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Dissecting Thoracic Aortic Aneurysm Rupturing into Pulmonary Artery - A Rare Case Report

Ramkumar R[1], Ratish Radhakrishnan[2], John K Joy[3]

[1]Government Medical College, Thiruvananthapuram
Email: *drramkumaronline[at]gmail.com*

[2]Government Medical College, Thiruvananthapuram
Email: *drratishr[at]gmail.com*

[3]Government Medical College, Thiruvananthapuram
Email: *johnkjoy1993[at]gmail.com*

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1. Introduction

Aortic aneurysm is defined as the permanent localized or diffuse dilation of aorta more than 1.5 times the normal caliber. Thoracic aortic aneurysms (TAA) lead to catastrophic complications like dissection and rupture. Dissecting TAA is indeed a life - threatening condition that require prompt intervention. The incidence of TAA rupture is 5 per 100, 000 persons per year with a mortality rate of 97 - 100 %.1 Rupture into the pulmonary artery is an extremely rare complication, with only very few cases reported in literature. The Strongest predictor for rupture of TAA is diameter of the aneurysm. The odds ratio for rupture was increased to 27 - fold when the size of aneurysm exceeded 6cm.2Aorto - pulmonary fistula may be due to Aortic dissection, Marfan disease, trauma, inflammatory aortitis.

We present a case of a 58year old gentle man with DeBakey type III or Stanford type - B (figure 1) dissecting TAA rupturing into the pulmonary artery, leading to cardiogenic shock upon presentation to the emergency department. After initial resuscitation the patient underwent descending thoracic aortic replacement with a dacron graft combined with left pneumonectomy and repair of the pulmonary artery.

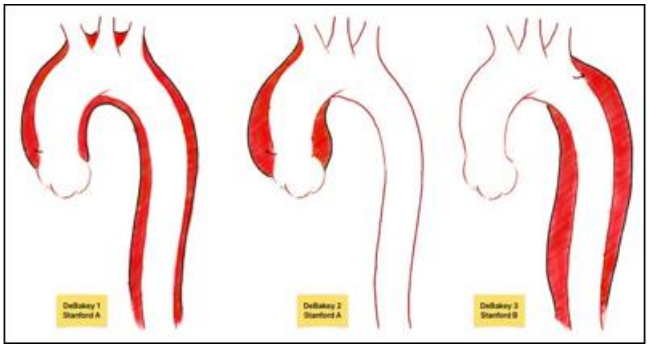

Figure 1: (Arrow pointing towards the intimal tear)

2. Case Report

A 58 year old male, chronic smoker, with hypertension, dyslipidemia and chronic obstructive pulmonary disease presented to the emergency department with sudden onset central chest pain, dyspnea and hemoptysis. Initial assessment revealed signs of cardiovascular shock and respiratory failure necessitating emergency intubation and mechanical ventilation. Auscultation revealed continuous murmur. Chest X - ray was suggestive of mediastinal widening (figure 2). Contrast enhanced computed tomography three dimensional reconstructed images revealed an aneurysmal Sac communicating into the pulmonary artery. (Figure 3)

Figure 2

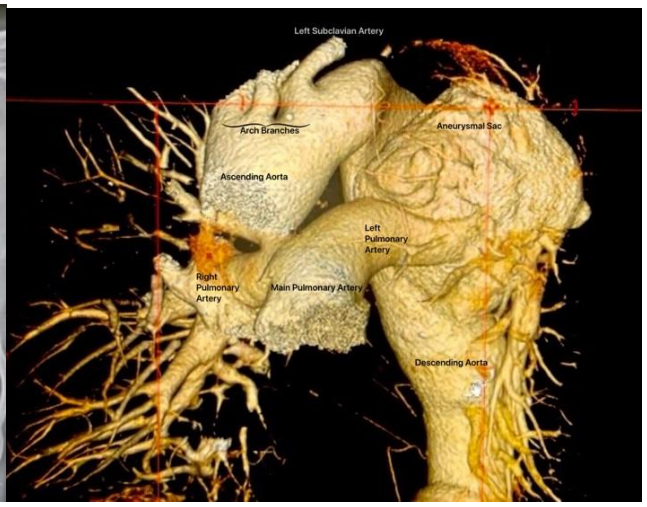

Figure 3

Emergency contrast enhanced computed tomography of thorax revealed a 10 cm x 9.5 cm x 10 cm saccular TAA (figure 3) extending from 1 cm distal to origin of left subclavian artery, dilated main pulmonary artery (figure 4 - MPA - Main pulmonary artery) rupturing into left pulmonary artery (figure 5 - LPA - left pulmonary artery) The aneurysmal sac compressing left main bronchus. (figure 6). The coronary arteries were normal. Transthoracic echocardiography suggested Good left ventricular function with severe tricuspid regurgitation and right ventricular dysfunction.

