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Silent giant: 80 mm pulmonary artery aneurysm

Un gigante silencioso: aneurisma de arteria pulmonar de 80 mm

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An 84-year-old male with chronic myelodysplastic syndrome on low-dose prednisolone and hypertension presented with methicillin-resistant *Staphylococcus aureus* native tricuspid valve endocarditis. Initial evaluation included a chest X-ray showing hilar enlargement, prompting further imaging (Fig. 1). A computed tomography showed a giant pulmonary artery aneurysm (PAA) measuring 80 mm (Fig. 2). Upon review of prior imaging, it was found that this aneurysm remained stable since its incidental discovery, 3 years earlier, during evaluation for atypical chest pain (Fig. 3). At that time, transthoracic echocardiography (TTE) demonstrated mild right ventricular dysfunction, severe pulmonary regurgitation, and an estimated right ventricular systolic pressure (RVSP) of 71.6 mmHg. Unfortunately, the patient was lost to follow-up, and no further investigations were pursued to clarify the aneurysm's etiology.

During the current admission, repeat TTE demonstrated stable findings, including mild biventricular dysfunction, RVSP of 61 mmHg, severe pulmonary regurgitation, and a new tricuspid valve vegetation (0.5 × 0.6 cm) with moderate tricuspid regurgitation (Fig. 4). In the context of a myelodysplastic syndrome with poor prognostic features, further diagnostic workup was not undertaken. Surgical intervention was deemed too high-risk due to the patient's frailty and multiple comorbidities. He was discharged on intravenous antibiotic therapy, with conservative monitoring of the aneurysm and a palliative approach for his hematologic disease.

PAA is typically defined as a dilation exceeding 1.5 times the upper limit of normal[1]. It is a rare and often asymptomatic condition that can result from various causes, including congenital heart disease, syphilis, atherosclerosis, trauma, pulmonary hypertension, or, in some cases, idiopathic mechanisms[2]. In idiopathic cases, proposed theories include congenital weakness of the pulmonary artery wall, abnormal embryologic development of the truncus arteriosus, or cystic medial degeneration[3]. In our case, the long-term stability of the aneurysm, together with the absence of compressive symptoms or rupture, supports an idiopathic origin.

Idiopathic PAA is generally considered a benign condition, with most patients surviving into their sixth or seventh decade.[3] However, progressive enlargement may occur, leading to complications such as rupture, dissection, or compression of adjacent structures[3]. Reported mechanisms of sudden death include compression of the left main coronary artery, pulmonary artery rupture, or dissection resulting in cardiac tamponade[3]. In our case, the aneurysm did not compromise the coronary arteries, as demonstrated in figure 2D and E.

Regular follow-up is recommended to monitor for aneurysm progression and the development or worsening of pulmonary regurgitation[3]. Although surgical intervention is generally considered for aneurysms exceeding 5.5 cm in diameter, our case highlights the potential for long-term stability in large idiopathic PAAs in the absence of symptoms or complications[4].

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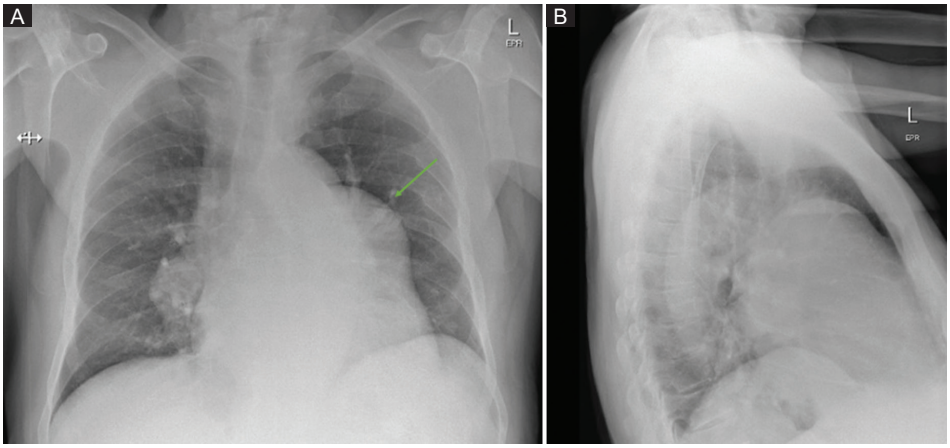

Figure 1. A and **B:** frontal and lateral chest radiographs demonstrate significant bilateral hilar enlargement, more prominent on the left (arrow).

