

# A Case of Stanford B Aortic Dissection with Aortic Arch Aneurysm Post Patent Ductus Arteriosus Device Closure Repair

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## Abstract

## Case Report

Aortic Dissection (AD) causes a fatal cardiovascular emergency due to an intimal breach in the aorta, leading to the separation of the aortic wall layers either antegrade or retrograde results in development of a true and false lumen that will eventually compromise blood flow to the affected vital organs. On the other hand, PDA device closure repair has become an established safe intervention with an excellent post-procedure outcome. However, complications post-procedure have been reported, with vascular-related complications although rare but deems significant when it happens. We reported a case of chronic Stanford B Aortic Dissection with Aortic Arch Aneurysm following the history of PDA Device Closure repair. Its pathophysiology is described based on device-duct disproportions, foreign body inflammatory reactions and aortic wall chronic irritation. Although it's considered rare, there were few published case reports on similar events reported as acute and chronic aortic dissection. This case aims to look upon the relationship of Aortic Dissection as a rare but possible complication post PDA Device Closure Repair procedure.

**Keywords:** Aortic Dissection, Stanford Type B, PDA Device Closure, Aortic Arch Aneurysm, Frozen Elephant Trunk.

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## INTRODUCTION

Aortic dissection is raised from the disrupted integrity of the aortic wall, which results in intimal layer tearing that can propagate antegrade or retrograde, which provides significant ischemic symptoms and signs involving multiorgan in systemic circulation.

Stanford Aortic Dissections is divided into Type A and Type B depending on its anatomical aortic tearing location. Type A involves ascending aorta and type B involves descending aorta, distal to left subclavian artery. It can present as acute or chronic; it occurs after 14 days from onset of symptoms.

Chronic Stanford B Aortic Dissection mainly affects approximately 30 people per million individuals per year. This report describes a 39-year-old hypertensive male with a history of Patent Ductus Arteriosus (PDA) Device Closure Repair at the age of 24-year-old who presented with chronic hoarseness of the voice with evidence of left vocal cord palsy for six months prior to AD diagnosis.

Computed Tomography Angiography (CTA) confirmed Stanford Type B AD with periaortic hematoma, mass effect and Aortic Arch Aneurysm

(AAA). The patient's blood pressure and pain were strictly controlled and subsequently proceeded with the Frozen Elephant Trunk Procedure with Thoracic Endovascular Aortic Repair (TEVAR) plan for later.

The occurrence of Chronic Stanford Type B AD post PDA device closure is uncommon and rare, but it carries significant implications of aortic leakage and rupture with multiorgan malperfusion that can lead to organ failure and mortality. Thus, immediate surgical intervention after clinical stabilization is performed in the majority of circumstances, especially in an acute setting.

This report aims to enlighten of the potential association between late aortic pathology following prior PDA intervention. Thus, deems the importance of imaging surveillance post-procedure.

## CASE PRESENTATION

This 39-year-old Chinese gentleman was referred to our Cardiothoracic surgery centre as an incidental finding of Stanford B Aortic Dissection (AD) with Aortic Arch Aneurysm (AAA).

A patient with underlying young hypertension with a history of PDA Device Closure Repair procedure done at the age of 24 years old and diabetes mellitus in March 2025.

The patient has a brief three-month history of hoarseness of voice in October 2024, thus was under regular follow-up by the Ear, Nose and Throat (ENT) department. Flexible Nasopharyngoscopy by ENT team shows left vocal cord palsy. Subsequently, the patient proceeded with a Contrast- Enhanced Computed Tomography (CECT) Neck and Thorax screening that was performed on April 2025 with findings of Stanford B AD with Aortic Arch Aneurysm (AAA).

Urgent admission was done, and the patient's blood pressure was kept under tight control of Systolic 100-110 with usage of antihypertensive includes beta blockers, alpha blockers, calcium channel blockers and ace inhibitors.

