ANAESTHETIC HAZARDS OF AORTOCAVAL FISTULA

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SUMMARY
A 66-yr-old man was anaesthetized for repair of a suspected ruptured aortic aneurysm. He became deeply cyanosed and suffered two episodes of asystole as the surgeons entered the abdomen. After the aorta was cross-clamped his condition improved markedly and no further problems ensued. Surgical exploration demonstrated intact aneurysms of the aorta and left iliac artery, the former containing a 3–4 cm long aortocaval fistula. The aneurysms and fistula were repaired and his further course was uncomplicated. Potential causes for the cyanosis and cardiac arrest in this patient are discussed.

KEY WORDS

CASE REPORT
A 66-yr-old man presented via the accident department with a history of 4 days abdominal and lower back pain. He had developed oliguria over 24 h. His past medical history included exertional angina and a myocardial infarct 10 years previously. He was taking a thiazide diuretic for mild hypertension. On examination he was sweaty, pale and in obvious discomfort from a painful pulsatile abdominal swelling. His heart rate was 110 beat min⁻¹ and arterial pressure 160/90 mm Hg; there were no signs of congestive heart failure. His lower limbs were mottled and swollen but retained palpable peripheral pulses, whilst the superficial veins of the lower limbs and abdomen were not obviously distended. There was no venous congestion of the head and neck. ECG demonstrated no signs of acute myocardial ischaemia. A diagnosis of ruptured aortic aneurysm was made.

Two large-gauge peripheral cannulae and a right subclavian central venous catheter were inserted and he was brought to the operating theatre without premedication. One litre of 0.9 % saline was infused before surgery. The central venous pressure was +8 cm H₂O before induction of anaesthesia (the catheter was seen to lie in the right internal jugular vein on a later chest x-ray). The haemoglobin concentration was 14 g dl⁻¹ before operation.

In the operating theatre, a urinary and left radial artery catheter were inserted using local anaesthesia. The abdomen was prepared and draped for surgery. Anaesthetic monitoring included continuous ECG, measurement of direct and indirect arterial pressure, end-tidal carbon dioxide, fractional inspired oxygen and anaesthetic vapour concentrations. After preoxygenation, a rapid sequence induction was performed with the administration of etomidate 10 mg and suxamethonium 75 mg. The trachea was intubated and anaesthesia maintained with fentanyl 300 ug, atracurium 25 mg and 60 % nitrous oxide and 1 % enflurane in oxygen. The heart rate and arterial pressure remained stable during the period of intubation.

Surgery was commenced immediately after induction and tracheal intubation. On entering the abdomen, all bleeding points were noted to be deeply cyanosed. The intestine was discoloured, with gross venous engorgement. Simultaneously, the patient's head and neck became increasingly plethoric and cyanosed. The question of oesophageal intubation or other causes of hypoxia were considered. Manual inflation of the lungs was performed with 100 % oxygen and the position of the tracheal tube rechecked by direct laryngoscopy and hearing bilateral air entry in the chest. Progressive sinus bradycardia, hypotension and asystole rapidly followed. I.v. calcium chloride 10 mmol and adrenaline 1 mg were given while external cardiac massage was performed by
the surgical team. A sinus bradycardia (40 beat min\(^{-1}\)) with hypotension (AP 50/30 mm Hg) returned within 2 min to be followed rapidly by another episode of bradycardia and asystole. A further 0.25-mg bolus of adrenaline was given. Whole blood 1000 ml was infused rapidly during this period.

The aorta was cross-clamped as quickly as possible, after which cardiac output rapidly returned. The iliac arteries were clamped also. Incision of the intact aneurysm sac revealed a 3-cm long aortocaval fistula with approximately 400 ml of loose clotted blood within the aneurysm. Brisk venous bleeding through the fistula was controlled by digital pressure initially and then by passing balloon catheters above and below the fistula via the femoral vein. After the aorta had been cross-clamped the patient developed hypertension (AP 250/110 mm Hg) and tachycardia (190 beat min\(^{-1}\)). These disturbances settled over 10–15 min, when the inspired concentration of enflurane was increased and practolol 5 mg and fentanyl 200 \(\mu\)g were administered. The aneurysms were repaired using a dacron aorto-bifemoral graft and the vein repaired by direct suture.

The remainder of the anaesthetic was uncomplicated. No further significant inotropic support was required, a good diuresis (1550 ml) followed the administration of 20% mannitol 100 ml and an infusion of dopamine 2 \(\mu\)g kg\(^{-1}\) min\(^{-1}\). A total of 10 units of blood and 4 units of fresh frozen plasma were given, mainly to cover heavy blood loss from a bleeding iliac vein, damaged by a balloon catheter. The patient was returned to the intensive care unit overnight, where recovery was uneventful. Minor disturbances of coagulation were observed over the next 2 days, but no further blood products were needed. There were no significant disturbances of cardiovascular, respiratory, hepatic or renal function in this period. The patient left hospital 9 days after surgery with some residual swelling of both legs.

