Case Report

Intraoperative Aortic Adventitial Dissection Causing Failure to Separate from Cardiopulmonary Bypass

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ABSTRACT

Despite advances in thoracic aortic surgery, the incidence of perioperative major morbidity and mortality remains significant. Hemorrhage remains an important contributor to early complications and a predictor of adverse outcomes. We describe a case of failure to separate from cardiopulmonary bypass during aortic valve, ascending and aortic arch replacement for progressive enlargement of aortic aneurysms. This was likely due to a large posterior mediastinal hematoma which was only identified post-mortem and resulted from an unrecognized sentinel tear in the distal aortic graft anastomosis. The hematoma produced major compression of the left bronchus, pulmonary artery, and pulmonary veins. Failure to detect the aortic anastomotic tear, and the subsequent hematoma expansion, led to failed separation from CPB and made all resuscitative efforts including VA-ECMO support ineffective. Therefore, we believe that locating the site of adventitial dissection and expeditiously repairing it is a critical step in managing such complication and should be done while supportive measures are provided.

Key Words: Aortic surgery, CPB weaning, mediastinal hematoma.

Received: 11 March 2024**, Accepted:** 19 May 2024

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ISSN: 2090-925X, The Egyptian Journal of Cardiothoracic Anesthesia 2024, Vol.18, No 2

INTRODUCTION

Despite advances in thoracic aortic surgery, the incidence of perioperative major morbidity and mortality remains significant. Hemorrhage remains an important contributor to early complications and a predictor of adverse outcomes[1]. A host of technical and procedural issues can cause difficulty separating from cardiopulmonary bypass (CPB) during such procedures, however, hemorrhage and hematoma expansion is rarely directly involved. We describe a case of unanticipated failure to separate from CPB, in a patient undergoing aortic valve, ascending aorta, aortic arch, and proximal descending aortic replacement for progressive enlargement of the aortic root and arch, where unrecognized posterior mediastinal hematoma expansion compressed the broncho-vascular bundles bilaterally, and seriously complicated CPB separation, and contributed to overall poor outcome.

CASE REPORT

A 70-year-old female with past medical history of hypertension and preserved ejection fraction presented with progressive aneurysmal dilatation of the aortic root and arch and associated moderate aortic valve regurgitation. Surgical history included supracoronary ascending aortic replacement to repair acute type A aortic dissection 18 years earlier (Figure 1).

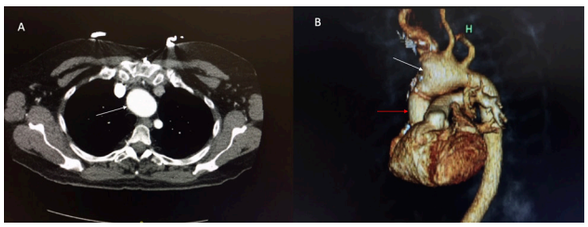

Fig.1: Panel A showing a preoperative multi-detector axial computerized tomographic (CT) image with a white arrow pointing to the aortic arch aneurysm. Panel B showing a preoperative 3D reconstruction image with a white arrow pointing to the aortic arch aneurysm, and a red arrow pointing to the pre-existing ascending aortic graft.

Planned surgery included aortic valve, ascending aorta, aortic arch, and proximal descending aortic replacement. Intraoperative Transesophageal echocardiography (TEE) confirmed the aortic pathology and showed preserved left and right ventricular function. Surgery was uneventful,

DOI: 10.21608/ejca.2024.276056.1002

but the descending aortic wall was noted to be friable, and most of the sutures resulted in small intimal tearing, the surgeon accordingly employed several pledgeted sutures as needed.

Subsequently, during separation from CPB, hemodynamic parameters were acceptable but elevated airway pressure was noted along with difficulty in achieving adequate tidal volumes, and under-inflation of the left lung observed in the surgical field. Bronchoscopy demonstrated a newly developed obstruction of the distal trachea and left mainstem bronchus, so a left sided double lumen endotracheal tube was placed to overcome the obstruction. Bilateral lung ventilation was instituted by ventilating both tracheal and bronchial lumens of the double lumen tube. Separation from CPB was achieved, and heparin anticoagulation was reversed. Immediately following termination of CPB, TEE demonstrated a normally functioning bio-prosthetic aortic valve, and mild left and right ventricular dysfunction. Shortly afterwards, progressive increase in pulmonary arterial and decrease in systemic arterial pressures was associated with development of right ventricular dilatation and failure, despite escalating doses of several inotropic infusions. Adequate reheparinization was achieved and CPB was emergently resumed.

Multiple subsequent re-attempts at CPB separation failed despite high dose intravenous inotropic support, inhaled nitric oxide, performing a vein bypass graft to the right coronary artery (RCA) for possible RCA dissection, implantation of femoral Intra-aortic balloon pump, and Veno-arterial Extracorporeal Membrane Oxygenation (VA-ECMO) support via central cannulation. We were unable to maintain adequate ECMO flows, and despite maximal resuscitation efforts, the patient expired in the operating room.