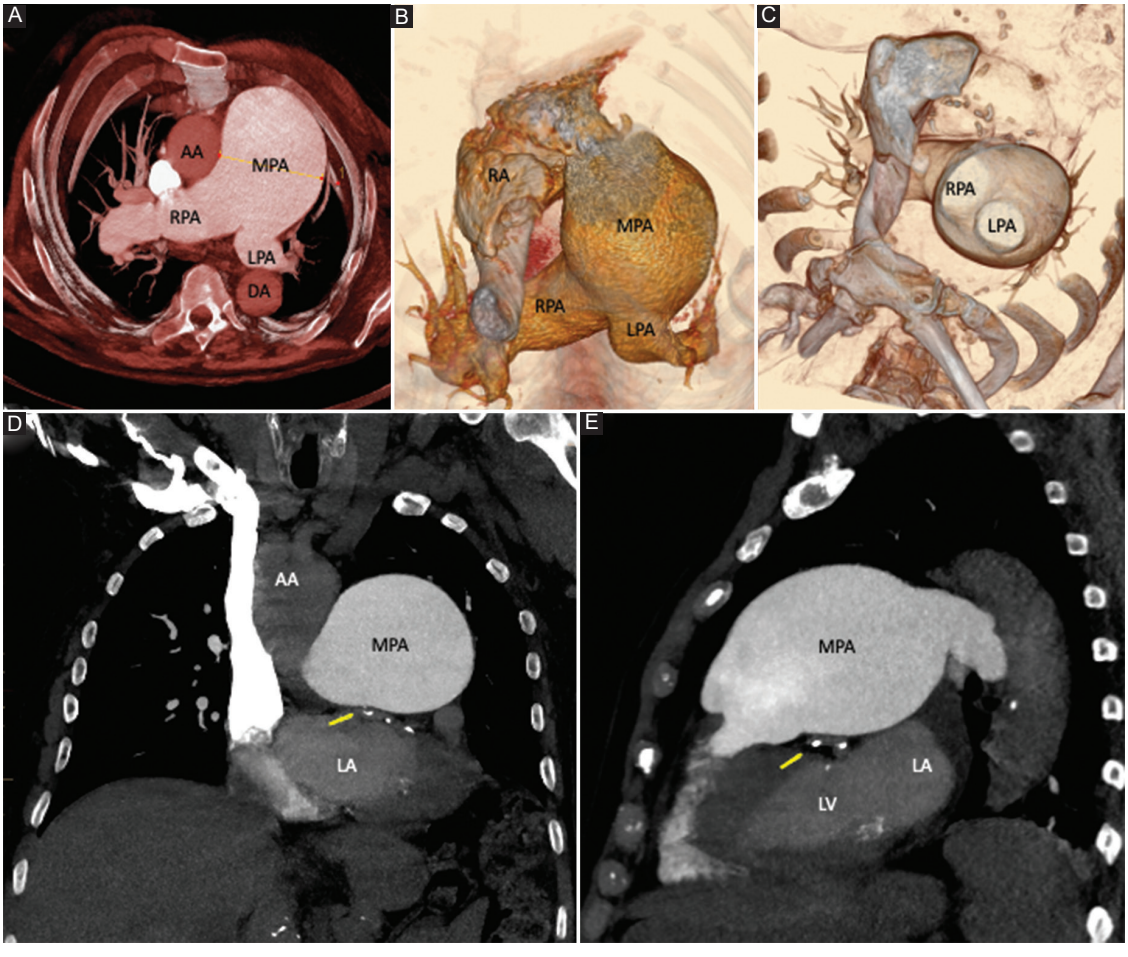

Figure 2. A-C: axial CT and three-dimensional reconstructions of the chest CT showing the main pulmonary artery aneurysm. **D:** coronal and sagittal **E:** MIP CT images demonstrate the pulmonary artery aneurysm in 2024 and its close relationship with the moderately calcified left coronary artery (yellow arrow). CT: computed tomography; AA: ascending aorta; DA: descending aorta; LA: left atrium; LV: left ventricle; MIP: maximum intensity projection; MPA: main pulmonary artery; RPA: right pulmonary artery; LPA: left pulmonary artery; RA: right atrium.

