Case Report

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Incidentally Detected Dual-feeder Pulmonary Arteriovenous Malformation Mimicking a Pulmonary Mass on CT: A Case Report

Radiology Section

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ABSTRACT

Pulmonary Arteriovenous Malformations (PAVMs) are uncommon abnormal vascular communications between pulmonary arteries and pulmonary veins, resulting in right-to-left shunting. Although many lesions are asymptomatic and incidentally detected, PAVMs are clinically important because they may cause hypoxaemia, paradoxical embolism, stroke, or cerebral abscess. A 56-year-old female presented with fever, cough, and breathlessness of one week's duration. She was a known case of type 2 diabetes mellitus and systemic hypertension on regular medical treatment. On examination, the patient was febrile, with a reduced resting Oxygen Saturation (SpO_2 82% on room air), requiring supplemental oxygen. Initial chest radiograph demonstrated a focal, lobulated opacity in the right mid- and lower-lung zones. Non contrast chest Computed Tomography (CT) revealed a well-defined, multilobulated, soft-tissue density lesion in the right middle lobe abutting the pleura and pericardium, raising suspicion for a pulmonary neoplasm. Further evaluation with contrast-enhanced CT pulmonary angiography demonstrated intense, homogeneous enhancement of the lesion, with two feeding arteries arising from the right middle lobar pulmonary artery and drainage into the left atrium via a large accessory pulmonary vein, consistent with a complex aneurysmal PAVM. The diagnosis was subsequently confirmed on catheter digital subtraction angiography. The patient was managed conservatively with supportive care and optimisation of associated co-morbid conditions and close clinical follow-up. This case highlights the importance of contrast-enhanced CT angiography in differentiating PAVMs from pulmonary mass lesions and in accurately delineating angioarchitecture anatomy for management planning.

Keywords: Angiography, Computed Tomography, Digital subtraction, Pulmonary circulation, Vascular malformations

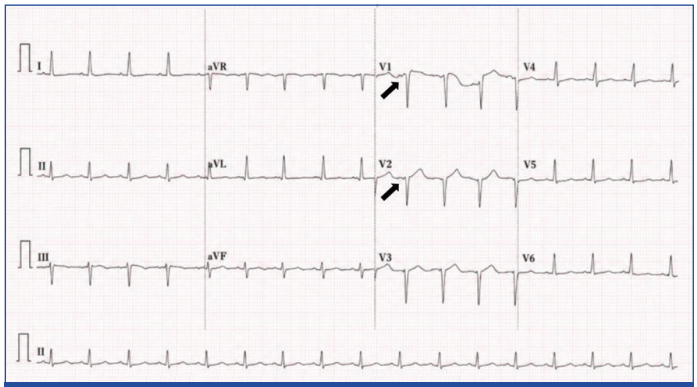
CASE REPORT

A 56-year-old female presented with fever, cough, and progressive breathlessness for one week. Breathlessness was insidious in onset and gradually progressive, associated with reduced exercise tolerance. The cough was non-productive and associated with intermittent fever. There was no history of chest pain, haemoptysis, cyanosis, orthopnoea, paroxysmal nocturnal dyspnoea, syncope, or previous similar complaints. There was no history of recurrent epistaxis, mucocutaneous telangiectasia, gastrointestinal bleeding, or family history suggestive of hereditary haemorrhagic telangiectasia. The patient was a known case of type 2 diabetes mellitus and systemic hypertension on regular medical treatment. Past surgical and family history was unremarkable.

On examination, the patient was conscious and oriented. The patient was febrile with a body temperature of 38.1°C (normal 36.5° - 37.5°C) , blood pressure was 129/70 mmHg, pulse rate was 80/minute, respiratory rate was 22/minute, and resting peripheral SpO_2 was 82% on room air (normal: 95-100%), for which oxygen supplementation was initiated. There was no cyanosis, clubbing, pedal oedema, mucocutaneous telangiectasia, or respiratory distress. Respiratory system examination revealed bilaterally reduced basal air entry.

