

Management of Giant Pseudoaneurysm of Ascending Aorta Presenting as Mass in Neck: Rare Complication of Cardioplegia Needle

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Abstract

Ascending aortic pseudoaneurysms are rare and occur infrequently from anastomotic dehiscence of suture lines and cannulation sites from previous aortic surgery. It is managed by open surgical approach but carries a high risk of pseudoaneurysm rupture at the time of sternotomy. We describe a successfully managed case of ascending aortic pseudoaneurysm presenting as a pulsatile mass in the neck. Its orifice was located on the lateral wall of the ascending aorta, which was caused by the cardioplegia cannula inserted during a previous cardiac surgery. We also summarize the anesthetic challenges and complications of such a case.

Key words: Ascending aorta pseudoaneurysm, cardioplegia needle, deep hypothermia

INTRODUCTION

Ascending aortic pseudoaneurysms are rare and occur infrequently from anastomotic dehiscence of suture lines and cannulation sites from previous aortic surgery.^[1] They can also occur consequent to infection, genetic disorders, or trauma.^[2,3] The incidence, risk factors, and natural history of aortic pseudoaneurysm are unknown because few cases have been reported. False aortic aneurysms result from disruption of the intima and media of a vessel. They are contained by the adventitia and the surrounding structures of the mediastinum.^[4] We report a case of a pseudoaneurysm of ascending aorta presenting as a pulsatile mass in the neck anteriorly and the challenges we faced while managing the case.

CASE REPORT

A 36-year-old, 45 kg female patient presented to the outpatient department of cardiothoracic surgery with a pulsatile swelling in her neck anteriorly [Figure 1]. She was a known case of mitral stenosis with a history of mitral valve replacement (MVR) first done 10 years back. She had undergone a revision MVR with tricuspid ring annuloplasty through a right thoracotomy approach 7 months back as she had become symptomatic again. Surgery and recovery was uneventful and she was discharged from the hospital. She had a history of hypothyroidism and

was on tab. thyroxine 75 mg once daily. She was also taking tab. warfarin, tab. diltiazem since 7 months. She had no history of orthopnoea or dysphagia.

Fifteen days before admission, she noticed a swelling in her neck, which was accompanied by headache. She did not give a history of fever. On examination, her vitals were: blood pressure (BP) was 110/70 mmHg and heart rate was irregularly irregular. Her blood investigations showed a Hb of 7.5 g%, white blood cell count of 7500/cm³, and liver function and renal function tests were within normal limits (wnl). International normalized ratio (INR) was 2.5, and thyroid function test revealed euthyroidism.

Her computed tomography (CT) scan with contrast showed unruptured pseudoaneurysm arising from the right lateral wall of ascending aorta 6 × 10.2 × 13.2 cm (AP × TR × CC), with the neck measuring 2.8 mm. There was compression and displacement of superior vena cava and part of the inferior vena

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cava to the right [Figure 2]. Cardioplegia needle entry point placed on the lateral wall of ascending aorta during revision surgery was suspected to be the likely cause. Two-dimensional echo showed a dilated left atrium and mitral valve prosthesis *in situ*, with left ventricular ejection fraction (LVEF) of 60%.

She was started on low molecular weight heparin (after stopping warfarin) and tab. metoprolol.

Decision was taken to take her for surgery on an urgent basis after transfusing her with 2 packed cells.

In the operation theatre, noninvasive blood pressure cuff, pulse oximeter, and electrocardiogram (ECG) were attached. Her blood pressure was 100/70 mmHg and heart rate was 120/min in atrial fibrillation. A left radial artery cannulation was performed under local anesthesia and sedation, which revealed a ventricular rate of 68/min.

Patient was premedicated with inj. fentanyl 150 mcg and inj. midazolam 0.5 mg intravenous (IV). Because CT scan showed no compression of airways we decided to induce the patient first. She was induced with inj. etomidate 0.2 mg/kg IV and intubation was facilitated with inj. rocuronium 0.5 mg/kg mg IV. A 7.5 no. cuffed tube was passed under direct vision laryngoscopy. The surgeons then cannulated the femoral artery and vein and femorofemoral cardiopulmonary bypass (CPB), and a flow rate of 2 L/min was started after full heparinization. The perfusionist was then asked to cool the patient to 21°C to induce hypothermic arrest of heart. Incision was also made to start ministernotomy. When the temperature reached 24°C, the patient's heart rate dropped to 36/min, the surgeons noticed torrential bleeding from the sternotomy site due to rupture of the sac. Chest was opened quickly and internal suckers of CPB were turned on. Clots were removed expeditiously from the sac, and the surgeons allowed the patient to cool down further by occluding the hole in the ascending aorta with a finger. We also placed ice packs around the patient's head and added inj. thiopentone sodium 125 mg in the CPB to prevent neurologic injury. At 21°C, bypass was stopped, the surgeons quickly closed the defect with 4-0 Prolene suture (Ethicon, Inc, Somerville,

NJ), and CPB with rewarming was started. Total arrest time was 2 min. The sutures were reinforced by Teflon pledgets during rewarming. At 32°C, the heart went into ventricular fibrillation and was cardioverted using sterile external pads with 120 J. Sinus rhythm was restored and after completion of rewarming we could successfully wean the patient from bypass after transfusing her with 2 packed cells and starting inj. dopamine at a rate of 5 mcg/kg/min. Additional inj. fentanyl and inj. rocuronium were given to the patient. Her blood gas values were within normal limits and hematocrit was 24.

