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An Unusual Combination of Giant LAD Aneurysm with Coronary AV Fistula Draining into the Main Pulmonary Artery and Common Coronary Ostium: A Case Report

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Abstract: ***The coexistence of a giant left anterior descending artery (LAD) aneurysm, a coronary arteriovenous (AV) fistula draining into the main pulmonary artery (MPA), and a common coronary ostium supplying both the right coronary artery (RCA) and left main coronary artery (LMCA) from a single leftward-facing coronary sinus is an extraordinarily rare constellation of congenital coronary anomalies. We report a 54-year-old female presenting with a one-year history of palpitations and exertional dyspnoea (NYHA Class II), found to have a continuous murmur at the pulmonary area. Multimodality imaging including 2D echocardiography with transesophageal echocardiography (TEE) and CT coronary angiography (CTCA) identified a giant proximal LAD aneurysm measuring 6.3 × 4.9 cm, a tortuous LAD-to-MPA fistulous connection with continuous left-to-right shunt, left atrial dilatation, preserved LV function (EF 60%), and a common RCA-LMCA ostium from the leftward-facing coronary sinus. The patient underwent successful surgical repair of the LAD aneurysm and fistula with concomitant coronary artery bypass grafting (CABG) using two saphenous vein grafts and has recovered well. This case highlights the diagnostic challenges of complex congenital coronary anomalies in adults, the indispensable role of CTCA in anatomical delineation, and the importance of multidisciplinary surgical planning.***

Keywords: coronary artery aneurysm; coronary AV fistula; pulmonary artery fistula; single coronary ostium; CABG; congenital coronary anomaly

1. Introduction

Coronary artery aneurysms (CAAs) are defined as focal dilatations exceeding 1.5 times the diameter of the adjacent normal coronary segment. The overall incidence of CAAs on coronary angiography ranges from 0.3% to 5.3%, with a pooled mean incidence of approximately 1.65%.[1] Giant CAAs, conventionally defined as those exceeding 2 cm in diameter, are even rarer, with a reported incidence of only 0.02%.[2] In order of frequency, the most commonly affected vessels are the RCA (40%), the LAD (32%), the left circumflex artery (23%), and the LMCA (3.5%).[3] Saccular aneurysms show a particular predilection for the LAD over other coronary arteries.[3]

Coronary arteriovenous fistulae (CAVFs) are rare abnormal connections between a coronary artery and a cardiac chamber, coronary sinus, or great vessel, first described by Krause in 1865.[4] They are detected in 0.1–0.2% of all coronary angiographic studies and account for 0.2–0.4% of congenital cardiac anomalies.[5] Approximately 90% of fistulae drain into right-sided cardiac structures: 40% into the right ventricle, 25% into the right atrium, 15–20% into the pulmonary artery, and 7% into the coronary sinus.[6]

The concurrence of a giant LAD aneurysm with a coronary-to-pulmonary artery fistula is exceedingly rare, with only isolated cases in the global literature.[7,8] The additional finding of a common coronary ostium supplying both the RCA and LMCA from a single leftward-facing sinus- a single coronary artery (SCA) anomaly with an angiographic incidence of approximately 0.04%[9] - renders this combination extraordinary. We report a 54-year-old female from AMCH, Dibrugarh, in whom all three anomalies were identified concurrently on multimodality imaging.

2. Case Report

Clinical Presentation

A 54-year-old female with a background history of hypothyroidism (on thyroxine replacement) presented to the Cardiology Department of AMCH, Dibrugarh, with a one-year history of palpitations (NYHA Class II) and exertional dyspnoea (NYHA Class II). The symptoms had remained in the same functional class throughout. There was no history of paroxysmal nocturnal dyspnoea, bilateral lower limb swelling, right hypochondrial pain, chest pain, or engorged neck veins. There was no history of cyanosis, clubbing, or failure to thrive in childhood.

Physical Examination

General examination revealed no pallor, icterus, cyanosis, clubbing, or peripheral oedema. Pulse rate was 92 beats per minute, regular, with normal volume and character; no radio-radial or radio-femoral delay was noted. Blood pressure was 128/78 mmHg (right upper limb), 126/78 mmHg (left upper

limb), 132 mmHg (right lower limb), and 134 mmHg (left lower limb), all in the supine position. SpO_2 was 98% on room air. Cardiovascular examination revealed normal heart sounds with physiological splitting of S2; no S3, S4, or ejection clicks. A continuous murmur was audible over the pulmonary area. Respiratory examination was unremarkable.