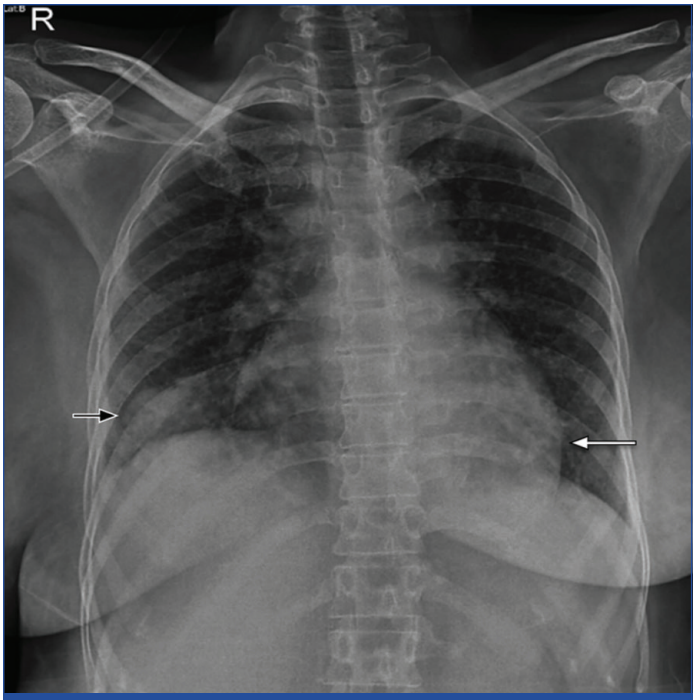
Spirometry demonstrated preserved pulmonary function with Forced Expiratory Volume in one second (FEV1) of 89% predicted (normal: ≥ 80% predicted) and FEV1/FVC ratio of 92% (normal: >70%). Laboratory investigations revealed elevated inflammatory markers- Erythrocyte Sedimentation Rate (ESR) 93 mm/hr (normal 20-35 mm/hr), C-Reactive Protein (CRP) 249.9 mg/L (normal <10 mg/L), and renal function derangement-Serum creatinine 1.8 mg/dL (normal 0.7-1.3 mg/dL), which improved during hospitalisation with conservative management.

Electrocardiogram (ECG) demonstrated a normal sinus rhythm with Q waves in leads V1-V3. [Table/Fig-1]. Frontal chest radiograph

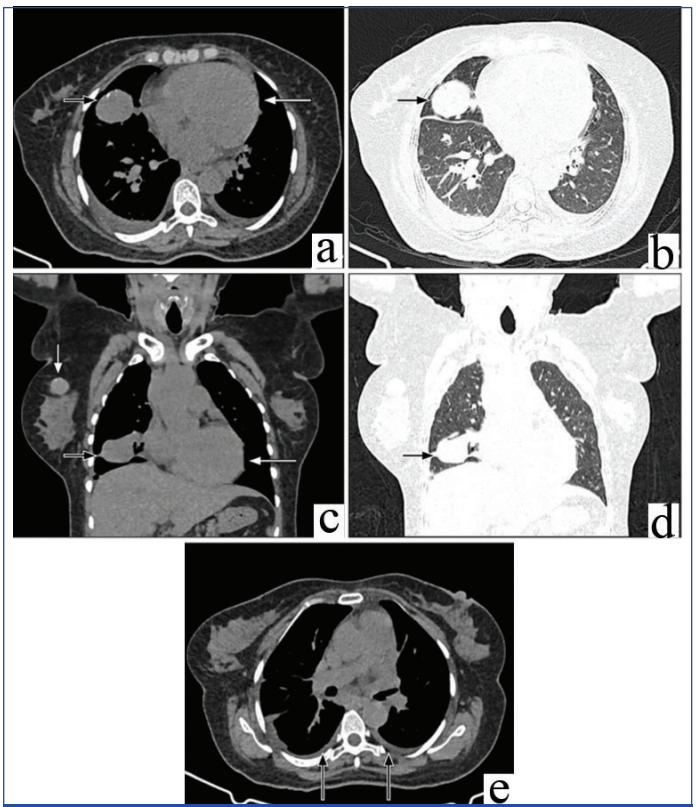


[Table/Fig-1]: ECG shows sinus rhythm with Q waves in the anterior leads (short black arrow).