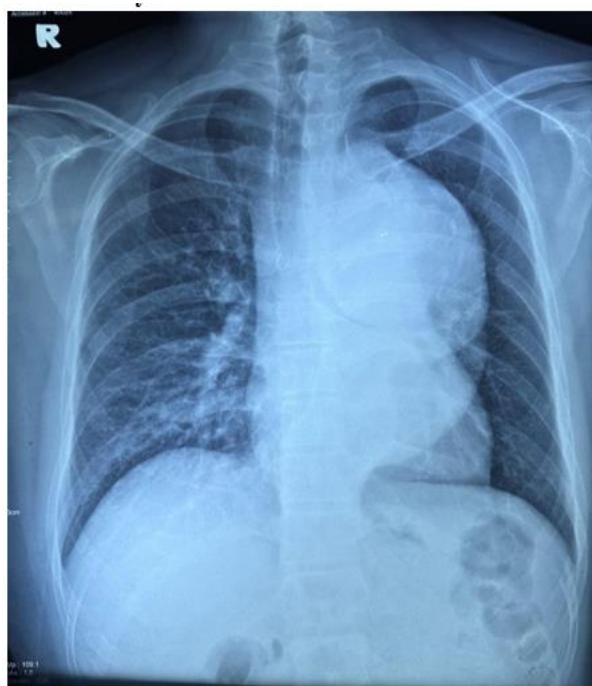
Routine investigation was done including: 1) full blood count; haemoglobin 12.9g/dl, total white count  $8.1 \times 10^3/\text{UL}$ , platelet  $240 \times 10^3/\text{ul}$ , 2) renal profile; urea 4.18mmol/L, creatinine 75umol/L, 3) coagulation profile; International Normalised Ratio 1.3, 4) arterial blood gas; PH 7.41, PCO2 34.1mmHg, PO2 72.6mmHg, HCO3 22.4mmol/L, Lactate mmol/dl, 5) crossmatch, 6) electrocardiogram (ECG); sinus rhythm, 7) chest x-ray; widened mediastinum, no pleural effusion, 8) echocardiogram; Ejection Fraction 55-60%, normal valves function, no pericardial effusion, 9) ECG Gated Computed Tomography Aorta (CTA) Thorax to Midhigh; Stanford B aortic dissection with periaortic hematoma, and mass effect. PDA Ligation device noted.

Intimal flaps are seen along the descending thoracic aorta and upper abdominal aorta. The entry point at T6/T7 level. The dissecting flap ends at the ostium of the superior mesenteric artery at T12 level. Periaortic hematoma noted along the arch of aorta and descending thoracic aorta, measuring 0.9cm in maximal thickness. No active contrast extravasation or pooling contrast in the delayed phase. Measurements: Ascending thoracic aorta: 3.2x3.3cm, Arch of aorta: 5.4x5.6cm, Mid descending aorta: 8.3x8.4cm, Descending thoracic aorta at level diaphragm: 3.4x3.0cm, Abdominal aorta at level coeliac axis: 3.3x2.8cm, Abdominal aorta prior to bifurcation: 1.7x1.6cm. The aneurysmal dilatation is causing splaying of the main pulmonary arteries and displacement of the main bronchus anteriorly. Brachiocephalic trunk, subclavian and common carotid arteries patent. No haemothorax or hemo-pericardium. Full assessment was done prior to surgical intervention. Subsequently, the patient was scheduled for Frozen Elephant Trunk in July 2025. Patient operative details will be explained further below.

Post-operative patient initially recovers well. However, the patient's further recovery was unfortunately complicated with multiple bouts of sepsis secondary to hospital acquired pneumonia, bacteraemia and surgical site infection during prolong stays in the critical care unit and ward. Requiring a total of three surgical Sternal Wound Debridement due to Ruptured Infected Pseudoaneurysm with Infected Sternal Wound in September 2025.

