

# Complex pseudoaneurysm of ascending aorta: Unusual cause of right heart dysfunction-implications to the anesthesiologist

Prachi Kar, Ramachandran Gopinath, Durga Padmaja, R. V. Kumar<sup>1</sup>

Department of Anaesthesia and Intensive Care, <sup>1</sup>Department of CTVS, Nizams Institute of Medical Sciences, Punjagutta, Hyderabad, Telangana, India

## Abstract

Pseudoaneurysm of ascending aorta (PAA) is a rare complication occurring after cardiac surgery. Because of rarity of the condition, most standard teaching and anesthetic literature do not highlight on these postoperative aortic complications. Right heart dysfunction associated with PAA is scarcely reported. We describe here two cases of PAA with right heart involvement and discuss the possible anesthetic challenges.

**Key words:** Pseudo aneurysm of ascending aorta, pseudoaneurysm to right atrial fistula, right coronary artery compression, right ventricular dysfunction

## Introduction

Pseudoaneurysm of ascending aorta (PAA) is a rare clinical entity, which may occur after thoracic trauma or following cardiac surgery. Natural course of the pseudoaneurysm following cardiac surgery is unpredictable. These aneurysms are reported to produce symptoms due to mass effect or fistulous communications with the cardiac chambers. Right heart dysfunction is a less often reported complication of PAA. The literature concerning the anesthetic implications of these cases is also scarce. We report two cases of postcardiac surgery PAA, presenting as right heart dysfunction and discuss the possible challenges in anesthetic management.

## Case Reports

### Case 1

A 2-year-old child who was operated for closure of ventricular septal defect (VSD) a year previously, presented to our

hospital with the complaints of pedal edema, puffiness of face, hoarseness of voice and shortness of breath for last 7 days duration. The chest X-ray revealed an opacity in the right lung field. Transthoracic echocardiography (ECHO) revealed a PAA with fistulous communication to right atrium (RA) [Figure 1a] and shift of interatrial septum to left side, moderate pulmonary hypertension and mild to moderate right ventricular (RV) dysfunction (TAPSE-1.3). A three-dimensional reconstruction computed tomography (CT) scan confirmed the PAA with a size of 5.5 cm × 4.6 cm × 4.8 cm, neck of 1 cm and fistulous communication to RA [Figure 1b] and multiple venous collaterals suggestive of obstruction of superior vena cava (SVC) flow although no direct compression of SVC could be demonstrated [Figure 1c]. The child was scheduled for repair of PAA. Anesthesia was induced with titrated doses of fentanyl and midazolam so as to achieve a balance between hemodynamics and sympathetic stimulation, which may trigger a rise of pulmonary vascular resistance. Due to impaired SVC drainage, airway edema leading to difficult intubation was anticipated in this patient. Difficult airway cart was kept ready, and ease of ventilation with face mask was confirmed before administering the muscle relaxant. Patient was ventilated with 100% oxygen to maintain normocarbia. N<sub>2</sub>O and high airway pressures were avoided so as to minimize further worsening of RV function. After institution of femorofemoral bypass and midline sternotomy, the patient was cooled to 25°, aorta was cross-clamped and cardioplegia was given. Thereafter, the PAA opening was closed with a synthetic patch under transient period of low flows and reinforced with a bovine pericardium (St. Jude Medical).

Address for correspondence: Dr. Prachi Kar,  
Flat No A-102, Lake View Mirra Residency, Brahmanwadi, Begumpet,  
Hyderabad - 500 016, Telangana, India.  
E-mail: prachikar@yahoo.co.in

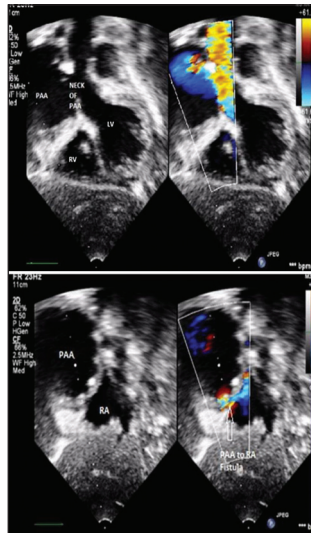
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Further, the pseudoaneurysm to RA fistula was closed with a similar patch. Injection milrinone 25 mcg/kg bolus over 10 min followed by infusion at 0.375 mcg/kg/min, dopamine 5 mcg/kg/min, and nitroglycerin 0.25 mcg/kg/min were initiated during rewarming. Child was weaned off from cardiopulmonary bypass uneventfully on these supports. The child was extubated the next day and had an uneventful postop course with regression of the preoperative symptoms. He was doing well at 30 days follow-up.

