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Case Report

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Anaesthesia Management in Idiopathic Pulmonary Artery Aneurysm Surgery

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Abstract

Pulmonary artery (PA) aneurysm is a very rare disease. Surgical treatment is important in symptomatic patients. Due to its anatomical condition, the rupture of such aneurysms may lead to sudden right ventricular failure or sudden death. The aim of this report is to present the use of balanced general anaesthesia management in a patient who underwent PA aneurysm repair surgery. Surgical repair was planned in a 55-year-old man with chest and back pain as well as haemoptysis. With the help of balanced anaesthesia, stress on the aneurysm wall was prevented by avoiding sudden blood pressure increases. With appropriate ventilation methods, we attempted to avoid an increase in pulmonary vascular resistance, and therefore, the pressure on the aneurysm, as well as an increase in the postoperative right ventricle. With the help of appropriate anaesthesia and ventilation techniques, uncomplicated and successful anaesthetic management was effected in the repair of a PA aneurysm.

Keywords: Anaesthesia management, pulmonary artery aneurysm, pulmonary vascular resistance

Introduction

Aneurysm is defined as the focal dilatation of a blood vessel involving all the three layers of the vessel wall (1). Pulmonary artery (PA) aneurysm is a condition in which the diameter of the PA is higher than the upper limit in adults (29 mm) (1). However, the actual pathological dilatation is considered to be 40 mm or higher (2). The incidence of PA aneurysm in the general population is very low. Etiological factors responsible for PA aneurysm are congenital heart diseases, infections, connective tissue diseases (such as Marfan syndrome), vasculitis (such as Behcet's disease), chronic pulmonary embolism, and idiopathic reasons (3, 4). There are four criteria for the formation of idiopathic PA aneurysm: isolated dilatation of the pulmonary truncus (distal branches of the arterial tree may be affected); absence of an intracardiac or extracardiac shunt; absence of chronic cardiac or pulmonary diseases (1); and absence of pulmonary vascular tree diseases (such as syphilis) (5). PA aneurysms are usually asymptomatic. Clinical symptoms include cough, dyspnea, chest pain and haemoptysis. Pulmonary hypertension and right ventricular (RV) failure are important factors for clinical management.

Because of the sudden risk of death, high-risk surgery and rarity of a case, we aimed to present the anaesthesia management for this particular case involving graft surgery in an idiopathic PA aneurysm.

Case Presentation

In the current report, the patient is a 55-year-old man (171 cm and 87 kg) with a known diagnosis of hypertension. His idiopathic PA aneurysm was first identified six years before the current admission. He was admitted for a week with the symptoms of recurrent haemoptysis. Further, he had chest and back pain for a long time. The rheumatologic and infectious markers were normal. PA dilatation was visible on the direct chest X-ray (Figure 1). The CT scan revealed that the pulmonary truncus diameter was 53 mm and aneurysmatic. Transesophageal echocardiography (TEE)

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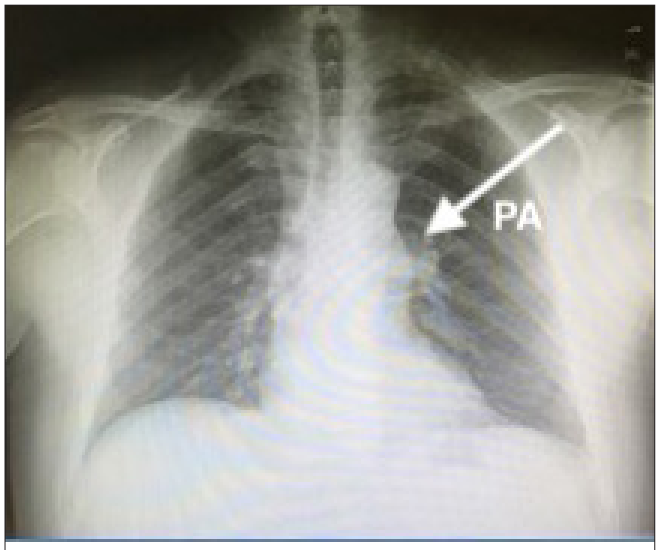

