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CASE REPORT
Heart failure

A case report on the successful interventional treatment of a rare cause of left heart failure in a 69-year-old woman

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Background Dyspnoea is very common in elderly patients and can be caused by a variety of different diseases. However, the initial diagnosis of patent ductus arteriosus (PDA) as a cause of left heart failure is very rare in this patient population.

Case summary A 69-year-old physically active woman with known hypertension presented with worsening exertional dyspnoea. Echocardiography showed a dilated left ventricle with moderately reduced left ventricular ejection fraction, and evidence for PDA. The PDA was confirmed by computed tomography angiography and successfully closed by implantation of an Amplatzer PDA occluder II 06-06 mm. As a result, the heart failure symptoms receded completely.

Discussion Congenital heart diseases should be considered as heart failure causes even in older adults. In addition to the standard medical therapy, there may be effective interventional treatment options to reverse the symptoms of heart failure in such patients.

Keywords Case report • Heart failure • Patent ductus arteriosus • Closure • Older adults • Amplatzer duct occluder II

Learning points

- Congenital heart diseases should be considered as heart failure causes even in older adults, especially when common causes can be excluded.
- The patent ductus arteriosus (PDA) may be overlooked in adults if the diastolic part of its characteristic murmur is difficult to auscultate or as long as the PDA is not easily visible in transthoracic echocardiography.
- In case of doubt, computed tomography angiography confirms the diagnosis. It is also very helpful for planning interventional PDA closure in adults.

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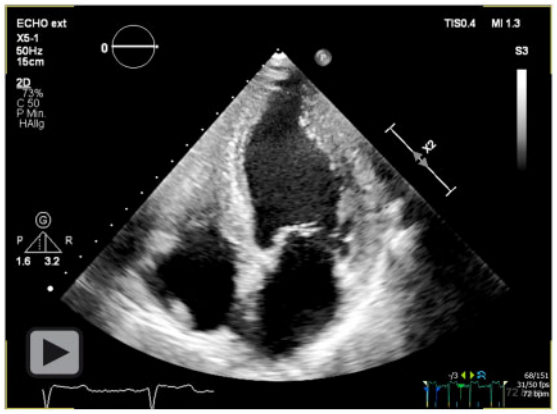
Introduction

Dyspnoea is very common in elderly patients and can be caused by a variety of different conditions, mostly lung and heart diseases. The initial diagnosis of patent ductus arteriosus (PDA) as a cause of left heart failure is very rare in this patient population and depends on a high clinical awareness of congenital heart disease in older adults. The PDA is a vascular structure that connects the proximal descending aorta to the roof of the main pulmonary artery near the origin of the left branch pulmonary artery.[1] It is the third most common congenital anomaly in which the arterial duct, which normally closes spontaneously after birth within 24–48 h in full-term infants, remains permanently open.[1] Over the years, the associated left-to-right shunt leads to volume overload of the left ventricle and heart failure.[1–3] The time until heart failure symptoms develop depends on the magnitude of shunting. The flow depends on the resistance of the PDA, i.e. length, narrowest diameter, and configuration.[1] The initial clinical manifestation and diagnosis of PDA in elderly people is very unusual. Here we report on a 69-year-old, physically active woman who presented with worsening exertional dyspnoea and PDA. The symptoms of heart failure were successfully reversed by an interventional PDA occlusion.



Video 1 Transthoracic echocardiography (four-chamber view) at initial diagnosis of patent ductus arteriosus. The left ventricle was enlarged (left ventricular end-diastolic diameter 63 mm) with a moderately reduced global systolic function (left ventricular ejection fraction 35%) without regional wall motion abnormalities. The right ventricular function was normal.