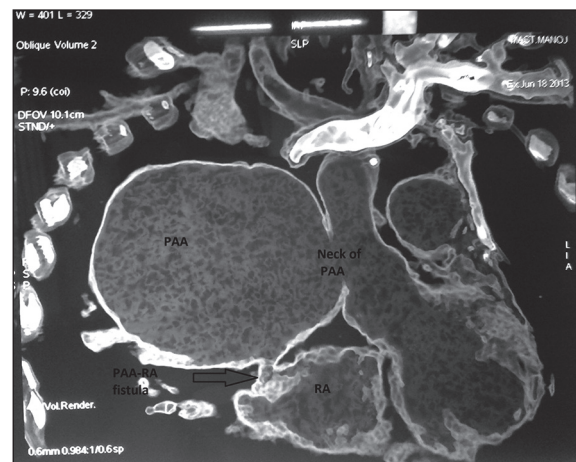
### Case 2

This case describes a 17-year-old boy who was electively operated for VSD closure and aortic valve replacement for aortic regurgitation with a 25 St. Jude nonrotatable valve. He was readmitted 1 year later with gradually enlarging pulsatile swelling of the chest, off and on episodes of chest pain, pedal edema and raised liver enzymes suggestive of right heart failure. Transthoracic ECHO showed a large PAA with

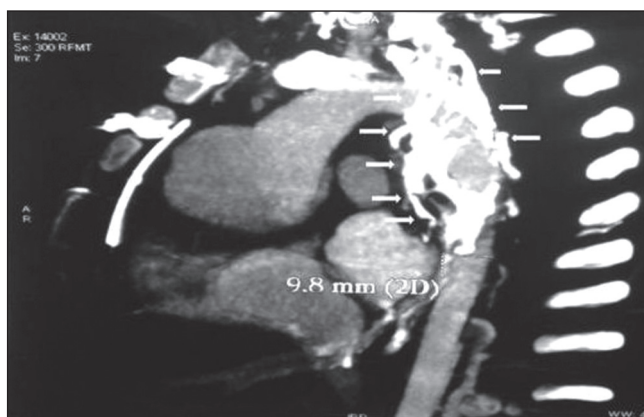
peripheral thrombus, and compression of RV outflow tract (RVOT) [Figure 2a], mild to moderate RV dysfunction. CT angiography confirmed the presence of a 10.5 cm × 5.9 cm × 11.2 cm pseudoaneurysm arising from the anterior wall of ascending aorta with a small neck (7 mm) [Figure 2b]. It was abutting the ostioproximal right coronary artery (RCA) with significant compression [Figure 2c]. Bony erosion of sternum by aneurysm was also noted. Anesthesia was induced with fentanyl 200 mcg, midazolam 3 mg, sevoflurane and intubated with rocuronium 30 mg. Induction of anesthesia and intubation were uneventful. Femorofemoral bypass was established before commencing sternotomy so that in the event of PAA rupture during sternotomy immediate circulatory arrest can be instituted. The patient was gradually cooled to 24° while avoiding ventricular fibrillation and thus ventricular distension. Unfortunately, pseudoaneurysm ruptured during sternotomy and a circulatory arrest was immediately instituted. The ostium of the PAA could be accessed within 5 min, and a finger was insinuated into the aneurysm cavity to occlude it. The aorta was