Figure 1. Chest X-ray showing the PA aneurysm

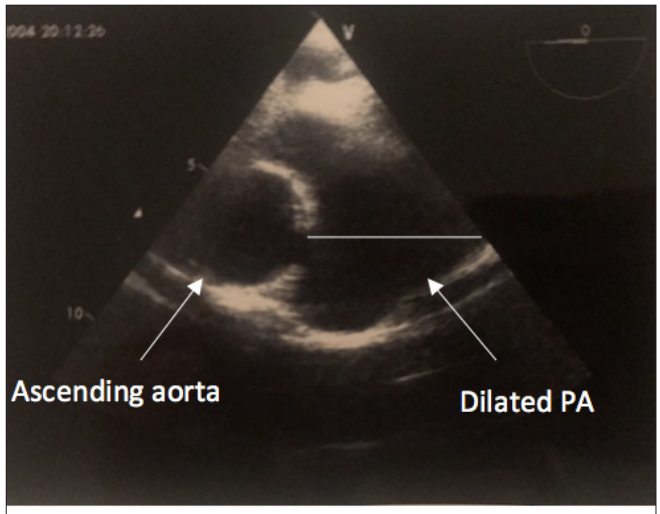

Figure 3. Midesophageal ascending aorta short-axis TEE image showing the dilated PA besides the ascending aorta