Postmortem examination revealed a 3 mm aortic tear at one of the prolene sutures in the distal aortic anastomosis of descending aorta that was not appreciated intraoperatively (**Figure 2**). The tear was associated with an adventitial dissection and a large (21 X 7 cm) posterior mediastinal hematoma that extended from the level of the cricoid cartilage to 5 cm below the diaphragm (**Figure 3**). The mediastinal hematoma, combined with the *in situ* ascending aortic graft from her previous surgery, compressed structures including distal trachea, left main bronchus, pulmonary artery, and pulmonary veins. Widespread tracking of the hematoma bilaterally along broncho-vascular bundles from the hilum to the visceral pleura was also evident and may also have affected pulmonary vascular flow and airway patency.

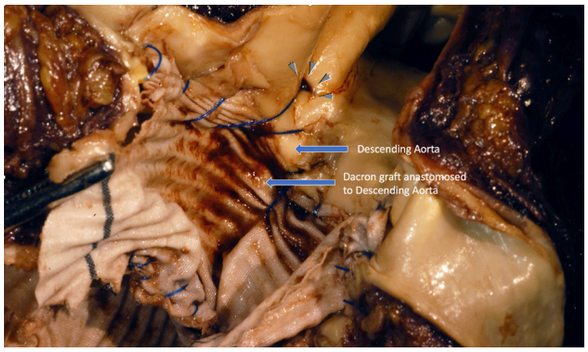

Fig.2: Postmortem picture of the distal aortic anastomosis of the descending aorta, with multiple arrows pointing to the small intimal tear, which was the origin of the adventitial dissection, that resulted in posterior mediastinal hematoma.

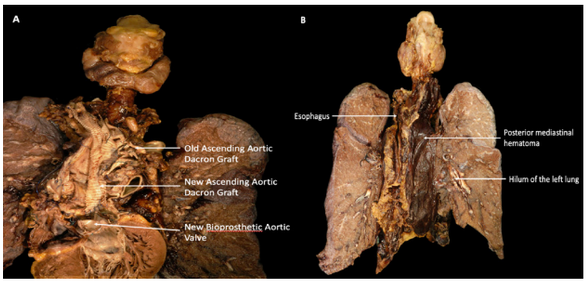

Fig.3: Panel A showing an anterior postmortem cross section of the patient's thoracic cavity showing the heart with the new bioprosthetic aortic valve, and both the pre-existing and new ascending aortic dacron grafts. Panel B showing a posterior postmortem cross section of the patient's thoracic cavity showing the posterior mediastinal hematoma.

DISCUSSION

Given what we learned from postmortem examination, identifying, and repairing the small aortic tear in the distal anastomosis might have limited hematoma expansion, and changed outcome of this case. During autopsy, it was evident that the hematoma was pushing the in situ fibrosed ascending aortic graft that was opened and left in place, against the left main bronchus, pulmonary artery, and pulmonary veins compressing them in the absence of clear bleeding in the surgical field. Despite extensive TEE interrogation, the aortic tear and expanding hematoma were not visualized. The left bronchial obstruction was our only clue to the expanding mediastinal hematoma.

Compression of the trachea, left sided bronchi, and pulmonary vessels is a well-known, although uncommon complication of thoracic aortic aneurysms. It usually occurs secondary to long standing extrinsic compression by slowly growing aortic aneurysms, where airway compression can present gradually either preoperatively[2], or postoperatively[3]. Acute postoperative airway compression has also been reported due to tracheomalacia caused by a longstanding aortic aneurysmal compression[4].

In our case, left main bronchial obstruction occurred acutely intraoperatively before separation from CPB, even though there was no evidence of preoperative airway compression, which is highly unusual in adults given their thicker tracheal and bronchial walls.

There has been previous reports demonstrating aortic dissection rupturing in the aortopulmonary window, with subsequent extension into the broncho-vascular sheaths and reaching the pulmonary interstitium causing hemorrhage[5,6]. However, this is mostly reserved to dissections in the ascending aorta, where it shares a common adventitia with the pulmonary trunk[7]. Although our case shares a similar pathophysiology, the dissection occurred in the proximal descending aorta anastomotic site, and it was iatrogenic in nature.

Of note, extreme caution was used when the endotracheal double lumen tube was placed to bypass the obstruction, excessive force should not be used to avoid tracheobronchial disruption[8]. Although advancing the endobronchial tube beyond a narrow segment of the airway is extremely hazardous, we felt that if no resistance was met, it was safe to advance the tube under bronchoscopic guidance. Absence of preoperative obstruction makes inherent tracheal and bronchial wall weakness less likely. Unfortunately, bypassing the obstruction provided only a temporary relief, because the adventitial dissection was not detected intraoperatively, or surgically repaired.

Failure to detect the aortic anastomotic tear, and the subsequent hematoma expansion, led to failed separation from CPB and made all resuscitative efforts including VA-ECMO support ineffective. Therefore, we believe that locating the site of adventitial dissection and expeditiously repairing it is a critical step in managing such complication and should be done while supportive measures are provided.

CONFLICTS OF INTEREST

There are no conflicts of interest.

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