shows a focal, macrolobulated, fairly defined radiopacity in the right mid- and lower lung fields. Associated with mild cardiomegaly [Table/Fig-2]. Plain CT chest shows a parenchymal and sub-pleural, focal, multi-lobulated soft-tissue dense mass lesion in the medial and lateral segments of the right middle lobe of size ~42×32×41 mm (antero-posterior×transverse×cranio-caudal), volume ~27 cc, with central/eccentric few specks of tiny calcifications (no necrosis, haemorrhage, cystic changes, cavity formation, or fat component), and peripheral smooth to lobulated margins associated with a few pleural tags/tails and no perifocal perilesional groundglass opacities. Mass lesion extending medially abutting the pericardium and laterally abutting the costal pleura. Associated with mild cardiomegaly. Bilateral minimal pleural effusion (R>L) causing passive collapse of the basal segments of the bilateral lower lobes [Table/Fig-3a-e]. Incidental focal soft-tissue dense lesion (likely benign neoplastic aetiology-fibroadenoma) in the upper-inner quadrant of the right breast of size ~20×11×20 mm (anteroposterior×transverse ×cranio-caudal), central/eccentric focal speck of tiny calcification, peripheral smooth margin. [Table/Fig-3c].

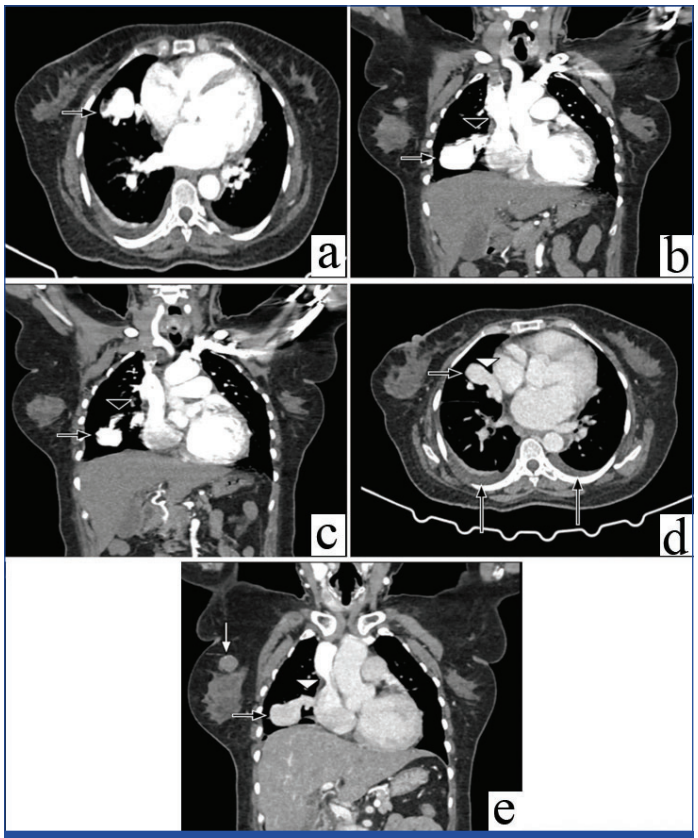


[Table/Fig-2]: Frontal chest radiograph demonstrating a focal lobulated radiopacity in the right mid and lower lung zones (black arrow) with mild cardiomegaly (white arrow).



[Table/Fig-3]: Multidetector computed tomography (MDCT) chest: (a) Mediastinal axial image showing pleural abutment of the lesion (black arrow) and cardiomegaly (white arrow); (b) Lung axial image showing pericardial abutment of the lesion (black arrow); (c) Coronal mediastinal image showing pleural abutment (black arrow), cardiomegaly (long white arrow), and incidental right breast lesion (short white arrow); (d) Coronal lung image showing pleural extension of the lesion (black arrow); (e) Axial mediastinal image showing minimal bilateral pleural effusion with basal atelectasis (black arrow).

Computed Tomography Angiography (CTA) of the chest shows a severe/intense homogeneous enhancing vascular mass lesion in the medial and lateral segments of the right middle lobe (enhancement identical to the pulmonary arterial attenuation with no intraluminal filling defect). Two distinct high-flow feeding arteries were identified arising from the medial and lateral segmental branches of the right middle lobar pulmonary artery (of size maximum 5 mm and 5 mm in diameter, respectively), with low resistance venous drainage through a single large right accessory middle lobar pulmonary vein (of size maximum 15 mm in diameter) draining into the left atrium [Table/Fig-4a-e].