Differential Diagnosis

Given the continuous murmur without childhood symptoms or cyanosis, the following differentials were considered:

1) Patent ductus arteriosus (PDA): small-to-moderate PDA can remain asymptomatic for decades, producing a continuous murmur from progressive left heart volume overload.
2) Coronary artery-to-pulmonary artery fistula: produces an identical continuous murmur to PDA.
3) Coronary cameral fistula draining into the right ventricle or right atrium: can also produce a similar continuous murmur.
4) Ruptured sinus of Valsalva (RSOV) aneurysm: typically presents acutely with sudden-onset dyspnoea and haemodynamic compromise, making it less likely given the one-year symptom duration.
5) Aortopulmonary (AP) window: small defects can present late with similar clinical findings.

3. Investigations

Electrocardiogram (ECG): Normal sinus rhythm; within normal limits (Figure 1).

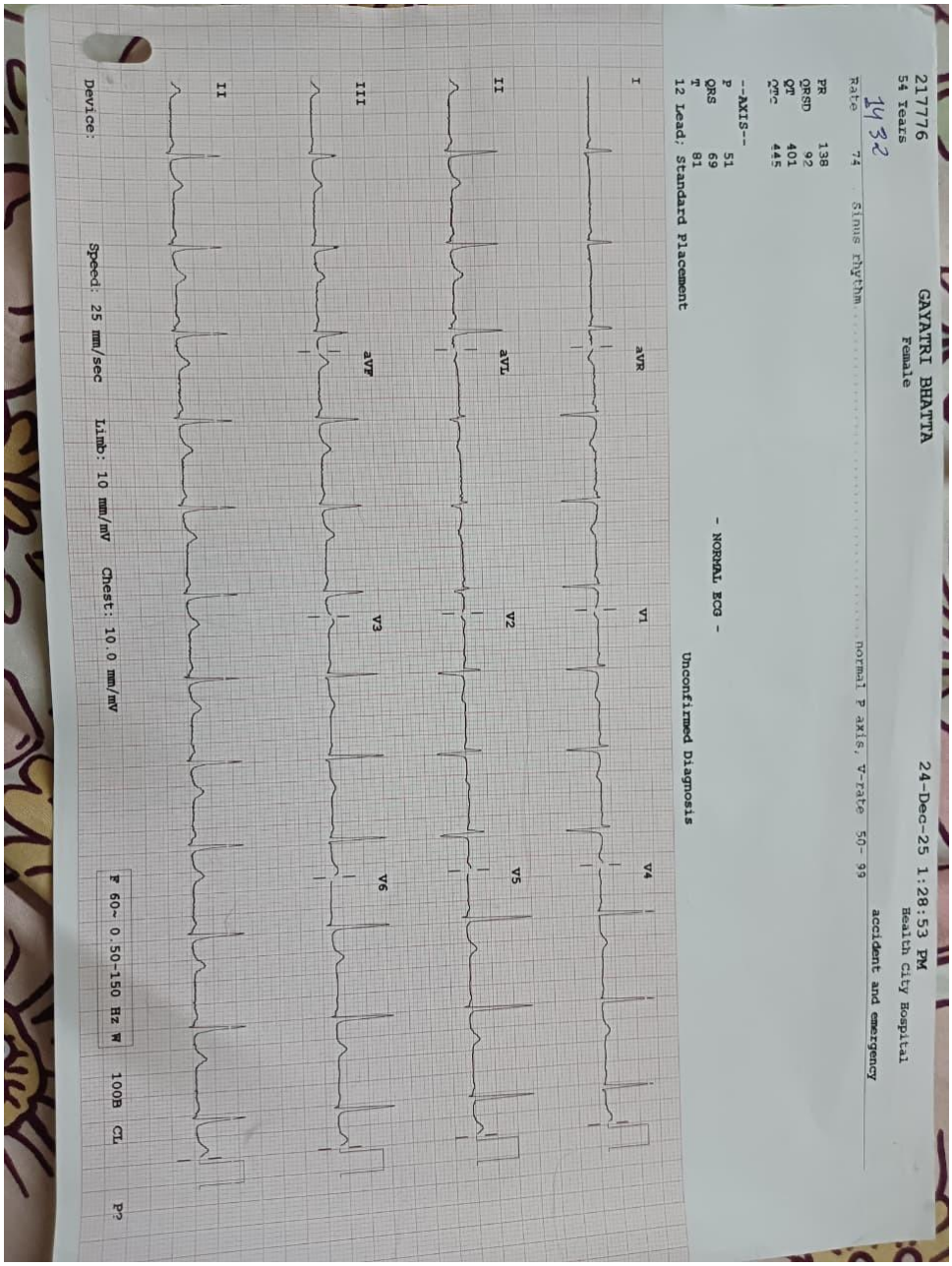


Figure 1: 12-lead ECG showing normal sinus rhythm with no significant ST-T changes.