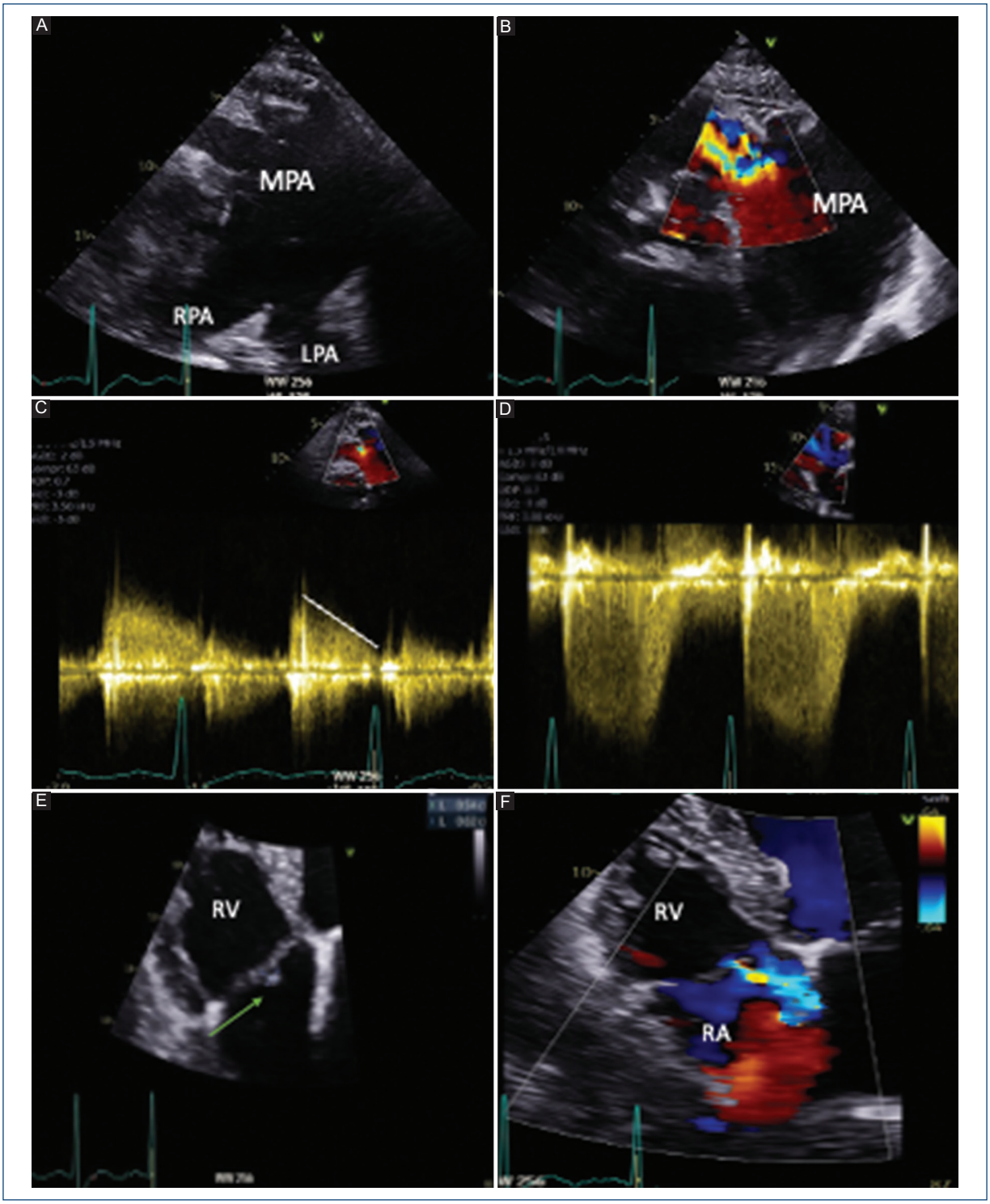

Figure 4. A: TTE 2D imaging of the main pulmonary artery and branch pulmonary arteries in the parasternal short-axis view. **B:** color Doppler of the pulmonary valve in the parasternal short-axis view, demonstrating severe pulmonary regurgitation. **C:** continuous wave (CW) Doppler of pulmonary valve regurgitation with pressure half-time (PHT, white line) demonstrating rapid flow deceleration during diastole, indicating severe regurgitation. **D:** CW Doppler of the tricuspid valve in the parasternal short-axis view, showing a maximum velocity of 3.3 m/s and an estimated right ventricular systolic pressure of 60 mmHg. **E:** apical 4-chamber view (right ventricular-focused view) showing an independently mobile echodensity on the atrial side of the tricuspid valve, measuring 0.5 mm × 0.6 mm (yellow arrow). **F:** color Doppler interrogation of the tricuspid valve in the apical 4-chamber view, highlighting moderate regurgitation. AA: ascending aorta; DA: descending aorta; MPA: main pulmonary artery; RPA: right pulmonary artery; LPA: left pulmonary artery; RA: right atrium; RV: right ventricle.