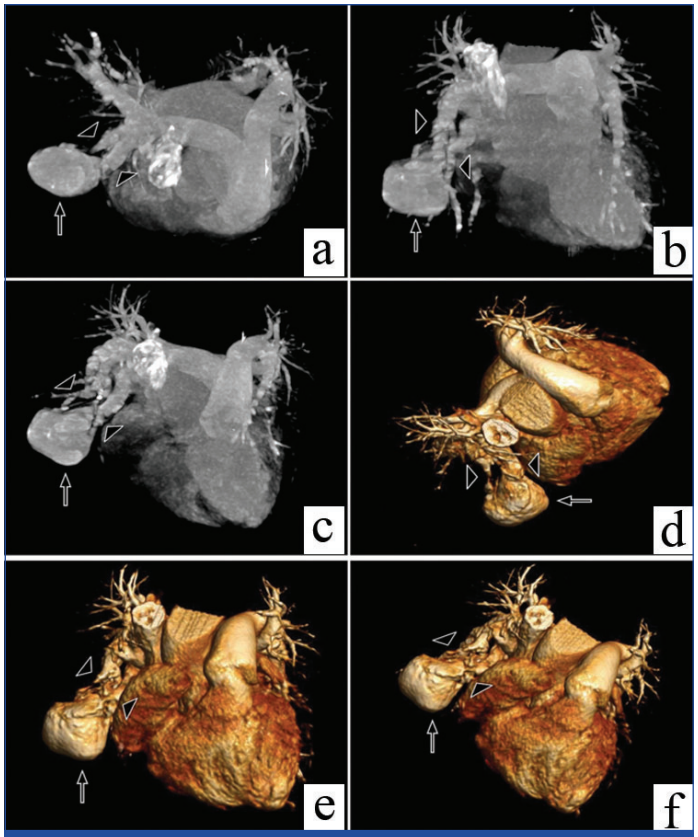
[Table/Fig-4]: Multidetector Computed Tomography (MDCT) Pulmonary Angiography (PA): (a) Axial arterial-phase image showing a homogeneously enhancing vascular mass lesion in the medial and lateral segments of the right middle lobe (short black arrow); (b, c) Coronal reformatted arterial-phase images demonstrating two high-flow feeding arteries arising from the medial and lateral segmental branches of the right middle lobar pulmonary artery (black arrowhead). Short black arrow represents the homogeneously enhancing vascular mass lesion; (d) Axial venous-phase image showing persistent homogeneous enhancement of the lesion with no intraluminal filling defect (short black arrow). A single large draining accessory middle lobar pulmonary vein drains into the left atrium (white arrowhead). Minimal bilateral pleural effusion with basal atelectasis (long black arrows); (e) Coronal venous-phase image demonstrating a single large draining accessory middle lobar pulmonary vein draining into the left atrium (white arrowhead). Incidental well-defined homogeneously dense soft-tissue lesion with tiny calcification in the upper-inner quadrant of the right breast, likely fibroadenoma (short white arrow). Short black arrow represents the persistent homogeneous enhancement of the vascular mass lesion.

The imaging findings were consistent with a complex aneurysmal PAVM, characterised by multiple feeding segmental pulmonary arteries, an aneurysmal vascular sac, and a draining accessory pulmonary vein communicating with the left atrium, in accordance with established angioarchitectural classification criteria for complex PAVMs [1].