Chest X-ray (PA view): A well-defined homogeneous opacity was seen along the left cardiac border with visualisation of underlying vascular markings, suggesting non-parenchymal origin. The lesion appeared localised to the anterior mediastinal region. Cardiomegaly was noted with hilar congestion (Figure 2).

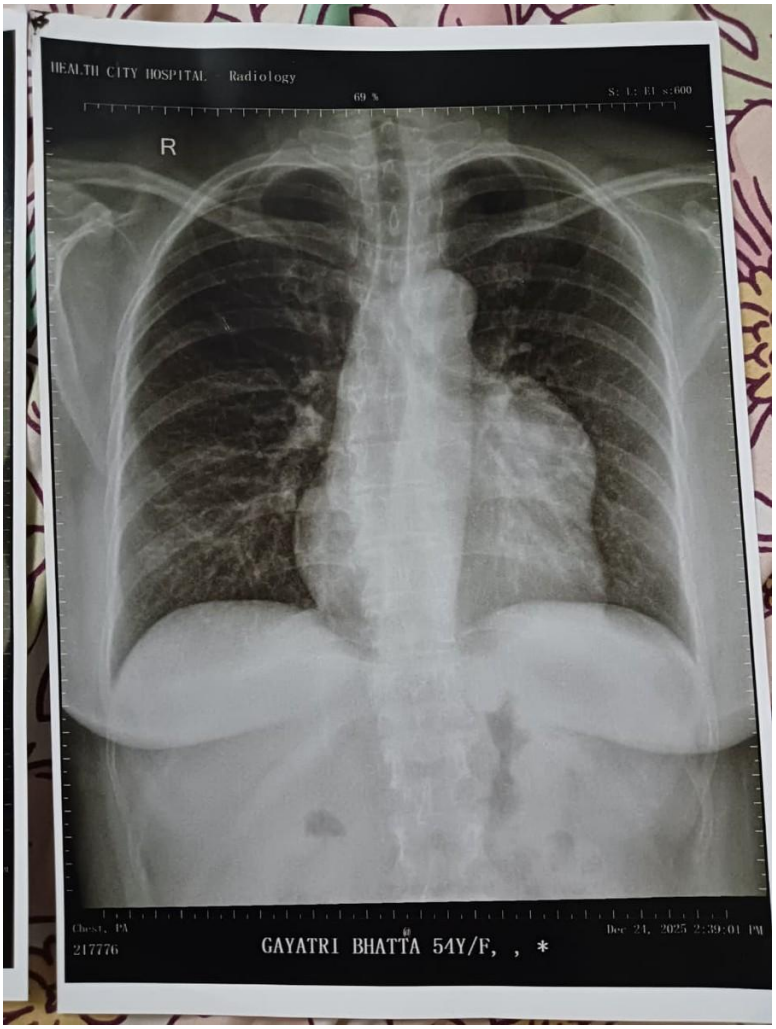
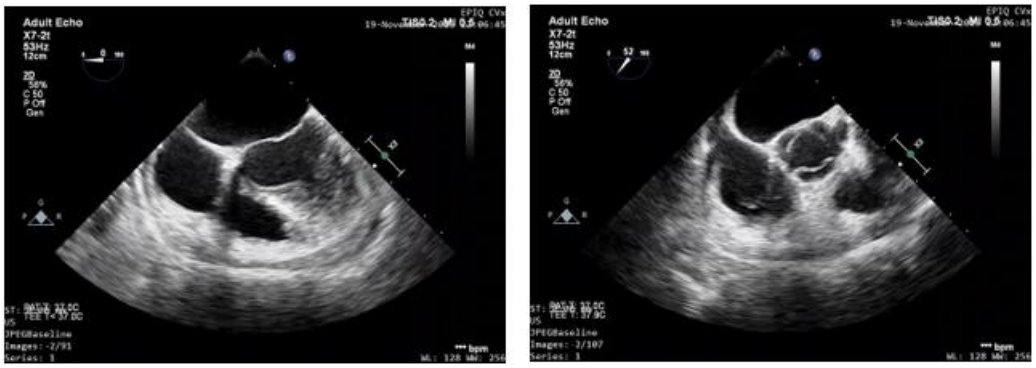
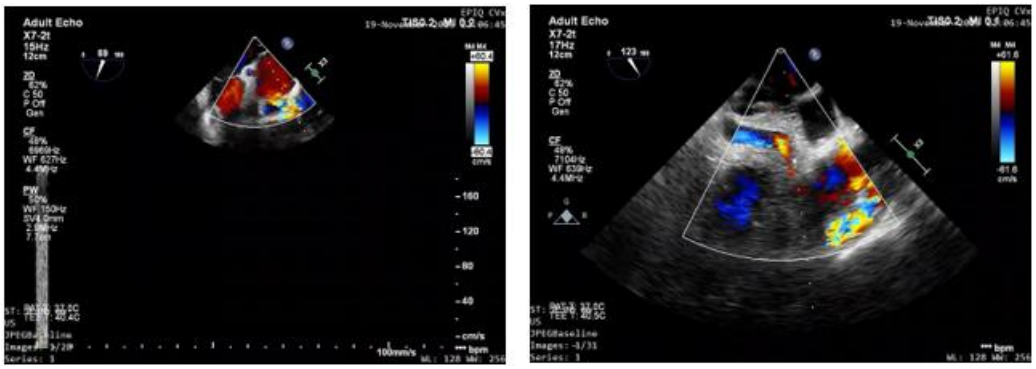