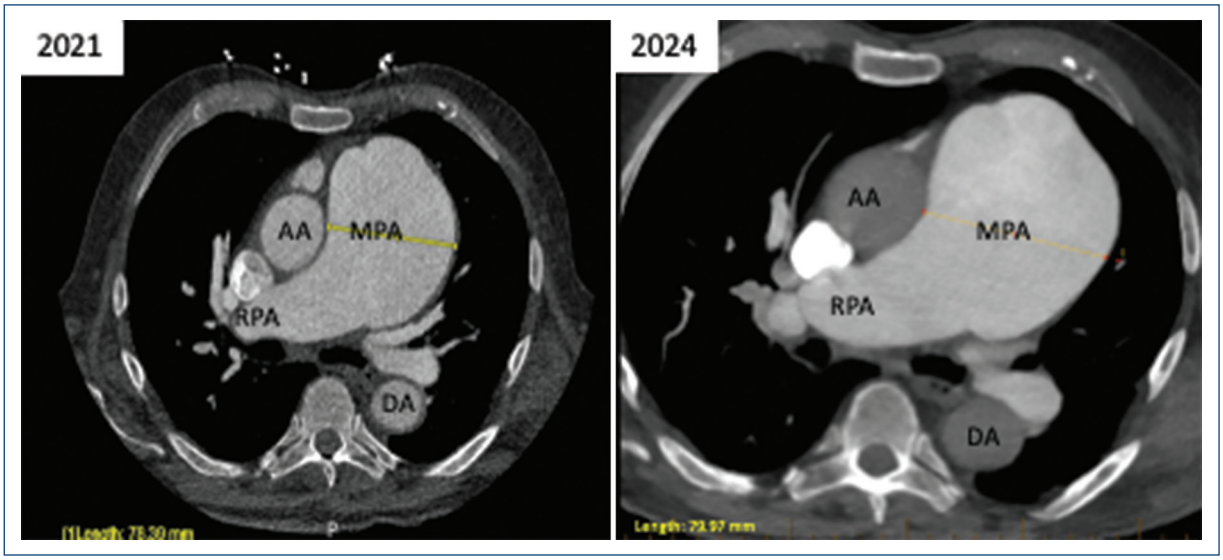

Figure 3. Axial CT images showing the maximum dimension of the pulmonary artery aneurysm in 2021 and 2024, demonstrating overall stability since 2021. CT: computed tomography; AA: ascending aorta; DA: descending aorta; MPA: main pulmonary artery; RPA: right pulmonary artery.

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Conflicts of interest

The authors declare no conflicts of interest.

Ethical considerations

Protection of humans and animals. The authors declare that no experiments involving humans or animals were conducted for this research.

Confidentiality, informed consent, and ethical approval. The authors have obtained approval from the Ethics Committee for the analysis of routinely collected and anonymized clinical data; therefore, individual informed consent was not required. Relevant ethical recommendations have been followed.

Declaration on the use of artificial intelligence. The authors declare that no generative artificial intelligence was used in the writing or creation of the content of this manuscript.

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