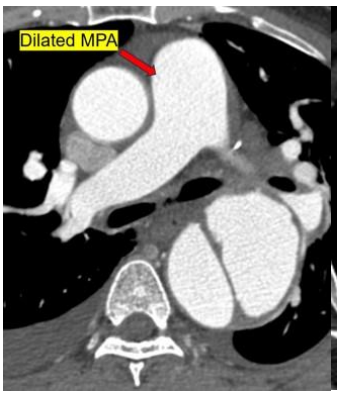

Figure 4

Figure 5

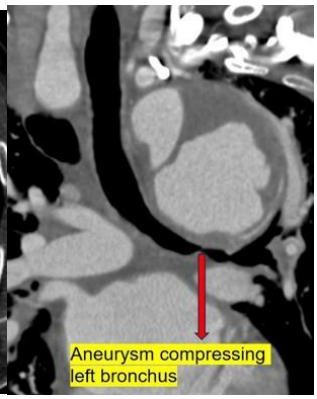

Figure 6

3. Treatment

Patient underwent open midline sternotomy with a 7 cm transverse incision from midline in the 4th left intercostal space. On cardiopulmonary bypass with peripheral right femoral artery and right atrial cannulation. Upper body perfusion maintained with ascending aorta cannulation. Aorta was cross clamped proximal and distal to the aneurysmal sac. Aneurysmal sac was opened longitudinally and thrombus delivered out. Multiple fistulous communications to the left pulmonary artery and its branches were identified & repaired. Descending thoracic aorta was replaced with 24 mm Dacron graft. (figure 7) Left lung was severely congested. The left main bronchus was severely deformed and collapsed. (figure 8 - The forceps is pointed at the aorto - pulmonary fistula site).

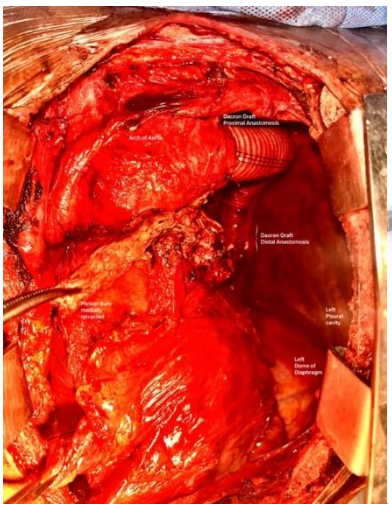

Figure 7

Figure 8

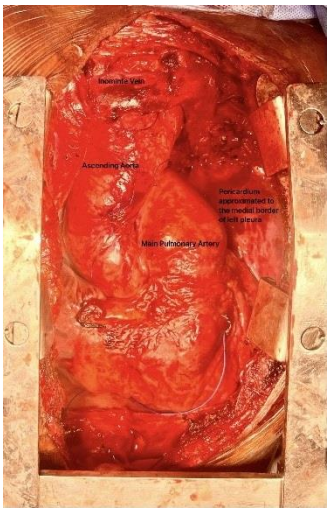

Figure 9

As the left lung appeared to be non - salvageable, left pneumonectomy was done. Staged sternal closure was done with a tailored compartmentalization of the pleural and pericardial cavity. The edge of pericardium was approximated to the medial border of respective pleural edge. (figure 9 - white arrows - pericardium sutured into the medial pleural border thus separating middle mediastinal compartment from the graft in left pleural cavity) The left pleural cavity is thus isolated to aid gradual filling of the cavity. The post - operative period was stormy with respiratory failure requiring tracheostomy and prolonged ventilation. Mediastinal and right pleural drain were removed on post - operative day 7 and left pleural drain was removed on post - operative day 13. X - ray chest on Post - operative day 3 (figure 10) and post - operative day 12 (figure 11) revealed gradual filling of the left pleural cavity. Post operatively developed neuropraxia of the right upper and lower limb which was managed with physiotherapy. The patient was discharged on post - operative day 32.

Figure 10

Figure 11

4. Discussion

Acute aortic dissection & aortic aneurysm rupture are leading causes of death among cardiovascular disease. Aortic dissection with intramural hematoma without intimal tear, penetrating atherosclerotic ulcer, and impending or ruptured aortic aneurysm have recently been categorized as acute aortic syndrome.[3] Deep coma or death due to sudden hypotension is seen in patients with free - wall rupture. Hemoptysis or hematemesis may be seen in erosion or sealed rupture into the lung or esophagus.[4] cardiogenic shock is seen when aneurysm ruptures into the pericardial cavity due to tamponade[5]. A rupture rate of 77 % was reported by Pressler and McNamara[6] and Bickerstaff et al[7] observed a higher rupture rate of 95%. In fact, more than 80% of mortality in patients with dissecting aortic aneurysms are due to rupture.[8] The key causes of mortality in TAA rupture are cardiac tamponade and hemorrhage.[9, 10] Most patients with thoracic aortic aneurysm rupture die within 6 hours of rupture. Only 40 % of the patients may present to the hospital alive. The mortality may be as high as more than 50 % at 6 hours and more than 75 % at 24 hours day after the initial event.[11]

5. Conclusion

This case underscores the complexity and challenges in managing dissecting TAA with unusual complications such as rupture into the pulmonary artery. Early recognition, prompt surgical intervention, and multidisciplinary collaboration are crucial in improving outcomes for such critically ill patients. The primary goal of initial treatment should be pointed to control pain and to achieve a stable hemodynamic state. The prompt surgical treatment by replacing the diseased aortic segment with an artificial graft should be followed. As the mortality rate of a ruptured TAA is high, the strategies to reduce the death rate like efficient screening for the diagnosis, identification of risk factors for rupture and efforts should be made to increase the number of surgeries for ruptured TAA.

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