Figure 2: Pre-operative chest X-ray (PA view) demonstrating cardiomegaly, a well-defined opacity along the left cardiac border, and hilar congestion.

Two-dimensional Echocardiography / TEE: A large aneurysmal dilatation of the LMCA/LAD measuring 6.4 × 5.0 cm was identified. A fistulous connection between the LAD and pulmonary artery with continuous left-to-right shunt was demonstrated on colour Doppler. The left atrium was dilated with preserved left ventricular systolic function and LVEF of 60% (Figures 3–6).



Figures 3- 4: TEE images demonstrating the giant aneurysmal dilatation of the left coronary system in 2D views



Figures 5- 6: Colour Doppler TEE images showing the fistulous left-to-right flow from the LAD into the pulmonary artery (mosaic turbulent jet).

CT Coronary Angiography (CTCA): The LMA, LAD, and LCx appeared diffusely dilated. Aneurysmal dilatation of the proximal LAD measuring 6.3 × 4.9 cm was identified. The distal LAD coursed abnormally between the MPA and the aneurysmal sac, subsequently forming tortuous and dilated channels coursing anteriorly to the MPA and forming a fistulous connection with the MPA. The left atrium was dilated. A common origin of the RCA and LMCA from the leftward-facing coronary sinus was noted as an incidental but clinically significant finding (Figures 7–11).

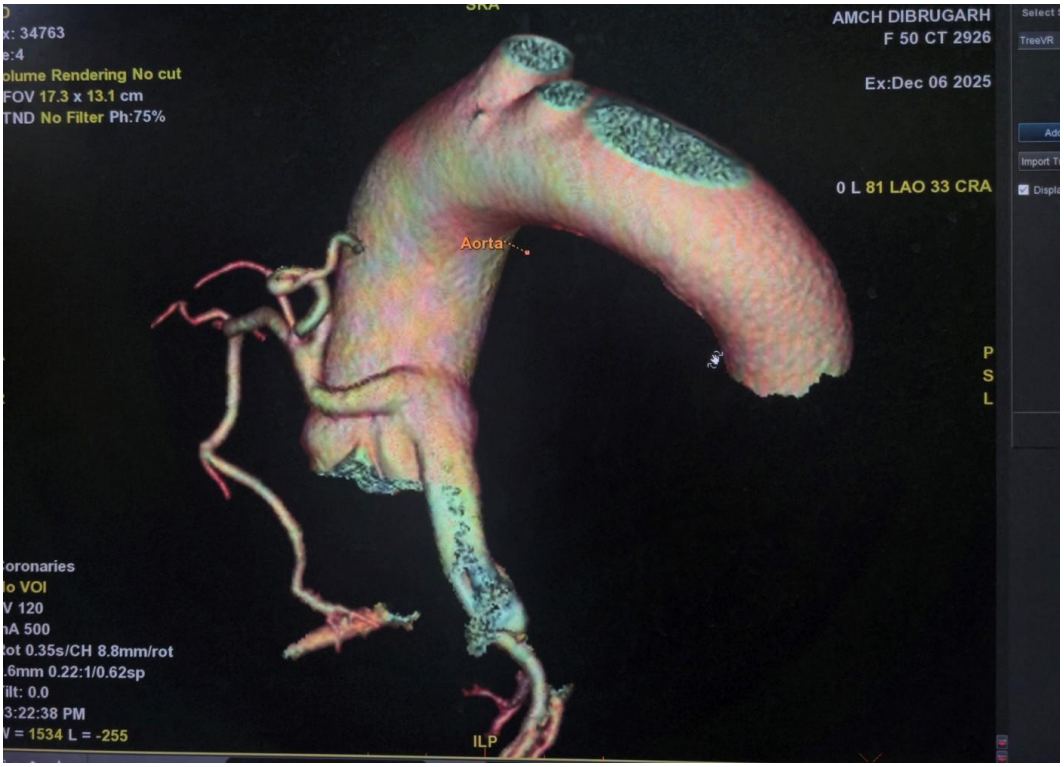


Figure 7: CTCA 3D volume-rendered reconstruction (LAO cranial view) demonstrating the giant LAD aneurysm arising from the proximal LAD and the tortuous fistulous channels coursing anterior to the MPA