## INVESTIGATION IMAGES

### 1) Chest X-Ray



**Figure 1: Chest X-Ray: Widened mediastinum.**

2) ECG CTA Thorax till Midthigh

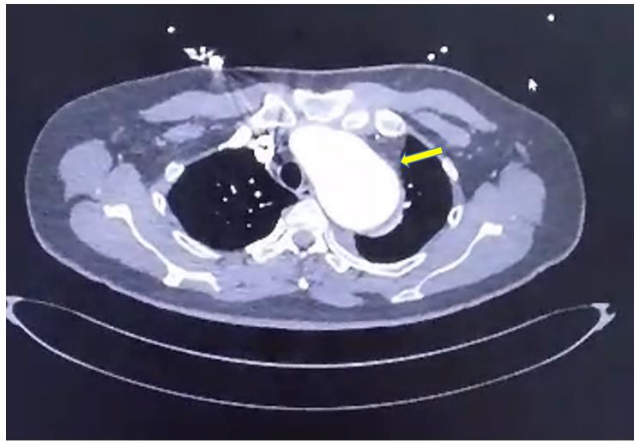


Figure 2: axial view of CTA: Yellow arrow: Arch of aorta aneurysm

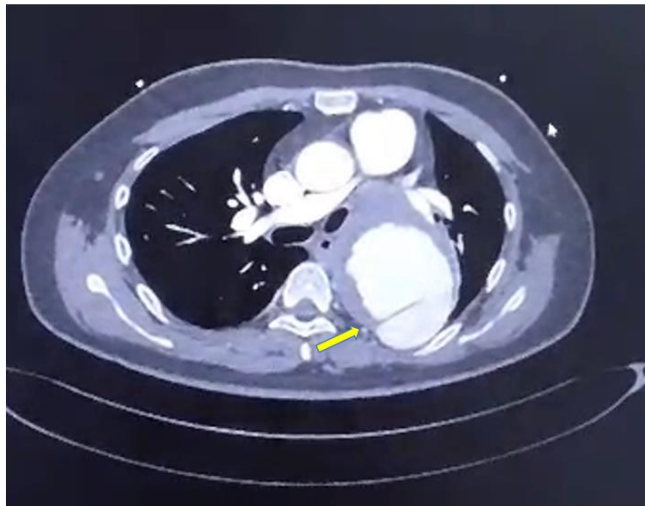


Figure 3: axial view of CTA showing; Yellow arrow: entry point of aortic dissection in descending thoracic aorta

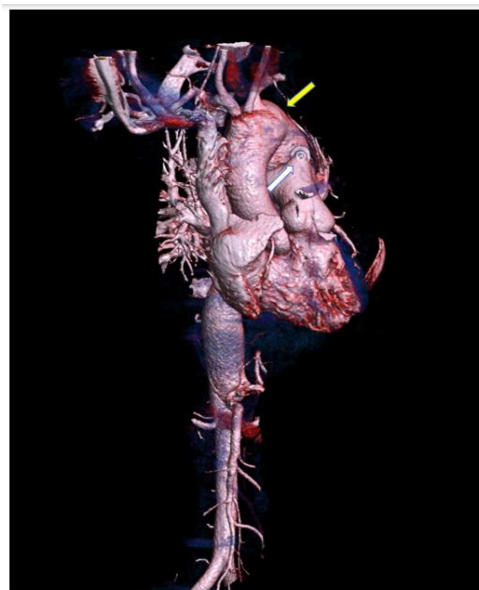
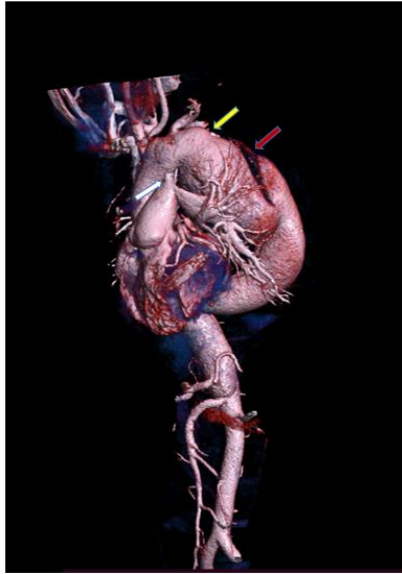


Figure 4: CTA 3D Image Anterior. White arrow: PDA device, yellow arrow: Aneurysmal portion of aortic arch and proximal descending aorta

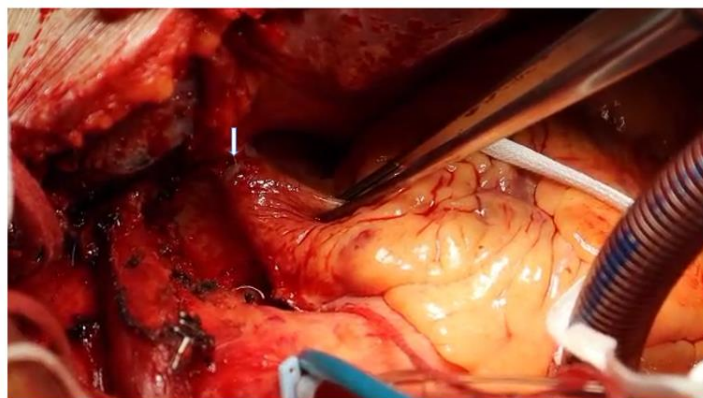


**Figure 5: CTA 3D Image Anterolateral. White arrow: PDA device, yellow arrow: Aneurysmal portion of aortic arch and proximal descending aorta, red arrow: Dissecting area descending thoracic aorta**