Timeline

Initial diagnosis of patent ductus arteriosus (PDA)	Outpatient with history of hypertension, referral for heart failure; PDA diagnosis in transthoracic echocardiography (TTE), left ventricular ejection fraction (LVEF) 35%
First admission	Diagnostic catherization: rule-out of significant coronary artery disease; borderline pulmonary hypertension; left-to-right shunt: pulmonary blood flow (Qp)/systemic blood flow (Qs) = 1.8; confirmation of PDA diagnosis (type A, conical form); computed tomography angiography for planning of interventional PDA closure
Second admission	Elective interventional PDA closure with Amplatzer duct occluder II, no residual shunt
5-month follow-up visit	Outpatient, no more symptoms of heart failure; TTE: no residual PDA shunt, LVEF unchanged
15-month follow-up visit	Outpatient, no symptoms of heart failure; TTE: no residual PDA shunt, LVEF slightly increased (40%), N-terminal pro-B-type natriuretic peptide normalized

Case presentation

A 69-year-old Caucasian woman was referred to our outpatient clinics because of worsening of dyspnoea during physical exercise (New York Heart Association functional class I–II). She reported that she had suffered from myocarditis 17 years before, but there were no medical reports available to confirm this. Cardiac magnetic resonance imaging was performed 8 years before and revealed dilated cardiomyopathy without active myocarditis (i.e. a slightly dilated left ventricle with a left ventricular ejection fraction of 39%). At the time of the examination, she had arterial hypertension, hyperlipidaemia, and diabetes (HbA1c 6%, on diet). Her cardiovascular medication consisted of ramipril 10 mg and simvastatin 40 mg once daily. A very soft systolic murmur was heard at the pulmonic valve auscultation point, otherwise, the cardiovascular examination was unremarkable. The ECG showed sinus rhythm with incomplete left bundle branch block with non-specific ST segment and T-wave changes. Transthoracic echocardiography showed a dilated left ventricle (left ventricular end-diastolic diameter 63 mm) with moderately reduced global systolic function (left ventricular ejection fraction 35%), normal right ventricular function (see *Video 1*), and a diagnosis of PDA was made based on a continuous, pulsatile and dynamic retrograde jet flow at main pulmonary artery (see *Figure 1*). The PDA was confirmed by computed tomography angiography (CTA) with three-dimensional reconstruction using volume rendering technique (see *Figure 2C*). The CTA showed a PDA of conical form (type A) with a length of 29 mm (see *Figure 2A*), a very small pulmonary ostial diameter of only about 1 mm, and an aortic ostial diameter of about 8 mm (see *Figure 2B*). Cardiac catheterization revealed no significant coronary artery disease, a significant extracardiac left-to-right shunt of 45% [1.87 L/min; pulmonary blood flow (Qp)/systemic blood flow (Qs) = 1.8], and borderline pulmonary hypertension [35/21/25 mmHg; pulmonary vascular resistance 270 dyne.s.cm^{-5} (3.4 Wood units); pulmonary vascular resistance index 426 dyne.s.cm^{-5}.m^{2}]. Therefore, in line with the 2020 European Society of Cardiology Guidelines for the management of adult congenital heart disease,[2] the PDA was closed with an Amplatzer duct occluder II 06-06 mm (Abbott, Vienna, Austria) (see *Figure 3*, *Videos 2* and *3*) with a transarterial approach.[4] Acetylsalicylic acid 100 mg once daily was added to the medication

after PDA occlusion for 6 months. Cardiac magnetic resonance imaging was not repeated before closure. After 5 months, the patient had fully recovered without signs of heart failure. She reported that she had regained physical exercise capacity, including unrestricted mountain hiking. Echocardiography showed no signs of residual flow in the duct, but left ventricular function did not improve significantly. The left ventricular end-diastolic diameter did not decrease either. There was a slight but not significant decrease in N-terminal pro-B-type natriuretic peptide (NT-proBNP: 1084 to 834 ng/L, delta within the biological variation[5]). After 15 months, the patient reported normal exercise capacity. Echocardiography showed no signs of residual flow in the duct, and the left ventricular ejection fraction improved slightly (40%). The NT-proBNP decreased to 154 ng/L (within the age-adjusted normal range).