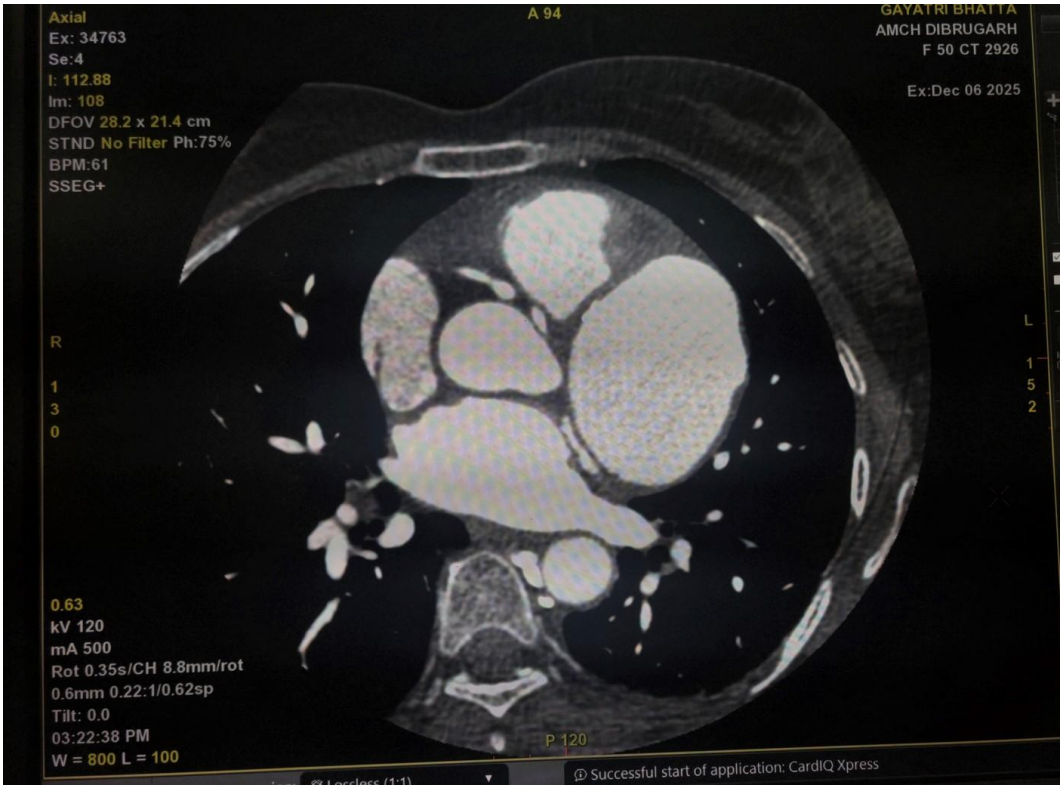


Figure 8: CTCA axial cross-section at the level of the aneurysm, demonstrating the massively dilated coronary aneurysmal sac with contrast enhancement, the main pulmonary artery, and cardiac chambers.