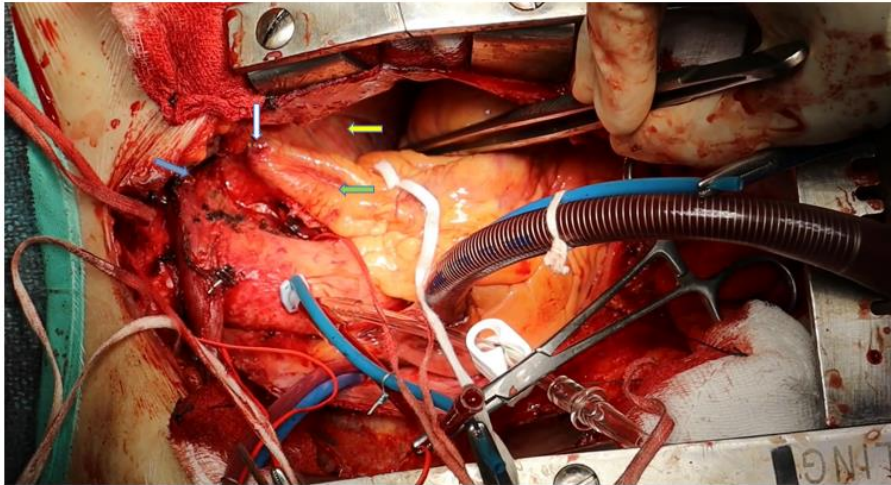


**Figure 6 : CTA 3D Image Posterior. Yellow arrow: Aneurysmal portion of aortic arch and proximal descending aorta, red arrow: Dissecting area descending thoracic aorta**

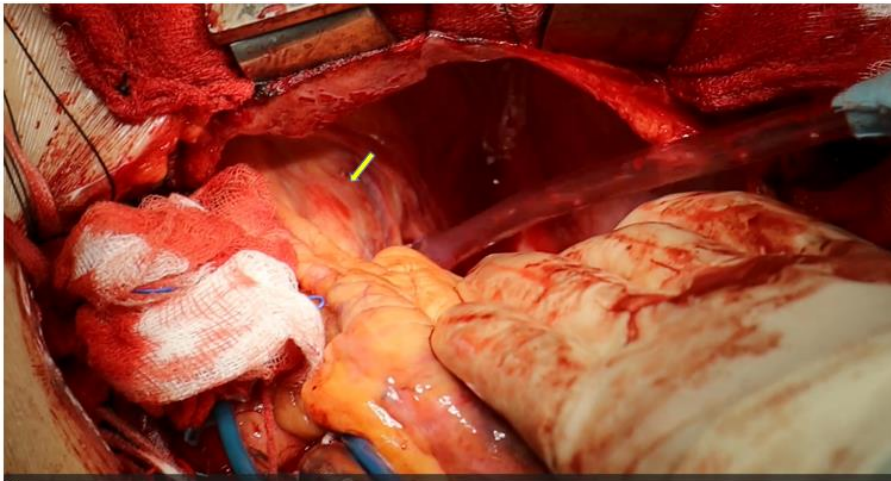
***SURGICAL TREATMENT***  
**OPERATIVE FINDINGS**



**Figure 7: Area pointed by white arrow is the inflamed area where PDA device located**

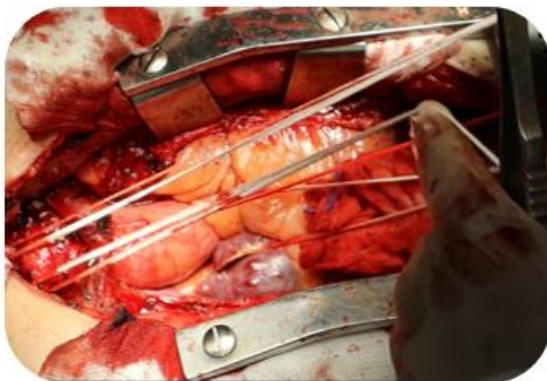


**Figure 8** White arrow: Inflamed area of PDA device, yellow arrow: Aneurysmal portion of proximal descending aorta, green arrow: Pulmonary artery, blue arrow: Aneurysmal Arch of aorta