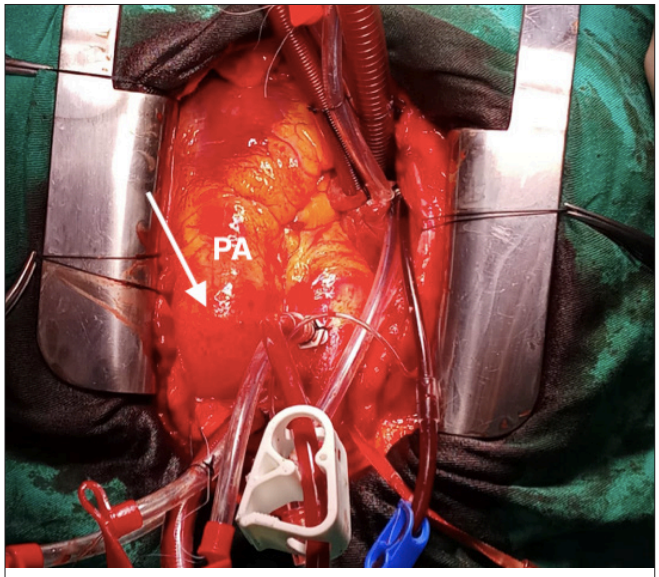

Figure 2. Intraoperative photograph showing the PA aneurysm after sternotomy

revealed that the PA diameter was 58 mm before bifurcation and the mean pulmonary arterial pressure was 20 mmHg (N: 10-18 mmHg). The RV dimensions were evaluated as normal, and there was no insufficiency. The patient was taken to the operating room for repair of the PA aneurysm. Routine electrocardiography, pulse oximetry and non-invasive blood pressure monitoring were performed. Further, a 20-gauge arterial catheter was inserted in the left radial artery using local anaesthesia under conscious sedation (2 mg midazolam). The patient was subjected to bispectral index (BIS™, Covidien, MN, USA) and near-infrared spectroscopy (NIRS, INVOS 5100, Somanetics Corporation, Troy, MI, USA) monitoring to evaluate the depth of anaesthesia and cerebral oxygenation. Pulse-induced contour cardiac output (PiCCO, Pulsion Medical Systems SE, Germany) monitoring was performed to evaluate the cardiac output, systemic vascular resistance and oxygen demand. Anaesthesia was induced using 0.1 mg kg^{-1} midazolam, 10 mcg kg^{-1} fentanyl and 0.6 mg kg^{-1} rocuronium. After the induction of anaesthesia, the patient was intubated without any complications (such as hypertension or tachycardia). The mechanical ventilation settings were adjusted such that the tidal volume was 6 mL kg^{-1}, PEEP was 3 cm H_2O, and FiO_2 was 50%; further, the patient's $PaCO_2$ value was between 30 and 35. A central venous catheter was inserted and central venous pressures were continuously monitored. Because of the risk of rupture of the PA aneurysm, a PA catheter was not inserted. A TEE probe was inserted to provide continuous cardiac imaging. Anaesthesia was maintained using inhaled sevoflurane (as MAC 1-1.3); dexmedetomidine infusion (0.2 mcg kg^{-1} h^{-1}); and additional doses of fentanyl, midazolam and rocuronium. Tranexamic acid infusion was started with an induction dose of 10 mg kg^{-1}, followed by 1 mg kg^{-1} h^{-1} after the induction of anaesthesia. The PA aneurysm was immediately visible after sternotomy and pericardiotomy (Figure 2). The TEE revealed an enlarged PA (Figure 3), and there was no pulmonic valve insufficiency or stenosis. After adequate ACT levels were achieved, cardiopulmonary bypass (CPB) was initiated, and the patient was cooled down to 28°C. Following aortic cross-clamping, myocardial protection was provided with a St. Thomas cardioplegic solution. It was observed that the pulmonary valve had four leaflets instead of three, but surgical intervention was not initiated (Figure 4). The PA was repaired by using a synthetic tube graft. The patient's variables during the operation are shown Table 1.

During the CPB period, the perfusion pressure was maintained between 50 and 80 mmHg. No decrease in NIRS values was observed by more than 20% of the basal value. Aortic

Table 1. The Patient's variables during the operation

	After induction	Before CPB	CPB 10. min	CPB 60. min	CPB 100.min	After CPB	Sternum closing
SAP/DAP (mmHg)	98/62	68/44				84/45	93/52
Perfusion pressure (mmHg)			50	65	77		
HR (beat min^{-1})	63	56	-	-	65	85	73
NIRS L/R	54/58	51/55	50/53	49/52	52/59	49/52	52/59
BIS value	36	45	47	50	49	52	49
CVP (mmHg)	7	10	3	2	5	1	11
CO (lt min^{-1})	5.3	4.1	-	-	-	3.3	5.6
Pump flow (lt min^{-1})	-	-	4.2	4.5	4.7	-	-
SVR (dyne sec cm^{-5})	1230	830	-	-	-	560	300
DO_2 (mL min^{-1} m^{-2})	1210	919	505	685	697	503	635
Urine (mL)	0	150	200	300	600	1100	1200
Hb (gr dL^{-1})	16.7	12.8	11.7	11.1	10.8	9.1	8.2
SvO_2 (%)	75	72	75	82	81	82	74
Body temperature (°C)	36.8	35.5	32	28	33	37.2	36.8

SAP: systolic arterial pressure; DAP: diastolic arterial pressure; NIRS: near-infrared spectroscopy; BIS: bispectral index; CVP: central venous pressure; CO: cardiac output; SVR: systemic vascular resistance; DO_2: oxygen delivery; Hb: haemoglobin; SvO_2: central venous oxygen saturation; CPB: cardiopulmonary bypass