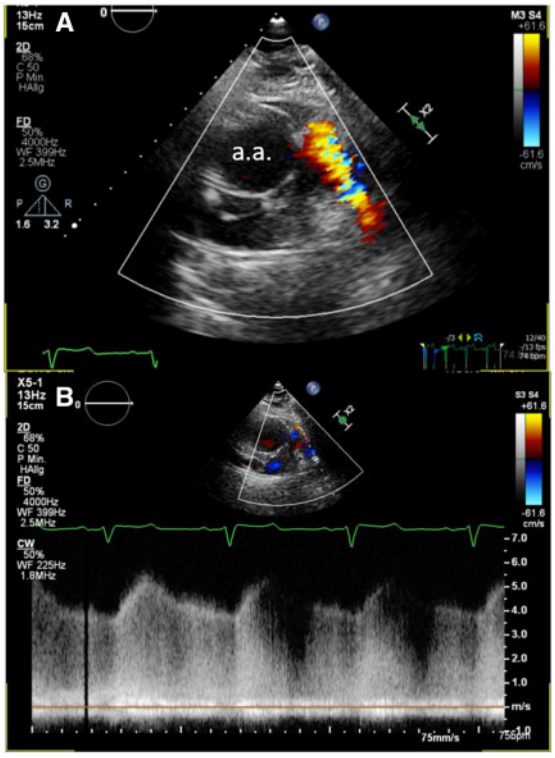


Figure 1 Parasternal short-axis view of transthoracic echocardiography. Top (*A*): Colour doppler imaging demonstrated a retrograde jet flow at main pulmonary artery. Bottom (*B*) Continuous wave Doppler demonstrated a continuous, pulsatile, and dynamic left-to-right ductal flow. a.a., ascending aorta.

Discussion

The primary diagnosis of a PDA in elderly heart failure patients is very rare these days. Its diagnosis requires a high clinical awareness of congenital heart disease, which is rare in this patient population. In women, PDA is about twice as common as in men.[1–3] Therefore, it is particularly important in women to exclude PDA in case of left ventricular dilatation that cannot be attributed to other causes. Usually, PDA patients are diagnosed by work-up of its incidentally discovered, typical murmur or by echocardiography performed for other reasons before symptoms develop. However, the diastolic part of its characteristic murmur may be missed in adults. The transthoracic echocardiographic signs of left ventricular volume overload are also nonspecific as long as the PDA is not easily visible. However, a retrograde jet flow at the main pulmonary artery, as seen in our patient, should raise suspicion of PDA. In case of doubt in echocardiography CTA allows a definitive diagnosis. Computed tomography angiography allows an excellent reconstruction of its configuration and an exact sizing of its dimensions, which is essential for planning an interventional PDA closure. The PDA can persist in a wide variety of sizes and shapes, i.e. conical, window-shaped, tubular, complex, and elongated.[1] The shape of this PDA with its small pulmonary ostium led to a high resistance, which explains its late clinical manifestation. However, especially in elderly people, the relationship between the

