INTRAOPERATIVE DIAGNOSIS AND TREATMENT OF MASSIVE PULMONARY EMBOLISM COMPlicATING SURGERY ON THE ABDOMINAL AORTA

B. R. HECKER AND C. LYNCH

SUMMARY

Massive pulmonary embolism associated with total cardiovascular collapse occurred during the surgical repair of a ruptured abdominal aortic aneurysm with an aortocaval fistula. Pulmonary artery pressure monitoring permitted immediate diagnosis whereas central venous pressures did not reflect the obstruction to right ventricular outflow. Pulmonary embolectomy without cardiopulmonary bypass was performed successfully.

Massive pulmonary embolism occurring during surgery, and with immediate surgical correction, has not been reported previously. The following case report illustrates the haemodynamic and pulmonary consequences of such an event, and describes successful surgical intervention without cardiopulmonary bypass.

CASE REPORT

A 64-year-old white male was admitted to a local hospital complaining of increasing abdominal pain, anorexia, nausea and vomiting for 3 days. An abdominal x-ray suggested the presence of an aortic aneurysm, approximately 14 cm in diameter. The patient was transferred to the University of Virginia Medical Center. On arrival he was alert, tachypnoeic and afebrile. Systemic arterial pressure (AP) was 90/40 mm Hg, heart rate (HR) 120 beat min\(^{-1}\) with normal heart sounds and no murmurs. A pulsatile mass was palpable from the xiphoid to the umbilicus. Examination of the lower extremities revealed palpable femoral pulses; pedal pulses were not palpable but present on Doppler examination. No signs of venous disease were found.

Monitoring of systemic and pulmonary artery pressures was established. Initial measurements were: AP 115/20 mm Hg; HR 110 beat min\(^{-1}\); central venous pressure (CVP) 22 mm Hg; pulmonary artery pressure (PAP) 41/24 mm Hg; pulmonary capillary wedge pressure (PCWP) 21 mm Hg; cardiac output (CO) 11.0 litre min\(^{-1}\). Electrocardiogram (ECG) revealed sinus tachycardia and non-specific ST–T wave changes with no signs of acute right ventricular strain. Arterial blood-gas values, on 40% oxygen space tent, were pH 7.41 unit, \(P_{\text{a}O_2}\) 12.5 kPa, \(P_{\text{a}CO_2}\) 3.86 kPa. Haematocrit was 40%. The urine output was 13 ml in 4 h. A supine portable chest x-ray showed prominent vascular markings with no effusions, parenchymal densities or atelectasis. An angiogram confirmed the diagnosis of abdominal aortic aneurysm (14 × 15 × 13 cm) and demonstrated a distal aortocaval fistula. Echocardiogram was normal with no evidence of thrombi or ventricular dysfunction.

On arrival in the operating theatre, the patient was alert and orientated (AP 70/20 mm Hg, HR 110 beat min\(^{-1}\), CVP 33 mm Hg, PAP 50/30 mm Hg). Induction of anaesthesia and intubation of the trachea were accomplished with incremental doses of diazepam 10 mg, fentanyl 500 ng, and pancuronium 8 mg i.v., without any change in the haemodynamic indices. Pupils were pinpoint and symmetrical. The patient was maintained throughout with \(F_{\text{Io}2}\) 1.0. With a ventilation of 9 litre min\(^{-1}\), initial intraoperative blood-gases were pH 7.20 unit with a base deficit of 15 mmol, \(P_{\text{a}O_2}\) 37.8 kPa, and \(P_{\text{a}CO_2}\) 4.26 kPa. Sodium bicarbonate 200 mmol was administered.

A further decrease in arterial pressure occurred coincident with the skin incision. An infusion of dopamine 5 \(\mu\)g kg\(^{-1}\) min\(^{-1}\) was started and whole blood administered. The aorta was cross-clamped distal to the renal arteries and the arterial pressure increased to 92/60 mm Hg. Shortly thereafter the PAP increased abruptly to 90/40 mm Hg and the arterial pressure began to decrease again. Arterial blood-gas values were pH 7.36 unit, \(P_{\text{a}O_2}\) 12.8 kPa, \(P_{\text{a}CO_2}\) 5.59 kPa. A presumptive diagnosis of pulmo-
nary embolism was made while the surgeons proceeded to control severe haemorrhage in the abdomen. CVP decreased to 25 mm Hg. Despite the continued infusion of blood, an increase in the dopamine infusion rate to 15 \(\mu\)g kg\(^{-1}\) min\(^{-1}\), intermittent boluses of calcium chloride (total 2 g), adrenaline 500 \(\mu\)g, and noradrenaline, the arterial pressure continued to decrease over the next few minutes to 30 mm Hg with no discernible pulse pressure. Heart rate decreased from 135 to 60 beat min\(^{-1}\) with no response to atropine 1.0 mg and isoprenaline (total 400 \(\mu\)g). The EEG became isoelectric; pupils were fixed and dilated.

