient was discharged 18 days after operation to a rehabilitation hospital, with plans to clip the second aneurysm at a later date. Her AP remained well controlled with metoprolol 100 mg twice daily.

**DISCUSSION**

The association between coarctation of the aorta and cerebral aneurysm was described first by Eppinger in 1871 [1]. In a literature review, Fukuda, Sako and Yonemasu found the incidence of coarctation among patients with cerebral aneurysms ranged from 0.19 to 1.9% [2]. The association appears more prevalent in young patients, however. Patel and Richardson [3] found that seven of 58 patients (12%) younger than 19 yr with subarachnoid haemorrhage had a coarctation, while Matson [4] found an incidence of three in 13 patients (23%) in a similar age group. A bicuspid aortic valve, as in the patient presented, may be expected to occur in 25–50% of patients with coarctation of the aorta [5]. Unfortunately, the coexistent coarctation may be overlooked. LeBlanc and colleagues [6] cited six cases of this combined anomaly, only one of which was diagnosed correctly at the time of admission. This presentation highlights the need to exercise a high index of suspicion of aortic coarctation in a young hypertensive patient presenting with a cerebral insult.

Our decision to use femoral arterial pressure monitoring as an index of spinal cord perfusion pressure was based upon the expectation of a significant decrease in distal aortic pressure during controlled hypotension. This technique has been practised in aortic coarctation repair as a means of objectively monitoring and managing distal aortic pressures during cross clamping, for prophylaxis of renal medullary ischaemia [7]. A mean distal aortic pressure in excess of 50 mm Hg has been suggested as compatible with adequate spinal cord perfusion [8]. Where circulation to the spinal cord may be compromised, Berendes and colleagues [9] have advocated that both femoral arterial and spinal fluid pressures be monitored, the difference representing the true perfusion pressure. During craniotomy, the CSF pressure is zero, making the latter measurement unnecessary in our patient.

Robine and co-workers [10] managed a 17-yr-old female with multiple cerebral aneurysms and aortic coarctation in a similar way. It was found...
that, with controlled hypotension, the gradient between mean radial and femoral AP decreased from 25 mm Hg to 5 mm Hg, but no explanation for this was offered.

In our patient, mean femoral AP approached mean radial AP as arterial pressure was decreased. This observation may be explained by an electrical analogy, visualizing the aorta as a fixed resistance (the coarctation) in parallel with a variable resistance (collaterals). During infusion of SNP, the collateral circulation dilates and the variable resistance approaches zero, reducing the pressure gradient across the aorta, regardless of the static nature of the coarctation. The inference from this is that, in a patient with poor collateral circulation, the distal aortic pressure may decrease markedly during controlled hypotension. The preoperative preparation must include an assessment of the adequacy of these collaterals, on clinical grounds (rib notching, chest wall bruits, poor femoral pulses) and angiographically.

An alternative explanation may lie in the effect of SNP on flow across the coarctation and collaterals. If the coarctation renders the lower body relatively ischaemic, with compensatory vasodilatation, during infusion of SNP the peripheral resistance of the lower body circulation may decrease to a lesser extent than that of the upper body circulation. In this case, for a given cardiac output, the flow across the coarctation/collateral complex may decrease with administration of SNP, with a reduction in the pressure gradient. The assumption in this hypothesis is that the collateral circulation is unresponsive to SNP and the resistance of the coarctation and collaterals remains fixed.

The place of evoked potential monitoring as an index of spinal cord integrity is unclear. Doyle [11] has indicated the potential for neurological damage in the absence of any electrophysiological change when using somatosensory evoked potentials. A likely explanation is that these potentials are monitors of posterior cord function, whereas the anterior cord is more at risk during episodes of hypotension.

In conclusion, this report emphasizes the importance of excluding aortic coarctation in young hypertensive patients with cerebral aneurysms. When controlled hypotension is indicated in patients in whom a coarctation of the aorta is present, a preoperative assessment of the collateral circulation assists in the identification of those at risk of spinal cord ischaemia. We suggest that femoral arterial pressure monitoring is a rational way of recognizing and managing distal aortic hypotension.

REFERENCES