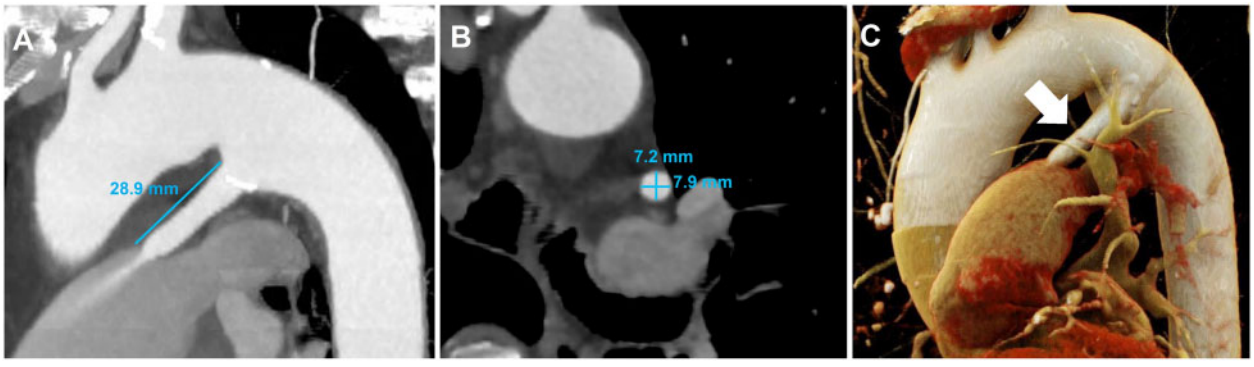


Figure 2 Computed tomography angiography of the patent ductus arteriosus. (*A* and *B*) Oblique sagittal and oblique multiplanar reformations: The duct had a conical (type A) shape with a length of 29 mm, a very small pulmonary ostial diameter of only about 1 mm, and an aortic ostial diameter of about 8 mm. (*C*) Three-dimensional volume rendering technique reconstruction shows a large patent duct (white arrow) from the distal part of the aortic arch to the pulmonary artery.

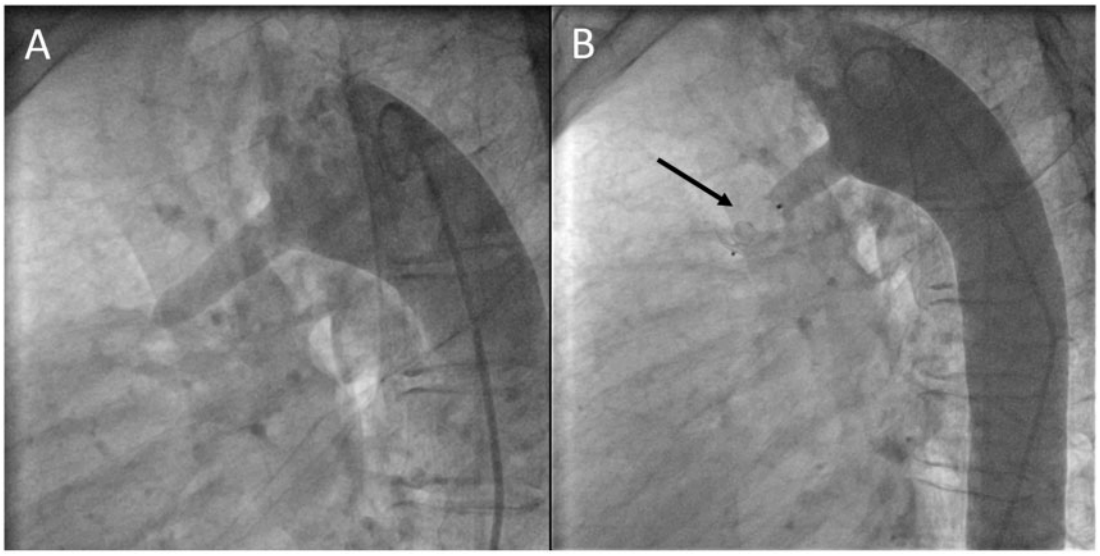
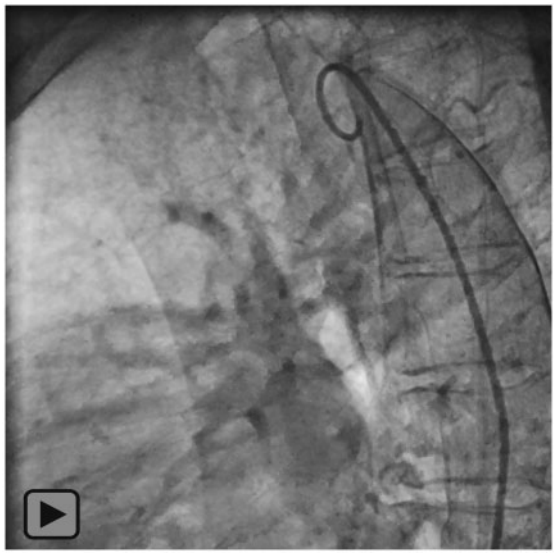
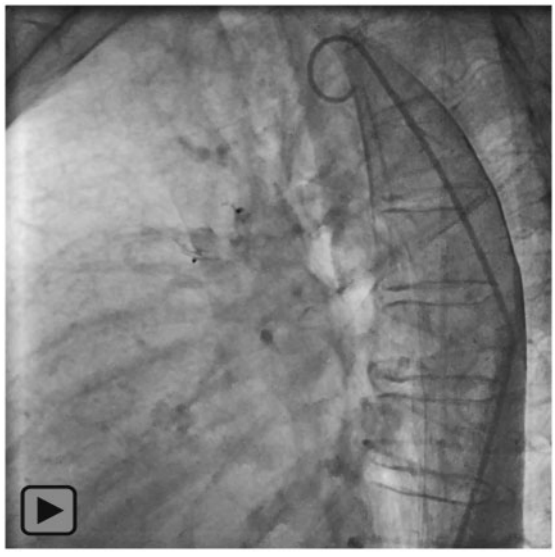