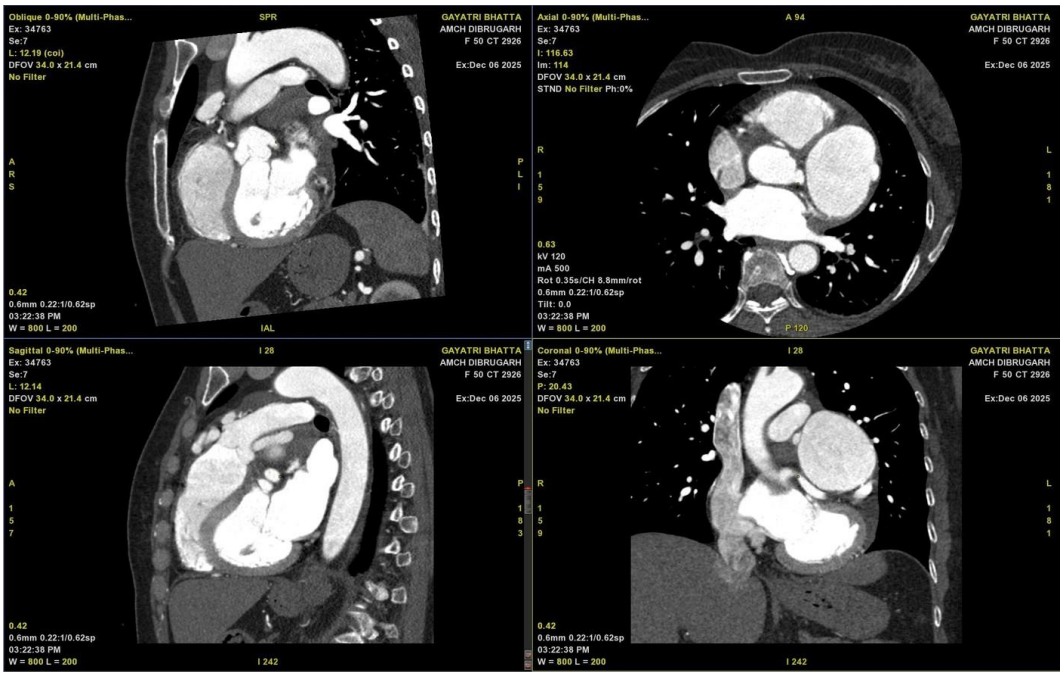


Figure 9: CTCA multiplanar reconstructions (oblique, axial, sagittal, coronal) showing the proximal LAD aneurysm, dilated LMA/LAD, and the tortuous LAD course.

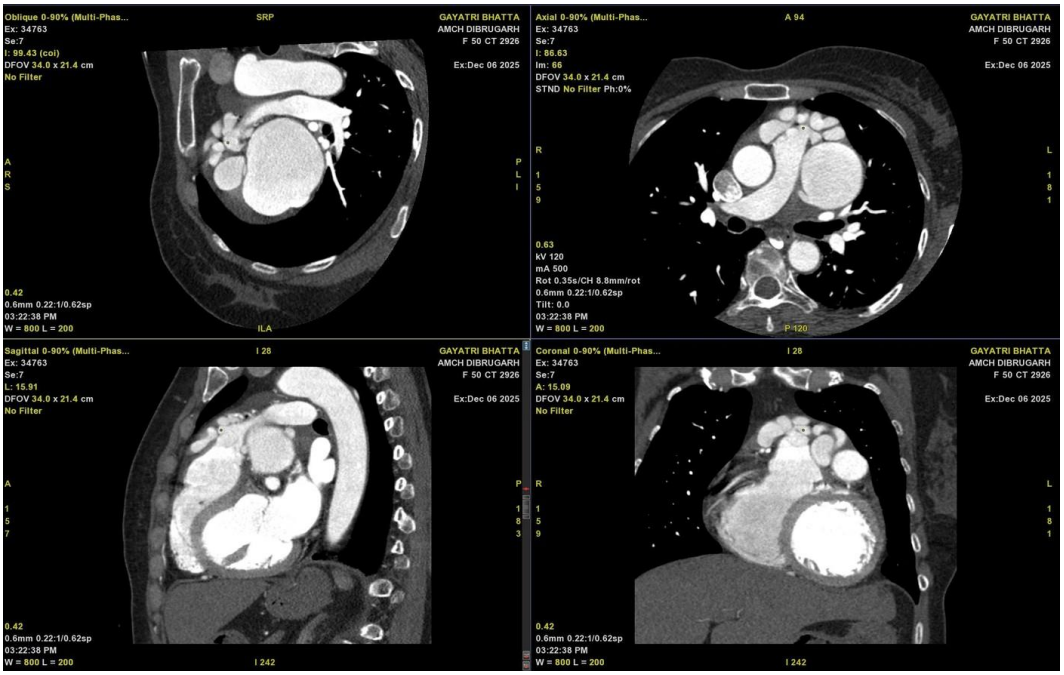


Figure 10: CTCA multiplanar reconstructions at a different level confirming the tortuous distal LAD forming the fistulous connection with the MPA, and the common RCA-LMCA ostium from the left-ward facing sinus