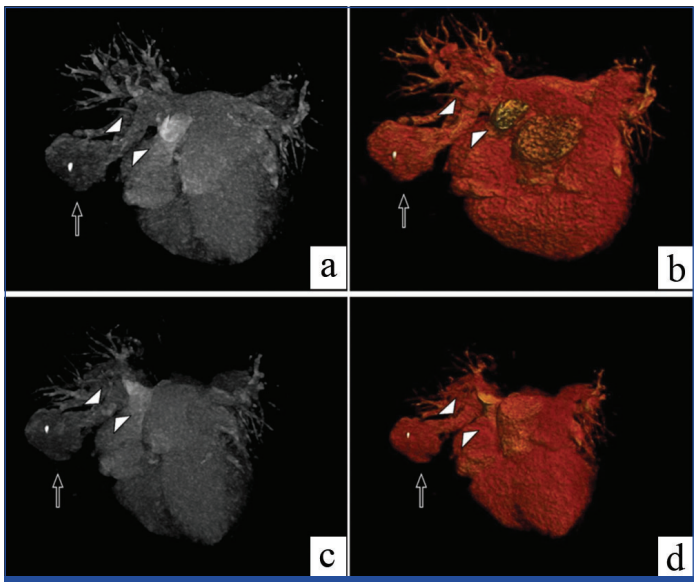
Conventional/Catheter Digital Subtraction Angiography (DSA): Selective right pulmonary arteriography reveals a direct communication between the branches of the right middle lobar pulmonary artery (medial and lateral segmental pulmonary arteries) and a large aneurysmal sac measuring approximately 38×30 mm, with subsequent venous outflow through a single large accessory right middle lobar pulmonary vein that has a diameter of about 15 mm, which drains into the left atrium [Table/Fig-5-7].

The slight difference in aneurysmal sac dimensions between CT and DSA is attributable to differences in imaging technique, measurement planes, and partial volume averaging. CT measurements were obtained in three orthogonal planes, whereas DSA measurements were derived from two-dimensional projection images.

The patient was treated conservatively during admission with oxygen supplementation (2-4 L/min via nasal prongs), anti-platelets (Clopidogrel 75 mg once daily), statins (atorvastatin 40 mg once daily), bronchodilator therapy (etofylline 115 mg plus theophylline 35 mg once daily), antihypertensive agents (enalapril 2.5 mg twice daily), diuretics (spironolactone 25 mg once daily and torsemide 5

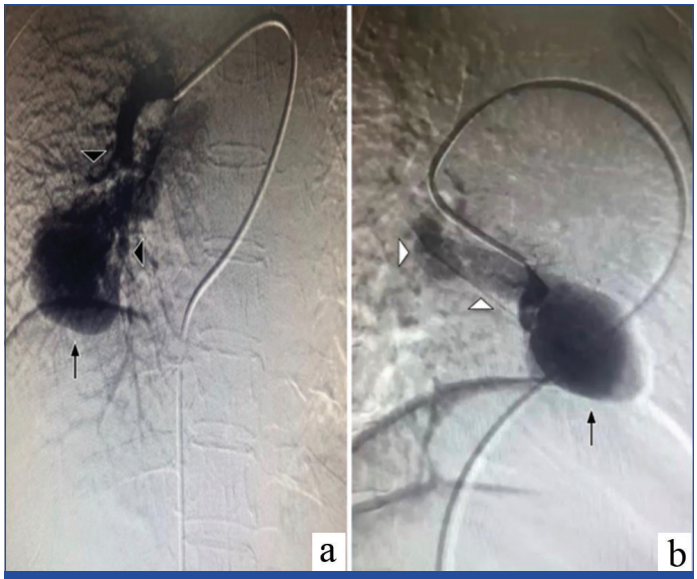


[Table/Fig-5]: Multidetector Computed Tomography (MDCT) pulmonary angiography (PA)- three-dimensional (3D) Maximum Intensity Projection (MIP) and Three-dimensional (3D) volume-rendered (VR) images - Arterial phase shows: (a) Axial MIP, (b) Sagittal MIP and (c) Coronal MIP arterial-phase images (d) Axial VR, (e) Sagittal VR and (f) Coronal VR showing a homogeneously enhancing vascular mass lesion in the medial and lateral segments of the right middle lobe (short black arrows). (a) Axial MIP, (b) Sagittal MIP and (c) Coronal MIP arterial-phase images (d) Axial VR, (e) Sagittal VR and (f) Coronal VR images demonstrating two high-flow feeding arteries arising from the medial and lateral segmental branches of the right middle lobar pulmonary artery (black arrowheads).