Figure 3 Interventional patent ductus arteriosus closure by an Amplatzer duct occluder II 06-06 mm. Invasive angiography before (*A*) and after interventional duct closure (*B*). There was no residual flow after closure. The Amplatzer duct occluder II is marked with an arrow.



Video 2 Invasive angiography before duct closure.



Video 3 Invasive angiography after duct closure using an Amplatzer duct occluder II 06-06 mm.

detected PDA and the individual symptomatology must be critically assessed, also with reference to incidental findings or relevant underlying pathology of patient's symptoms.[3] In our patient, a significant extracardiac left-to-right shunt was a plausible explanation for the observed left ventricular dysfunction and the worsening heart failure symptoms, as arterial hypertension was well controlled under drug therapy. Furthermore, coronary angiography ruled out significant coronary artery disease. However, arterial hypertension may have contributed to the development of left ventricular dysfunction. We found borderline pulmonary hypertension only with invasive pressure measurement. Therefore, the PDA was closed electively with an Amplatzer duct occluder II in agreement with the 2020 European Society of Cardiology Guidelines for the management of adult congenital heart disease.[2] Device closure is preferred when technically suitable.[2] In patients with evidence of left ventricular volume overload without pulmonary arterial hypertension (pulmonary vascular resistance <3 Wood units) PDA closure is recommended regardless

of symptoms (class 1C recommendation) and in patients with pulmonary arterial hypertension with a pulmonary vascular resistance between 3 and 5 Wood units in case of a Qp:Qs >1.5 (class IIa C recommendation).[2] There was no residual flow across the PDA after closure. Although global systolic left ventricular function, left ventricular end-diastolic diameter, and NT-proBNP did not improve significantly during a 5-month follow-up, the patient already had a clear benefit. There were no longer any heart failure symptoms and physical performance improved to normal levels. A 5-month follow-up period is probably too short to observe a reversal of left ventricular remodelling caused by volume overload due to a shunt that lasted for nearly seven decades. After 15 months, however, the left ventricular ejection fraction slightly increased and NT-proBNP decreased to the normal range.

Conclusion

Congenital heart diseases should be considered as a possible cause of heart failure in elderly patients when other causes have been excluded. In cases of congenital heart disease, as in our patient, symptoms may be reversed by interventional procedures in addition to the standard medical treatment of heart failure.

Lead author biography

Dr Johannes Mair is currently the head of the intensive coronary care unit of the Department of Internal Medicine III – Cardiology and Angiology of the Medical University Innsbruck in Austria. He is also an interventional cardiologist with an interest in the management of adults with congenital heart diseases. His main research interests are in the field of cardiac biomarkers. He is a founding member of the European Society of Cardiology (ESC) Study Group on Biomarkers in Cardiology of the Acute Cardiovascular Care Association (ACVA).

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

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Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The authors confirm that written consent for submission and publication of this case report including images and videos and associated text has been obtained from the patient in line with COPE guidance.

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