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An unknown wide persistent ductus arteriosus debuting with atrial fibrillation in older adult: a case report

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Abstract

Background The persistently patent ductus arteriosus represents a well-known common congenital heart defect; it is uncommon in adult patients, and in any case, it debuts with atrial fibrillation.

Case presentation A 75-year-old woman suffering from persistent ductus arteriosus (PDA) was admitted to the cardiology department because of atrial fibrillation, dyspnea and exercise intolerance. A PDA was detected on echocardiography and globally assessed through ECG-gated CT angiography.

Conclusions Patent ductus arteriosus is an uncommon clinical finding in adulthood, and atrial fibrillation, as a consequence of chronic, progressive left atrial enlargement, may be the initial symptom. We describe the ECG-gated CT angiography imaging features of unknown patent ductus arteriosus in an elderly patient who debuted with atrial fibrillation.

Keywords Ductus arteriosus, Atrial fibrillation, Ductus Botalli, Case report

Background

The persistently patent ductus arteriosus represents a well-known common congenital heart defect; Claudius Galen (130–200 C.E.) was the first to describe the ductus arteriosus in fetal cadavers. Anyway, the description of the ductus arteriosus is attributed to Vesalius' pupil Giulio Cesare Aranzio in 1564, who only claimed to have delved into Galen's report [1].

PDA is uncommon in adult patients, and in any case, it debuts with atrial fibrillation.

Isolated PDA accounts for 10–12% of all congenital cardiac abnormalities [2].

In adult patients, the clinical spectrum of a PDA can range from silent type to clinical manifestations such as congestive heart failure, pulmonary hypertension, volume overload condition, endocarditis, atrial fibrillation, or recurrent pneumonia [3]. PDA adult occurrence is unusual, albeit some patients survive with a minor disability, reaching considerable ages [4]. PDA clinical consequences depend mainly on the morphology of the ductus [5, 6]. The ductal morphology and dimensions have significant implications in managing PDA for both percutaneous closure devices and surgical closure approaches. Determination of morphological features of the duct, such as size, degree of calcification, and morphologic classification, is obtainable scrupulously through ECG-gated CT angiography.

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Case presentation

A 75-year-old patient, with no relevant medical history, was admitted to the Cardiology Department with heart failure symptoms including dyspnea and exercise intolerance.

ECG revealed atrial fibrillation and a PDA was discovered with echocardiography; high systolic pulmonary artery pressure (sPAP) was found, with accessory echo signs suggesting a high probability of pulmonary hypertension. Right heart catheterization confirmed this finding, with a measured mean pulmonary artery pressure (mPAP) of 45 mmHg.

Due to the ability to evaluate anatomical features and the presence of calcifications (PDA planimetry, length, minimum diameter and subtype) as a roadmap before considering closure, ECG-gated cardiac CT angiography was performed (Fig. 1). CT reconstructions showed a patent ductus arteriosus type E according with angiographic classification of Krichencko et al. [1] (Fig. 2). There was also left atrial and left ventricle enlargement with modest dilatation of the main pulmonary artery (Fig. 3). In the therapeutic management, considering the patient symptoms, PDA transcatheter occlusion was contemplated based on current indication. This aims to prevent bacterial endocarditis, irreversible pulmonary vascular disease and to correct volume overload and its possible consequences. Unfortunately, the patient refused the proposed treatment and was referred for drug therapy with Bosentan.

Patent ductus arteriosus (PDA) is an infrequent clinical condition in adulthood, even though cases of PAD in elderly or very elderly patients are reported in the literature [7].

PDA has an estimated annual mortality rate of 1.8% in the aged, so it needs to be treated to avoid spreading endocarditis, pulmonary vascular disease, and congestive heart failure with recurring atrial flutter and/or fibrillation. Furthermore, atrial fibrillation and atrial flutter are well-known complications of congestive heart failure caused by chronic volume overload generated by PDA [8].