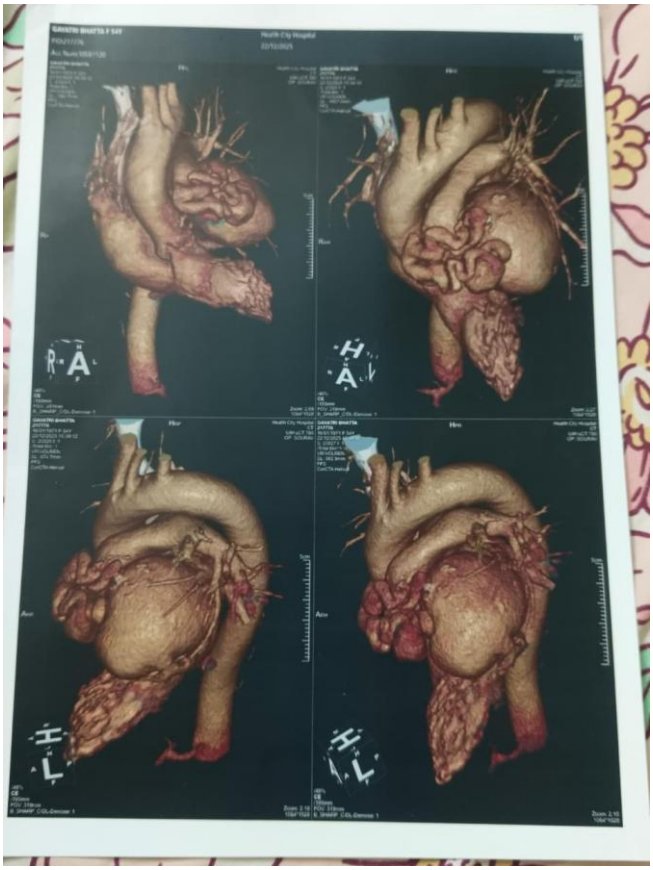


Figure 11: CTCA 3D volume-rendered reconstruction (four views) demonstrating the relationship of the giant aneurysm to the aortic root, MPA, and cardiac chambers.

Table 1: Summary of Key Investigations

Investigation	Finding
ECG	Normal sinus rhythm, within normal limits
Chest X-ray (PA)	Opacity along left cardiac border; cardiomegaly; hilar congestion
2D Echo / TEE	Aneurysm 6.4 × 5.0 cm (LMCA/LAD); LAD-to-PA fistula with L→R shunt; LA dilatation; LVEF 60%
CTCA	Proximal LAD aneurysm 6.3 × 4.9 cm; tortuous LAD-MPA fistula; common RCA-LMCA ostium from left sinus
Bloods	CBC, RFT, electrolytes, LFT, TSH — all within normal limits

Final Diagnosis

Aneurysmal dilatation of the proximal LAD with coronary AV fistula from the LAD to the main pulmonary artery, with common origin of the RCA and LMCA from the leftward-facing coronary sinus.

Management

The patient was referred to the cardiothoracic and vascular surgery (CTVS) team for definitive surgical repair. She underwent repair of the LAD aneurysm and fistula ligation with concomitant CABG using two saphenous vein grafts- SVG to mid-LAD (end-to-side) and SVG to mid-OM (end-to-side)- on 27 December 2025 at a higher centre in Guwahati.

Outcome

The postoperative chest X-ray demonstrated a satisfactory cardiac silhouette with sternal wires in situ (Figure 12). The patient is currently doing well at follow-up, with resolution of symptoms.

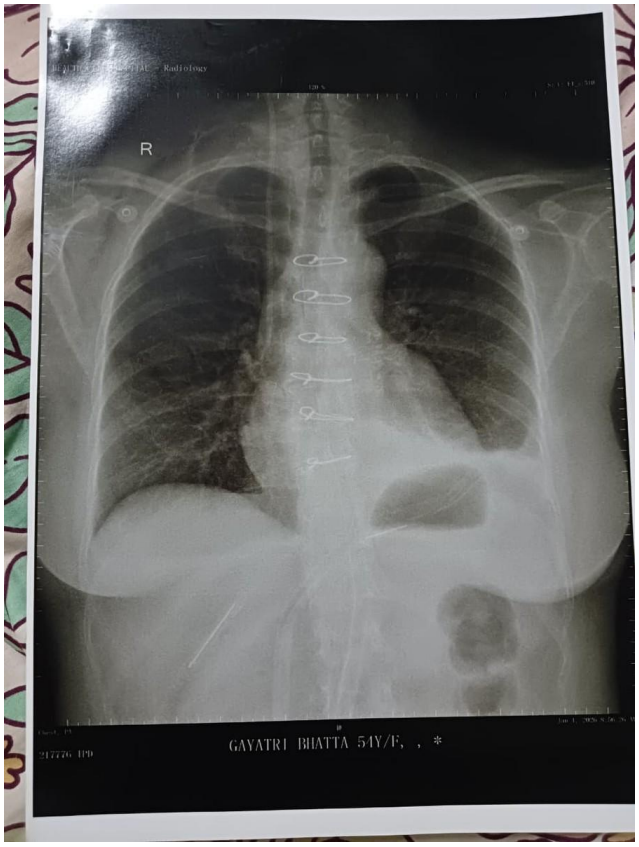


Figure 12: Post-operative chest X-ray (PA view) showing satisfactory cardiac silhouette, sternal wires, and resolution of the left cardiac border opacity