**DISCUSSION**

Aortocaval fistula is an uncommon but well recognized complication of aortic aneurysm which may present both acutely and chronically [1–3]. The incidence has been estimated as 1–4% [3] of aortic aneurysms, but it was not mentioned in a literature search related to anaesthesia, or in recent reviews on anaesthesia for aortic aneurysm surgery [4, 5]. Less commonly, the aetiology may be traumatic, following accidents or lumbar disc surgery. Rare causes include syphilis, mycotic aneurysms, Marfans syndrome and Ehlos–Danlos syndrome [1]. The complications of arteriovenous fistulae relate to their size, anatomical location and the cardiac reserve of the patient. Common late presentations include high output cardiac failure, abdominal pain, limb discolouration and oedema. Traumatic fistulae may be entirely asymptomatic or present insidiously, as they tend to be smaller and occur in younger patients who have otherwise normal vasculature and a greater cardiac reserve.

Acute decompensation, as occurred in this patient, is reported less often and usually involves rupture of an associated aneurysm sac. Many such patients would not survive to reach hospital. In this patient, as in other reports [2], the fistula was not recognized before operation. The presence of discoloured swollen lower limbs should have raised suspicion that a fistula might be present in this patient, and other signs, including a bruit, high central venous pressure or cardiovascular decompensation, were not recognized. Increases in venous pressure may be limited to the lower limbs and may not cause an increase in central venous pressure [3]. A bruit may be intermittent or absent as thrombus blocks the fistula.

It is postulated that the acute deterioration in this patient followed surgical disturbance of blood clot which was partially blocking the fistula. The large fistula then allowed a significant part of the cardiac output to flow into the inferior vena cava. This low resistance pathway would minimize arterial pressure and flow to vital areas such as the coronary circulation, causing rapid haemodynamic collapse. The rapid development of cyanosis would follow from a combination of venous hypertension and low arterial flow. Such a sudden haemodynamic collapse would be accentuated in the presence of ischaemic heart disease, as present in this patient. Alternatively, the induction of anaesthesia may have precipitated haemodynamic decompensation if the unrecognized fistula was already patent.

The choice of anaesthetic agents used in this patient was designed to maintain cardiac stability. The sequence of anaesthetic induction with etomidate, suxamethonium, positive pressure ventilation, nitrous oxide and enflurane would be expected to cause a reduction in cardiac output and arterial pressure. Progressive bradycardia and asystole would not be expected in the absence of
severe blood volume depletion or cardiac dysfunction. Major blood loss was not a problem at induction of anaesthesia as the aneurysm sacs were intact and there was only about 400 ml of clotted blood in the sacs. When surgical control of the aneurysm was achieved, there was no further haemodynamic deterioration. Reperfusion and significant blood loss produced no further haemodynamic deterioration. There was no evidence of perioperative myocardial ischaemia on serial electrocardiograms and cardiac enzyme studies. Unfortunately, the central venous pressure was not measured during the cardiac arrest, as the intraoperative measurements were performed intermittently by U-tube manometer. When stability was returning, CVP was +10–12 cm H$_2$O.

An adverse reaction to anaesthetic drugs was considered unlikely in the absence of bronchospasm, cutaneous vasodilatation and rapid improvement after aortic cross-clamping.

Embolization of blood clot, atheromatous debris or air through the fistula has been reported to be a cause of cardiovascular collapse in such patients [3]. This cause was not considered at the time, but is unlikely as collapse occurred before the aneurysm was opened. Early surgical control of arterial and venous limbs of the fistula is essential to avoid this complication.

Surgical closure of arteriovenous fistulae has been associated with a bradycardia termed the Nicoladoni (Branham) sign. This reflex may be elicited also by digital pressure on a peripherally sited arteriovenous fistula. The proposed mechanism for this reflex involves arterial baroreceptors sensing increases in systemic arterial pressure as the fistula is closed; reflex bradycardia follows, mediated by the vagus [6]. In the circumstances of this case, no such reflex was seen.

The optimum anaesthetic management of the patient with a significant arteriovenous fistula has not been reported. In the presence of a significant fistula, arterial pressure is maintained by increased cardiac output and drugs or manoeuvres should be chosen to minimize such changes. Drugs causing vasodilatation may reduce flow through a fistula, but their effects would be unpredictable and likely to worsen systemic haemodynamics. Vasopressor drugs may be ineffectual because of increased shunting of blood through the fistula. Early surgical control of the fistula is the key to achieving haemodynamic stability, as demonstrated in this patient. As in the case of a ruptured aneurysm, anaesthesia should not be induced without the abdomen prepared, with surgeons scrubbed and ready to enter the abdomen. Anaesthetists should be vigilant for the presence of related or separate cardiovascular pathology in such patients, which may be missed in the haste to get to theatre. The optimum anaesthetic technique is probably a high-dose opioid and neuromuscular blocker technique, as this minimizes changes in cardiac output and systemic vascular resistance.

Control of a fistula can be achieved rapidly after laparotomy by conventional cross-clamping above and below the fistula. Alternatively, control can be achieved by passing balloon catheters above and below the fistula, if necessary under x-ray control. Venous control above and below the fistula is necessary to control bleeding into the open aneurysm sac and potential embolization. If problems are anticipated before surgery, balloon catheters may be passed via the femoral vessels under local anaesthesia, before induction of general anaesthesia.

REFERENCES