[Table/Fig-6]: Multidetector Computed Tomography (MDCT) pulmonary angiography (PA) - Three-Dimensional (3D) Maximum Intensity Projection (MIP) and 3D Volume-Rendered (VR) images: (a,b) Coronal 3D MIP and VR images showing a homogeneously enhancing vascular mass lesion in the medial and lateral segments of the right middle lobe (short black arrow); (c,d) Coronal-oblique 3D MIP and VR images demonstrating a single large draining accessory middle lobar pulmonary vein draining into the left atrium (white arrowheads).

mg once daily) and antidiabetic medications (metformin 500 mg twice daily) throughout the hospital stay for approximately nine days. The patient improved clinically, with a resting peripheral SpO_2 of approximately 94% on room air at discharge (day 9), and was advised to avoid strenuous exertion, continue medical management for associated co-morbidities, and remain on regular clinical follow-up.



[Table/Fig-7]: Conventional/catheter Digital Subtraction Angiography (DSA): Selective right pulmonary arteriography: (a) Arterial-phase image showing feeding arteries arising from the medial and lateral segmental branches of the right middle lobar pulmonary artery (black arrowheads) communicating with a large aneurysmal sac (short black arrow); (b) Venous-phase image demonstrating drainage through a single large accessory right middle lobar pulmonary vein into the left atrium (white arrowheads), draining a large aneurysmal sac (short black arrow).

DISCUSSION

The PAVMs are rare vascular anomalies due to abnormal direct communications between pulmonary arteries and veins, producing right to left shunting that may lead to hypoxaemia and paradoxical embolic complications [1]. PAVMs may be single or multiple, unilateral or bilateral, and simple or complex. Most PAVMs are hereditary and occur in hereditary haemorrhagic telangiectasia, an autosomal dominant vascular disorder, and screening for PAVM is indicated in this subgroup. PAVMs may also be idiopathic, occur as a result of trauma and infection, or be secondary to hepatopulmonary syndrome and bidirectional cavopulmonary shunting [1].

The present patient had no history of recurrent epistaxis, mucocutaneous telangiectasia, gastrointestinal bleeding, or family history suggestive of Hereditary Haemorrhagic Telangiectasia (HHT), favouring a sporadic PAVM. Clinical presentation varies from incidental asymptomatic lesions to dyspnoea, cyanosis, haemoptysis, and neurological manifestations secondary to paradoxical embolisation [2].

Radiologically, chest radiographs may demonstrate a well-defined nodular or lobulated opacity. CT pulmonary angiography played a crucial role in diagnosis by clearly demonstrating the characteristic feeding artery - aneurysmal sac - draining vein configuration [1].

PAVM angioarchitecture is classified as simple or complex based on the segmental pulmonary artery anatomy, which is important for planning endovascular interventions [3]. Simple PAVMs are supplied by a single segmental pulmonary artery. The single segmental pulmonary artery will often branch distally into one to three subsegmental branches, all supplying the PAVM [3]. Complex PAVMs are supplied by two or more segmental pulmonary arteries [3]. The present lesion was categorised as a complex PAVM because of dual segmental arterial feeders.

Complex PAVMs require occlusion of all feeding arteries to achieve complete treatment and reduce the risk of persistence or recurrence. Balloon embolotherapy provided an effective treatment for PAVMs regardless of anatomic type [4].

The 2011 HHT consensus guidelines recommended embolisation for PAVMs with feeding artery diameter ≥3 mm, while noting that treatment of lesions as small as 2 mm may be appropriate in selected cases [5]. Serious complications such as ischaemic stroke and brain abscess are more commonly associated with PAVMs having feeding arteries ≥3 mm, although smaller lesions may also rarely cause

such events. Current practice therefore supports treatment of all symptomatic PAVMs and asymptomatic lesions with feeding artery diameter ≥2 mm [3,5]. In addition, embolisation of all angiographically visible PAVMs during the initial procedure has been shown to reduce the risk of reintervention and ischaemic stroke [3].

CONCLUSION(S)

The present case demonstrates an uncommon dual-feeder complex aneurysmal PAVM with accessory pulmonary venous drainage to the left atrium, initially mimicking a pulmonary mass on non contrast CT. Diagnosis was confirmed by contrast-enhanced Multidetector CT Angiography (MDCTA) and catheter angiography, which clearly showed the typical feeding artery, aneurysmal sac, and draining vein architecture, thereby differentiating PAVMs from pulmonary nodules, neoplasms, and other vascular mimics.

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