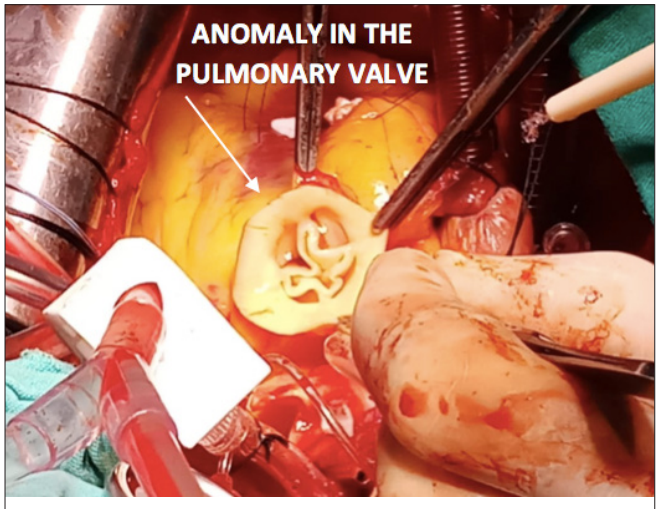

Figure 4. Anomaly in the pulmonary valve

cross-clamping and CPB durations were 128 and 177 min, respectively. At the end of the CPB, 1 g fibrinogen concentration was administered. Any blood or blood product was not required. After hemodynamic stability, the operation was ended. The patient was transferred to the intensive care unit with dexmedetomidine infusion, made an uneventful recovery from surgery, and was extubated at the postoperative 12th hour. Written consent for publication has been obtained from the patient.

Discussion

Pulmonary artery aneurysms are very rare and high-risk surgeries. They are important because they involve the risks of RV failure, aneurysm rupture and sudden death. Apart from these issues, there is no difference in terms of anaesthesia management as compared to the other types of cardiac surgeries. Tracheal intubation or surgical stimuli may be responsible for the development of sympathetic excitation and rise in blood pressure, depending on which aneurysm rupture may occur. It is important to avoid an increase in pulmonary vascular resistance (PVR) besides blood pressure. It may cause an increase in the RV afterload and tension on the aneurysm wall (3). On the basis of this information, we tried to avoid the factors that may cause an increase in PVR, such as hypoxia, respiratory/metabolic acidosis, and adrenergic/nociceptive stimuli (3). It is known that a high tidal volume and PEEP settings may cause pulmonary hyperdistension and an increase in PVR. Therefore, in our case, the mechanical ventilation settings were adjusted such that the tidal volume was maintained at 6 mL kg^{-1} and PEEP at 3 cm H_2O.

Anaesthesia induction and maintenance were achieved with routine cardiac anaesthesia agents. In the literature, there is no reported agent among anaesthesiologists that can provide a proven method for tackling PA aneurysms. However, But et al. (6) suggested that in patients with pulmonary hyperten-

sion undergoing mitral valve surgery, an alpha-2 adrenergic receptor agonist called dexmedetomidine was known to exert sympatholytic, sedative, amnestic and analgesic properties, as well as the average systemic and pulmonary arterial pressures were shown to reduce. Therefore, dexmedetomidine infusion after anaesthesia induction was started in our patient in the intensive care unit. Due to the difficult and complicated nature of the surgery, care was taken toward myocardial protection during the long-lasting aortic cross-clamping. After weaning from CPB, the RV was effectively evaluated, and the use of agents that reduce PVR (e.g., milrinone and inhaled nitric oxide) was considered. These agents dilate the pulmonary vasculature and reduce pressure on the surgical sutures (3). RV dysfunction is a concern in such patients. This can be difficult to detect in the intraoperative period as RV pressures cannot be monitored because of the risk of rupture. We believe that this management can be effected through TEE monitoring. Further, the patient had no primary cardiac pathology; therefore, we considered providing the indirect monitoring of RV dynamics by monitoring the left ventricular pressures and fluid status with PiCCO.

Conclusion

In PA aneurysm surgeries that may be associated with significant intraoperative problems (e.g., increased PVR or RV failure), close monitoring and ensuring sympathetic suppression without causing myocardial depression are important points. TEE monitoring is a very useful and less invasive monitoring method for a patient with such a critical aneurysm. Moreover, the monitoring of the left ventricle with PiCCO increases the viability of the data obtained regarding the patient. The use of the perioperative dexmedetomidine in the management of anaesthesia to achieve sympathetic suppression without myocardial depression should be promoted.

Informed Consent: Written informed consent was obtained from patient who participated in this case.

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