Anilkumar [6] discussed the pathophysiology of non-silent PAD and the consequences without proper treatment. Indeed, the natural history and clinical manifestations of PDA in patients with no associated congenital heart defects are primarily linked to the quantity of shunted blood and its consequences on the pulmonary artery and the left side of the heart. The risks associated with the shunt's quantification in moderate and large PDAs include the development of gradual arterial modifications in the pulmonary arteries, which can lead to pulmonary hypertension, as well as dilatation of the left atrium and left ventricle, causing congestive heart failure. If pulmonary overcirculation is not corrected, changes in the pulmonary circulation occur, which can result in an irreversible dramatic rise of pulmonary arterial pressure. When pulmonary vascular resistance overcomes the systemic one (chronic pulmonary arterial hypertension), the shunt reverses with a new direction from right to left,

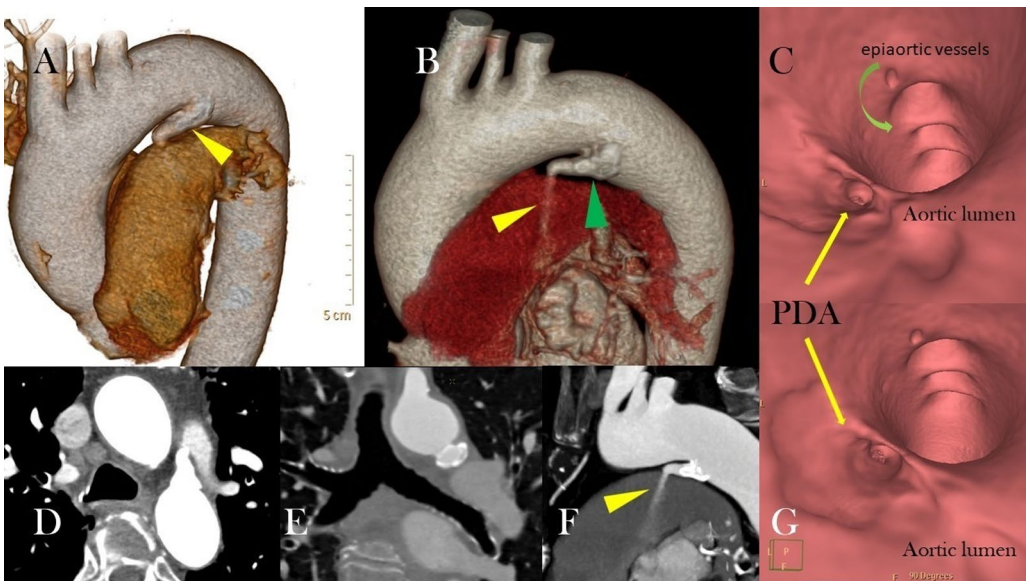

Fig. 1 ECG cardiac CT angiography with volume-rendered 3D reconstruction (**A** and **B**) shows the aorta and pulmonary arteries with PDA (yellow arrowhead in **A** and green arrowhead in **B**). "Positive jet" of enhanced blood flowing from the aorta to the low pulmonary artery via the PDA (yellow arrowhead in **B** and **F**). On axial (**A**), coronal (**E**) and sagittal (**F**) multiplanar reformation from retrospectively ECG-gated CT angiography showed PDA and ductal calcification in E and F. CT virtual angioscopic image shows aortic lumen, the PDA and its origin (thin yellow arrowcurved in **C** and **G**) and the origin of epiaortic vessels (curved green arrow in **C**)