**Figure 9:** Yellow arrow: Aneurysmal portion of proximal descending aorta distal to left subclavian artery.

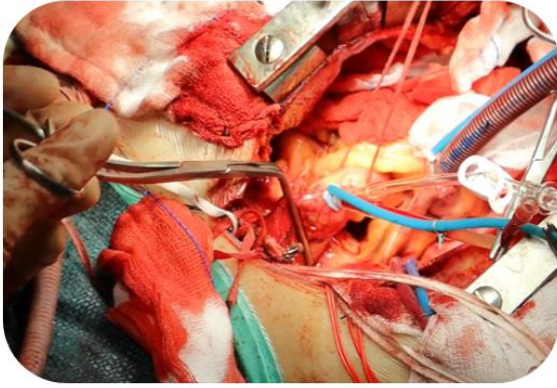
## 2) SURGICAL PROCEDURE AND TECHNIQUES



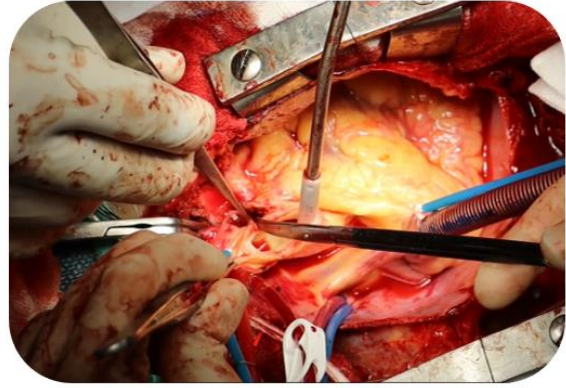
**1) Median sternotomy performed. Brachiocephalic trunk, left common carotid and left subclavian artery isolated.**



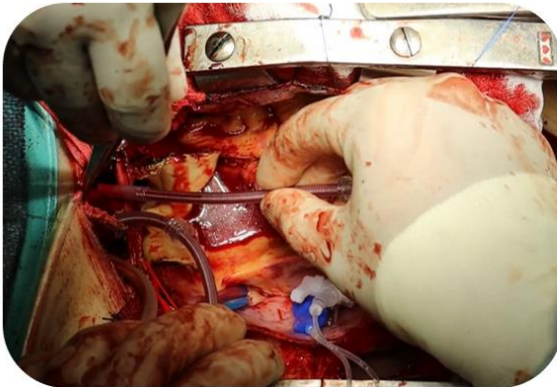
**2) Incision at left infraclavicular region and left axillary artery isolated, anastomosed to AlboGraft straight vascular graft 8mm. Initial systemic heparin given (3000 iu). Left axillary artery cannulated via the vascular graft**



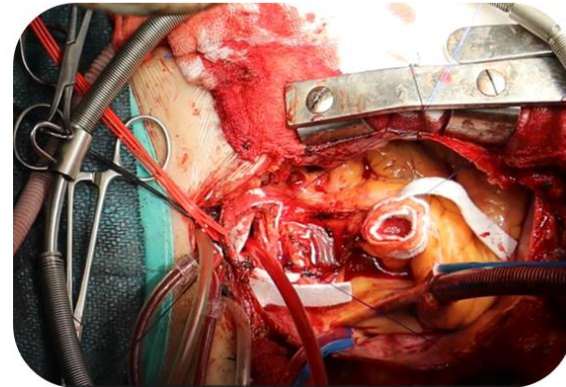
3) Full systemic heparin given. Right atrium cannulated with 2 staged venous cannula. Antegrade cardioplegia cannulated. CPB commenced and patient cooled down to 22°C. After aortic cross clamped, induction dose of cardioplegia (Del Nido) given via antegrade cardioplegia and maintained every 90 mins.



4) Aortotomy done over ascending aorta, findings as above.