4. Discussion

Giant LAD Aneurysm

The landmark CASS registry analysis by Swaye et al. identified aneurysmal coronary artery disease in 4.9% of a large registry population, characterising it as a variant of coronary atherosclerosis rather than a distinct clinical entity.[10] In contrast, the giant aneurysm in our patient- measuring 6.3 × 4.9 cm on CTCA- in the absence of significant atherosclerotic risk factors, strongly favours a congenital or developmental aetiology. Giant coronary aneurysms carry well-recognised risks of intra-aneurysmal thrombosis with distal embolisation, progressive left heart volume overload, and spontaneous rupture.[2] The anomalous intrapericardial course of the distal LAD between the MPA and the aneurysmal sac, with subsequent tortuous fistulous channels confirmed on CTCA, is consistent with congenital coronary maldevelopment, as described by Morita et al. in their review of giant LAD-to-pulmonary artery fistula aneurysms.[11]

Coronary AV Fistula to the Main Pulmonary Artery

The continuous murmur heard at the pulmonary area in this patient reflects a persistent left-to-right shunt from the high-pressure LAD into the low-resistance pulmonary circulation. Tirilomis et al. confirmed that exertional dyspnoea, palpitations, and chest pain are the typical presenting features of coronary AV fistulae in adults, consistent with our case.[12] The 2018 ACC/AHA Guidelines classify large, symptomatic coronary fistulae with left heart volume overload as a Class I indication for closure.[13] CTCA is the gold standard for delineating fistula anatomy prior to intervention, as confirmed by Verdini et al.[14] In this case, echocardiography demonstrated a 6.4 × 5.0 cm aneurysmal dilatation with continuous left-to-right shunt, left atrial dilatation, and preserved LV function (EF 60%), reflecting significant haemodynamic burden. PDA, RSOV, coronary cameral fistula, and AP window were systematically excluded based on the one-year indolent course, absence of childhood symptoms, and multimodality imaging findings.

Common Coronary Ostium Anomaly

The incidental detection of a common ostium supplying both the RCA and LMCA from the leftward-facing coronary sinus on CTCA represents the third element of this unusual triad. Anomalous origin of the LMCA from the right sinus of Valsalva is rare, with a reported angiographic prevalence of 0.02–0.05%.[15] When associated with an interarterial course between the aorta and the right ventricular outflow tract, it constitutes a recognised cause of sudden cardiac death in young athletes, as established by Basso et al.[16] In our patient, the course did not appear malignant; however, the anomaly substantially complicated surgical planning and reinforced the indispensable role of pre-operative CTCA.

Surgical Management

Surgical repair is the definitive treatment for giant coronary aneurysms with fistulae, particularly when associated with symptomatic volume overload or risk of rupture.[7,8] Percutaneous transcatheter closure was not feasible given the giant aneurysmal sac, complex fistula morphology, and concurrent ostial anomaly. The patient underwent repair of the LAD aneurysm, fistula ligation, and concomitant CABG (SVG to mid-LAD, end-to-side; SVG to mid-OM, end-to-side) on 27 December 2025. Simultaneous revascularisation was mandatory, as ligation of the anomalous LAD without distal reconstruction would have resulted in anterior wall ischaemia. This strategy mirrors cases reported by Ikeda et al. (2025) and Suzuki et al. (2022).[7,8] The patient recovered uneventfully and is doing well at follow-up.

5. Conclusion

This case illustrates an extraordinarily rare triad of congenital coronary anomalies- giant LAD aneurysm, coronary AV fistula to the MPA, and common RCA-LMCA ostium — presenting in adult life. The case underscores the pivotal role of CTCA in delineating complex coronary anatomy, the need for a systematic approach to the differential diagnosis of continuous murmur, and the importance of multidisciplinary surgical planning incorporating simultaneous aneurysm repair, fistula ligation, and coronary revascularisation. Clinicians should maintain a high index of suspicion for congenital coronary anomalies in adults presenting with unexplained cardiac murmurs, even in the absence of childhood symptoms.

Declarations

Patient Consent: Written informed consent was obtained from the patient for publication of this case report.
Competing Interests: The authors declare no competing interests.
Funding: No funding was received for this report.

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