Patient was shifted to the ICU and extubated uneventfully the next day and shifted to the ward on the 2nd day without any neurological sequel.

DISCUSSION

Pseudoaneurysm of the ascending aorta can present with a pulsatile suprasternal mass or as chest pain as a consequence of myocardial ischemia due to compression of coronary artery bypass grafts by the pseudoaneurysm, dysphagia, or stridor.^[5-7]

Previous cardiac surgery is the most frequent cause of aortic pseudoaneurysms; even so, pseudoaneurysm occurs in less than 0.5% of all cardiac surgical cases.^[2]

During CPB, the ascending aorta is usually chosen as a site for cannulation,^[8] and is a potential site for pseudoaneurysm formation. The aortic suture lines made during aortic valve replacement or the CABG insertion site are also potential locations.^[9] A needle may be inserted into the aorta during surgery for pressure measurements, or to purge the aorta of air, or to inject cardioplegic solutions.^[10] Pseudoaneurysms have formed at these needle puncture points.

The surgical management of ascending aortic pseudoaneurysms remains a challenge. Authors report a mortality rate ranging from 29% to 46%, most frequently as a consequence of fatal hemorrhage resulting from rupture of the pseudoaneurysm during sternal re-entry or surgical maneuvers for its repair.^[1]



Figure 1: Pulsatile mass in the neck

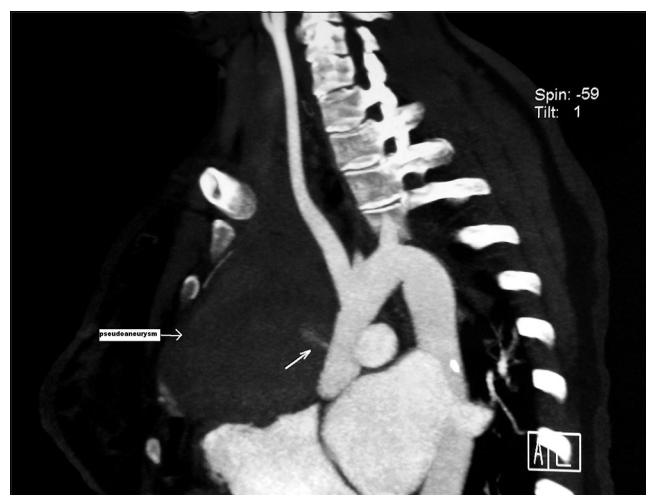


Figure 2: CT scan showing the pseudoaneurysm

The ascending aortic pseudoaneurysm mainly bulges anteriorly while eroding the bony structures of the sternum. The most important part of the surgery is to avoid bleeding during re sternotomy and to maintain proper cerebral perfusion,^[11] hence CPB is established before sternotomy.

Femoral or axillary arterial cannulation for CPB has been suggested, whereas others have used femorofemoral and bilateral carotid artery cannulation.^[12]

Hypothermic circulatory arrest, which is used to control the depletion of intravascular volume and to enable the preservation of cerebral function, provides time to perform the mediastinal dissection and to control the aortic defect.

Our concerns as anesthesiologist are airway distortion due to aneurysmal compression of airways, blood loss, avoiding adverse neurological sequel, and preparedness for cardioverting heart caused by arrhythmias arising due to hypothermia.

In our case induction before CPB was possible because there was no airway distortion else. Accidental opening of the sac before full arrest led to bleeding, but was quickly controlled by the surgeons. Further cooling, inj. thiopentone sodium, and short arrest time prevented neurological injury. Later, hypothermia caused ventricular fibrillation, which was cardioverted to sinus rhythm by external paddles as ministernotomy did not allow full exposure to the heart to place internal paddles. Ministernotomy was chosen as surgeons expected a lot of adhesion due to previous surgeries. Aortic cross clamping and cardioplegia were not required because the defect was small which also led to favorable outcome.

CONCLUSION

Post cardiac surgery pseudoaneurysms should be prevented by performing proper suture technique, careful handling of the aorta wall, strict asepsis, and aggressive treatment of the perioperative infection. If a large-sized pseudoaneurysm is located at the retrosternal space, then there is a very high risk of massive bleeding. To avoid this risk, we performed femorofemoral bypass before opening, and under deep hypothermia with

transient circulatory arrest performed the surgery. We had a successful outcome without any neurologic sequel.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

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Conflicts of interest

There are no conflicts of interest.

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