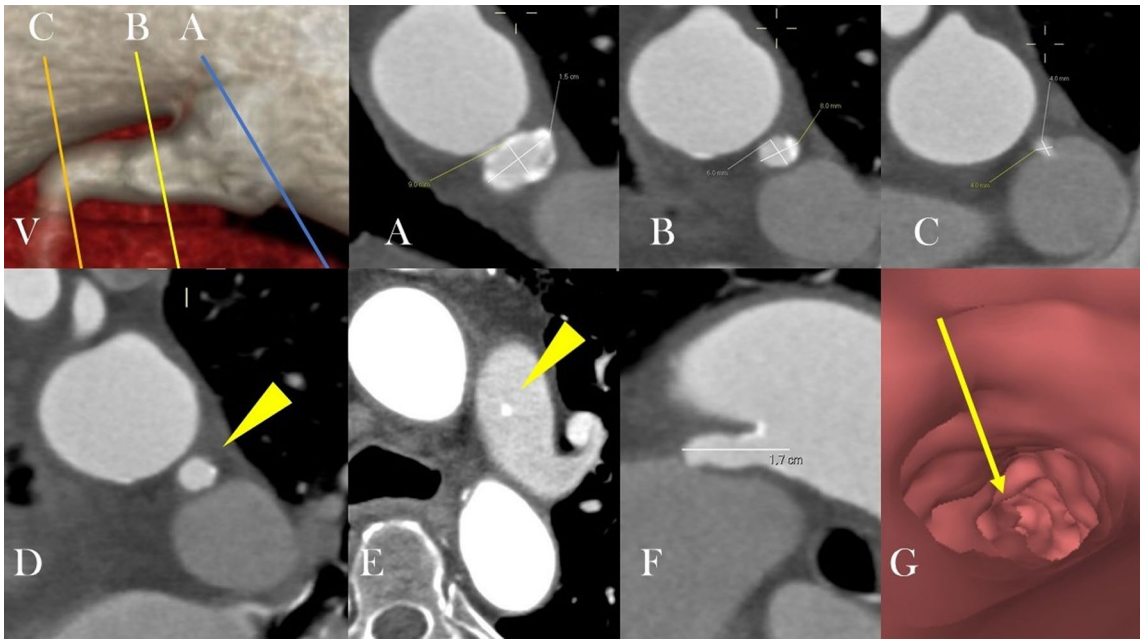

Fig. 2 Calcified PDA. V: volume-rendered three-dimensional image shows PDA. **A**, **B** and **C** show multiplanar reformations (MPR) where the diameters of the duct were measured, respectively, at its aortic origin, at its middle third and distal third, at the entrance to the left pulmonary artery. **D**: The MPR image in the coronal plane shows the PDA between the aorta and the left main pulmonary artery, assuming the appearance of a small satellite between two planets (satellite sign, yellow arrowhead). **E**: axial view reveals a positive jet of contrast from the aorta to the less-opacified pulmonary artery (spot sign, yellow arrowhead) **F**: MPR image of PDA in the sagittal plane as a tubular structure in its entire length. **G**: the virtual angioscopic CT image does not show a linear valve-like structure at the pulmonic end of the PDA