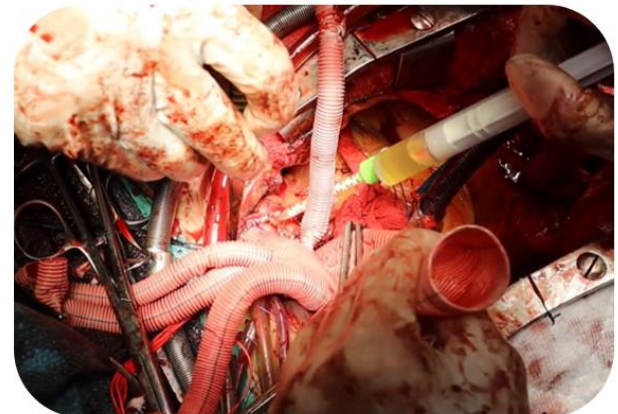
5) Once patient was cooled down to 22°C, patient was put on complete circulatory arrest. Pump off. Aortic cross clamp removed. Ascending aorta and arch excised until Zone 2 area. Antegrade cerebral perfusion was given through left and right common carotid arteries.



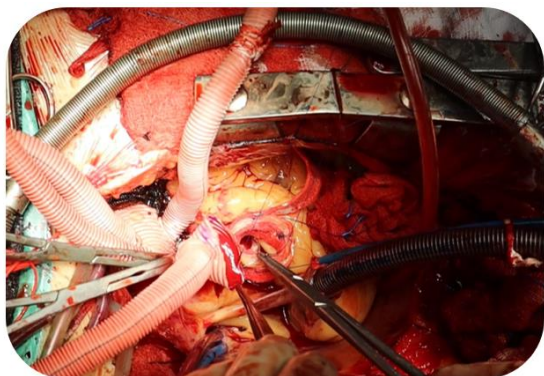
6) Proximal ascending aorta and Distal aortic arch wall was sandwiched between 2 layers of Teflon felt.



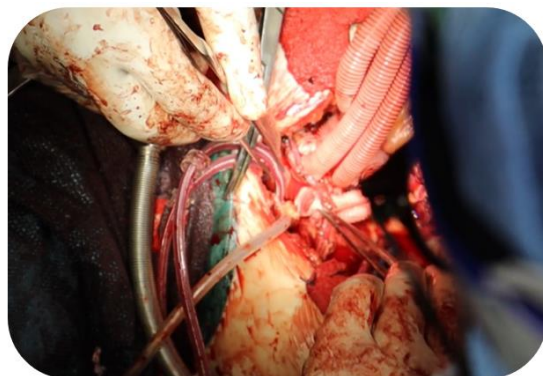
7) FET 28mm applied through guidance of guide wire inserted via femoral artery.



8) Distal anastomosis performed in 2 layers with Optilene 3/0. Aortic cross clamp was reapplied proximal to the anastomosis site and circulation was resumed to the descending aorta via side branch on FET graft.



9) Proximal end of FET graft was anastomosed to the proximal end of reinforced proximal ascending aorta in 2 layers of pledgetted Optilene 3/0. All surgical anastomosis sites were reinforced with biogluue.



10) Neck vessel anastomosis: Patient was rewarmed to 32°C. Left common carotid and brachiocephalic trunk was anastomosed end-to-end with the limbs of the FET trunk respectively with optilene 5/0. The axillary artery AlboGraft straight vascular graft was tunneled into the thoracic cavity via the left 2nd intercostals space and anastomosed end-to-side with the corresponding limb of the FET graft with optilene 5/0.

Patient was rewarmed. Antegrade cannula inserted over FET graft (neo-ascending aorta) for deairing, and was sutured and reinforced with pericardial and Teflon pledgets once deairing is complete and cannula removed. All suture lines were checked and areas of bleeding reinforced with pledgetted sutures. Patient weaned off cardiopulmonary bypass uneventfully. Venous decannulated. Routine chest closure done.

## DISCUSSION

Aortic Dissection following the PDA closure procedure is a possible complication that has been reported and published as case reports. However, to date, the data on the exact incidence post PDA closure dissection are not well substantiated in view of its rare prevalence.

Among published case reports include; 1) David W Baker , David S Celermajer. ‘Acute aortic dissection

as a late and fatal complication of transcatheter persistent ductus arteriosus occlusion: a case report’. *European Heart Journal Case Report*, 2020 Dec 11;5(1): ytaa484. doi: 10.1093/ehjcr/ytaa484. 2) Youna Park , Hong Ryang Kil , Sang Yoon Kim , Geena Kim. ‘A Rare Case of Aortic Dissection 10 Years Post Percutaneous Catheterization Retrieval of an Embolized PDA Device in a Patient with Down Syndrome.’ *Pediatric Report MDPI*. 2025 Aug 8;17(4):84. doi: 10.3390/pediatric17040084.