Sternotomy was performed and internal cardiac massage begun with less than a 5-mm Hg pulse pressure response. Cardiac massage was continued while vascular loops were placed around the superior and inferior vena cavae, an incision made in the main pulmonary artery and a 45-g saddle embolus measuring 7 x 4 x 2 cm removed. During the embolectomy, blood-gases were pH 7.23 unit, \(P_{\text{aO}_2}\) 7.05 kPa and \(P_{\text{aCO}_2}\) 5.99 kPa.

After evacuation of the embolus and closure of the pulmonary artery, the patient’s HR increased to 140 beat min\(^{-1}\) and arterial pressure increased to 90/60 mm Hg without mechanical or pharmacological support. PAP decreased to 50/30 mm Hg. Ten minutes after suture of the pulmonary artery, arterial blood-gases were pH 7.25 unit, \(P_{\text{aO}_2}\) 24.7 kPa and \(P_{\text{aCO}_2}\) 5.99 kPa. The remainder of the aortic repair proceeded uneventfully. Shortly before leaving the operating theatre cardiac output was 5.5 litre min\(^{-1}\); PAP was 50/25 mm Hg. Urinary output increased to 240 ml h\(^{-1}\) after the pulmonary artery closure. EEG showed some restoration of electrical activity and the pupils were midposition and slightly reactive.

Six hours after operation the patient was responsive and obeyed commands. The trachea was extubated on the 4th day following surgery. The ECG was unchanged from the preoperative study. The patient was discharged on the 14th day with no apparent sequelae.

**DISCUSSION**

The pathophysiology initially observed in this patient appeared to be high output cardiac failure secondary to aortocaval shunt. Although pulmonary embolization could have occurred before operation, this is unlikely since the patient’s CO was 11 litre min\(^{-1}\). The nature of the operative procedure, involving considerable manipulation of an atheromatous aorta which readily communicated with the lower pressure venous system, made embolization a highly probable event during operation. Diagnosis was not difficult based on the clinical and laboratory findings present: sudden cardiovascular collapse, increased pulmonary artery pressures (Glassford et al., 1981) and blood-gas values consistent with shunt rather than deadspace, that is, decreased \(P_{\text{aO}_2}\) (Moser, 1977). Although diagnosis of pulmonary embolus is possible during operation based on clinical findings or blood-gas values (Moser, 1977; Divekar, Kamdar and Pansave, 1981; Berry, 1982) a pulmonary artery catheter reliably provides instantaneous evidence of pulmonary artery obstruction (Glassford et al., 1981; Mangano, 1980). CVP monitoring may not provide evidence of pulmonary artery occlusion in patients who are haemorrhaging or otherwise incurring intravascular depletion. The CVP may actually decrease following embolism as occurred in this patient.

Pulmonary embolectomy, regardless of technique, is shadowed by controversy. From Trendelenburg’s first attempt at pulmonary embolectomy in 1908 to 1961 only 22 patients survived (Mattox et al., 1982). More patients died than were salvaged; of those who survived, many were permanently crippled neurologically. Since most of the patients who embolize and survive long enough to receive medical treatment have a low mortality rate, surgical embolectomy produces small benefit and imposes considerable risk (Moser, 1977). However, in patients with massive pulmonary embolus (defined as obstruction of more than 50% of the pulmonary arterial vasculature, associated with hypoxaemia and inadequate systemic perfusion while on vasopressor therapy) (Mattox et al., 1982), right ventricular decompensation is rapid and death occurs in 1–2 h (Glassford et al., 1981). Indeed, at 20 min, 50% of these patients are dead (Gorham, 1961). Mortality is high enough in this group of patients for them to be considered as candidates for immediate embolectomy.

Normothermic vena caval inflow occlusion without cardiopulmonary bypass (CPB) was used in this patient because immediate correction was required. Among patients who have not arrested before the intervention, survival with this technique is comparable to that obtained when CPB is used. Patients who arrest before corrective surgery, however, rarely survive when embolectomy is attempted without CPB (Clarke, 1981).

Combining inflow occlusion with CPB, Miller, Hall and Paneth (1977) reported 100% survival in 12
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patients who had not suffered circulatory arrest and a 46% survival rate in those who had sustained one or more cardiac arrests before surgery. Using the same technique, Mattox and colleagues (1982) reported a 50% survival rate among 40 patients who had cardiac arrest or refractory hypotension before embolectomy.

Immediate operative intervention is not necessary in most patients who develop pulmonary embolism. However, among patients with massive intraoperative pulmonary embolization, the risk of death is sufficiently great to warrant immediate surgical correction. Pulmonary artery pressure monitoring facilitates the diagnosis and treatment of this potentially lethal complication. When CPB is not available, inflow occlusion alone has been a life-saving procedure even for patients with complete cardiovascular collapse, as illustrated by the case presented.

REFERENCES