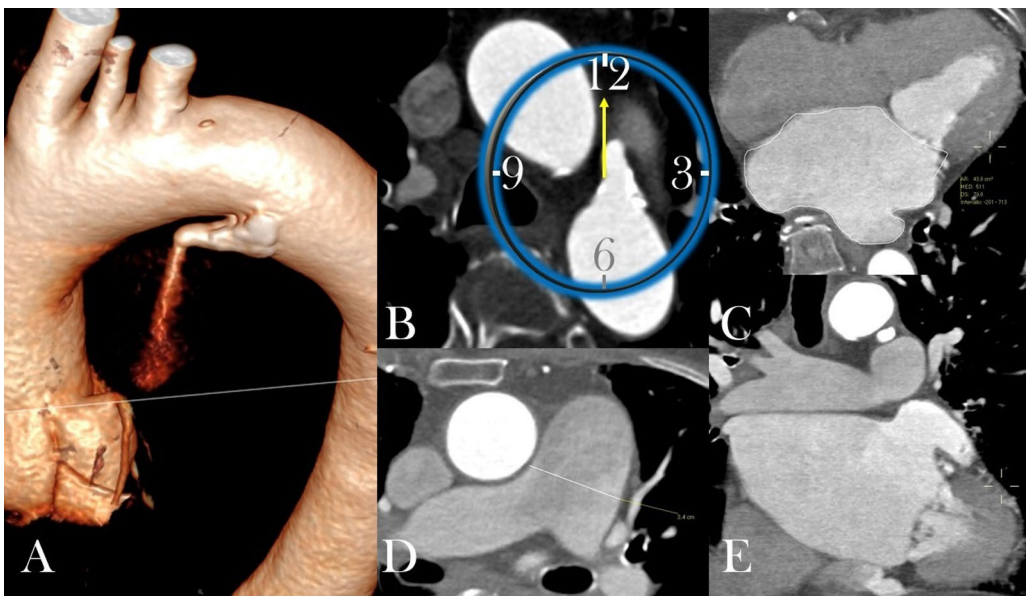

Fig. 3 **A**: Volume-rendered three-dimensional CT reconstruction shows PDA. **B**: Axial CT aortic angiography reveals PDA near the twelve o'clock position relative to the descending thoracic aorta just distal to the left subclavian artery. **C** and **E**: Axial and coronal planes on CT aortic angiography reveal marked enlargement of the left atrium due to left heart volume overload. **D**: Axial CT aortic angiography reveals enlargement of the main trunk of the pulmonary artery (pulmonary hypertension); the main pulmonary artery diameter was 34 mm.

leading to the fearful complication of Eisenmenger's syndrome [6].

All patients with no silent PDA can be considered for closure [6].

The presence or absence of left atrium or left ventricle enlargement indirectly estimates the shunt's degree, and it guides patient management; in particular, considerable shunt volume requires definitive treatment to remove PDA before irreversible pulmonary hypertension appears [6].

Percutaneous PDA closure is preferred in adult patients because surgery carries a higher perioperative risk due to ductal friability, calcification and associated conditions such as coronary artery disease or aortic atherosclerosis.

ECG-gated cardiac CT angiography is a noninvasive method for assessing patent ductus arteriosus in adults, providing detailed anatomic information [9, 10].

Although routine CT or MRI evaluation is not required, in older patients, CT proves very helpful in showing the calcifications and accurately describing the three-dimensional anatomy of the large PDA, which is valuable knowledge in planning therapeutic options.

The development of pulmonary hypertension, atrial enlargement with remodeling, and atrial fibrillation, in our case, can all be explained by the significant left-to-right shunt.

In elderly patients, atrial fibrillation could be caused by a large unknown PDA; this scenario, even though rare, should not be overlooked.

Conclusions

Our article describes a case of association between atrial fibrillation and an unknown significant PDA in an older adult woman, even though PDA has been the topic of extensive discussion in the literature and has attracted the attention of the scientific community for centuries.

This case emphasizes how important it is to accurately evaluate the hemodynamics and morphology of an PDA in older patients through appropriate imaging, as well as the significant clinical consequences that can result from it. Improving results requires prompt identification and intervention.

Additionally, we hypothesize that, ideally, our brief report serves as a cultural benchmark for radiologists and cardiologists who conduct or use cardiovascular imaging, summarizing the experience gained.

Abbreviations

PDA	Persistent ductus arteriosus
CT	Computed tomography
PAP	Pulmonary artery pressure
MRI	Magnetic resonance imaging

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None.

Author contributions
AP was involved in the conceptualization, supervision, investigation, and writing—original draft. AT contributed to the conceptualization and writing—original draft. ARC assisted in the investigation and supervision. BFPA, SF and PP performed the investigation. SV was involved in the conceptualization and supervision. All authors read and approved the final manuscript.

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Availability of data and material
The data that support the findings of this study are available from the corresponding author, [AT], upon reasonable request.

Declarations

Ethics approval and consent to participate
Not applicable.

Consent for publication
The patient gave consent.

Competing interests
None.

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