Its pathophysiology leading to aortic dissection is mainly explained through the aortic wall structural damage from mechanical interaction of the PDA occlusive device, which can be presented as early or late. Mechanical interaction can lead to irritation, endothelial damage and long-term chronic inflammation at the site of device placement, that eventually causes the intimal tear of the aortic wall.

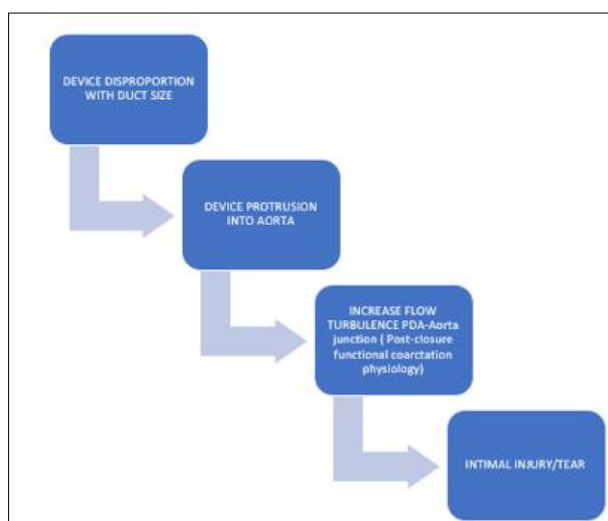
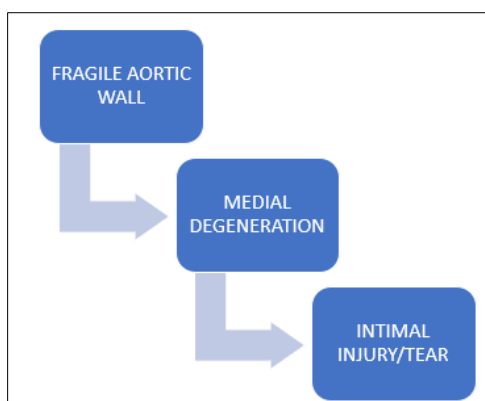
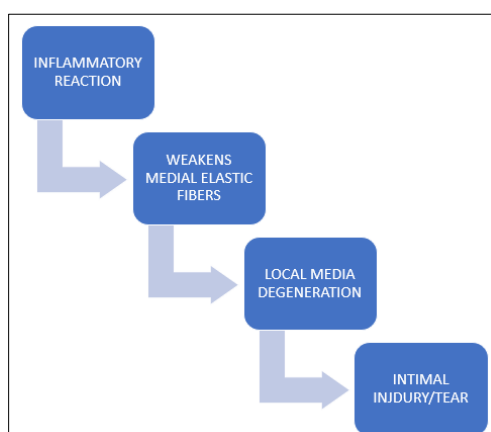


Figure 10: Pathophysiology-device disproportion



**Figure 11: Pathophysiology- aortic wall**



**Figure 12: Pathophysiology- inflammation**

In this case, intraoperatively we found that the tissue surrounding the PDA Closure device appears inflamed, hardened, fibrotic, and encompasses an area of aneurysm, however slightly proximal to the dissection area. This inflamed area can trigger a chronic inflammatory device-aortic wall interaction mechanism that leads to structural damage to the intimal layers of the aortic wall, especially at the transition zone near the device's edge. This chronic microtrauma accelerates intimal injury. In addition, the patient has underlying young hypertension that could further create a dissection propagation of the aortic inner wall due to chronic effects on fragmentation of elastic fibers and medial thinning.

However, any possible causal association as in this case report, should be furthered objectively proven through serial imaging; 1) computed tomography angiography to check on entry tear originating adjacent to the PDA device, pseudoaneurysm formation at device's edge, device protrusion into descending aorta and focal isthmus narrowing, 2) magnetic resonance angiography to evaluate chronic wall fibrosis, flow turbulence and compliance mismatch across device.

## CONCLUSION

Aortic Dissection following the PDA closure procedure is a rare but a possible complication with

further data on the exact incidence warrant for proper roundup and evaluations.

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