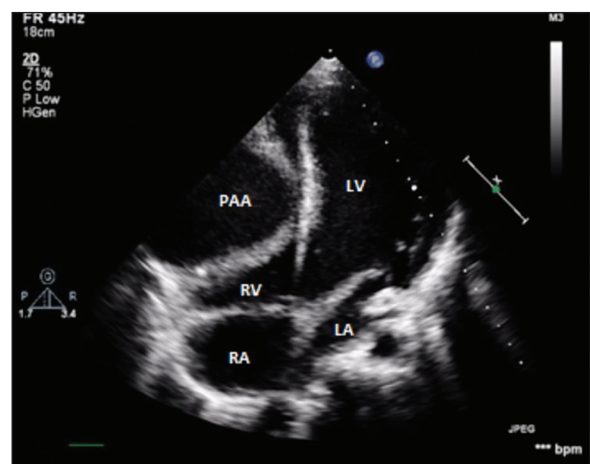
**Figure 1a:** Transthoracic echocardiography with color flow Doppler showing neck of pseudoaneurysm of ascending aorta (PAA) and PAA-right atrium fistula



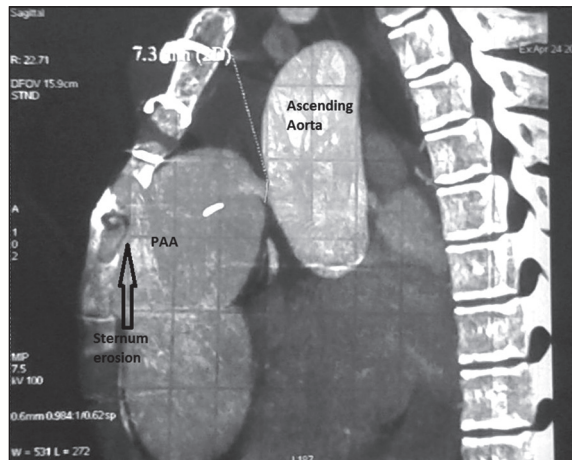
**Figure 1b:** Computed tomography scan showing neck of pseudoaneurysm of ascending aorta (PAA) and PAA-right atrium fistula



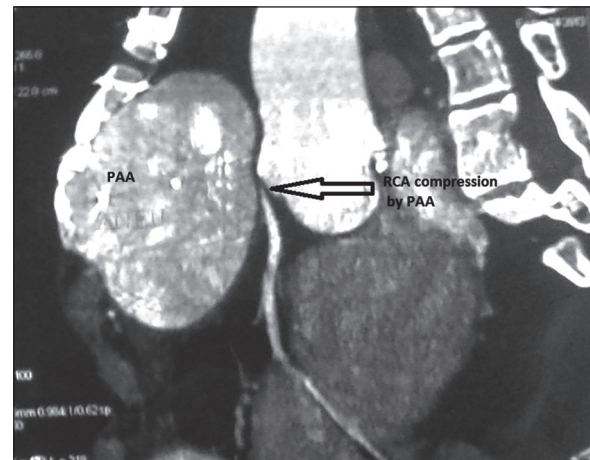
**Figure 1c:** Maximum intensity projection computed tomography images shows the collaterals as a result of superior vena cava obstruction (marked by white arrows)



**Figure 2a:** Transthoracic echocardiography, apical four chamber view shows the pseudoaneurysm of ascending aorta compressing the right ventricle and interventricular septum



**Figure 2b:** Computed tomography scan showing pseudoaneurysm of ascending aorta, its neck and sternum erosion



**Figure 2c:** Computed tomography scan showing compression of ostioproximal right coronary artery by pseudoaneurysm of ascending aorta

cross-clamped and cardioplegia could be administered through both the coronaries as the aneurysm was decompressed. The neck of pseudoaneurysm was closed with bovine pericardium patch. Injection milrinone infusion was started in the rewarming period in view of the RV dysfunction. The patient was successfully weaned off bypass. The postoperative course was uneventful. The patient was extubated on 2<sup>nd</sup> postoperative day and was discharged on 10<sup>th</sup> postoperative day.

## Discussion

Ascending aortic pseudoaneurysm is a rare, but life-threatening complication after cardiac surgery and thoracic trauma. Poor surgical suture technique, pre-existing aortic wall disease, perioperative infection are predisposing factors of pseudoaneurysm.<sup>[1]</sup> Moreover, inadequately treated infection may cause recurrence of the pathology.<sup>[2]</sup> Most common sites of origin of pseudoaneurysm include aortotomy or anastomotic suture lines, aortic and cardioplegia cannulation sites, cross-clamp sites, bypass graft sites, and needle puncture holes.<sup>[1,3]</sup> The presentation of PAA is highly variable. Some may remain asymptomatic and get detected incidentally while others present with severe symptoms. The size, site, anatomic location, and pressure effect on neighboring structures determines the symptoms of the PAA. SVC obstruction, aorto-pulmonary fistula, RV inflow obstruction, fistula into RA, RV and LV and angina are a few reported complications.<sup>[4-9]</sup> Right heart dysfunction is an unusual presentation of PAA.

Right ventricular dysfunction in the perioperative setting is mostly due to three causes. Pressure overload, volume overload and impaired RV contractility.<sup>[10]</sup> Pressure overload in most cases is a result of raised pulmonary vascular resistance, RVOT obstruction or pulmonary stenosis whereas volume overload is mostly due to tricuspid regurgitation, pulmonary regurgitation,

atrial septal defect or anomalous venous return. Impaired RV contractility may be due to RV ischemia, inadequate protection during cardiopulmonary bypass, or coronary air.

This report describes two cases of RV dysfunction, one presenting as volume overload (PAA-RA fistula), and the other as pressure overload (RVOT compression) with intermittent ischemia (RCA compression by PAA).

Pseudoaneurysm of ascending aorta-RA fistula is a rare presentation of PAA. This patient had a PAA-RA fistula which imposes a large volume overload on the RA and RV. The flow through the PAA fistula could have resulted in high RA pressure with a leftward shift of the atrial septum on the ECHO. This high RA pressure could also have caused impaired SVC drainage in our patient despite lack of any direct mechanical obstruction. Our patient presented with sudden onset severe puffiness of the face with hoarseness of voice suggestive of impaired SVC drainage. SVC obstruction can jeopardize the upper airway due to edema. This may be aggravated after induction in the supine position. The presence of hoarseness of voice as with our case should alert the anesthesiologist to plan airway access. Our second patient presented with off and on angina due to RCA compression which is another rare presentation of PAA.<sup>[9]</sup> The right coronary insufficiency along with RVOT obstruction could have resulted in RV dysfunction in this patient. These patients are also at risk for postoperative deterioration of RV dysfunction due to insufficient myocardial protection during cardiopulmonary bypass. Our second patient was specifically at risk of worsening of RV function due to circulatory arrest without cardioplegia for 5 min. However, lower temperature might have been protective. Milrinone, an inodilator, was the choice of inotrope in both our cases. It is known to be beneficial in patients with RV dysfunction.<sup>[11]</sup> Early institution

of an inodilator during management of our cases might have led to uneventful postoperative course.

Although percutaneous interventions have been reported as a management strategy for PAA, literature is scarce and is not well established. Surgery is still the gold standard. A big danger in pseudoaneurysm surgery is the risk of intraoperative rupture and is a major cause of mortality.<sup>[3]</sup> Erosion of the sternum further adds to the risk. Various techniques have been described for controlling the hemorrhage in PAA during redo sternotomy such as temporary occlusion of pseudoaneurysm ostium using balloon of Foley's catheter or even a fingertip.<sup>[12,13]</sup> As rapid exsanguination can occur, it is important to ensure availability of adequate blood and facilities for rapid transfusion.

Because of a rarity of the condition, most standard teaching and anesthetic literature do not highlight on these postoperative aortic complications of corrected cardiac lesions. These patients may also present for incidental surgeries. Thus, a thorough understanding of the underlying lesion, its pathophysiology, and anesthetic implications is essential for planning the management of these cases.

To summarize, right heart dysfunction though unusual can occur with PAA. Recognition and management of right heart dysfunction is important in improving outcomes. These two reports aid in understanding the anatomical and pathophysiological consequences of PAA and the implications for anesthetic management.

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