

Multiple pulmonary aneurysms and infective endocarditis as initial presentation of persistent ductus arteriosus

Múltiples aneurismas pulmonares secundarios a endocarditis infecciosa asociada a persistencia del conducto arterioso en el adulto

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Case presentation

A 28-year-old man, apparently healthy, started with progressive malaise, left subcostal pleuritic intermittent pain, recurrent fever, and unwilling weight loss during the past 3 months. After an anomalous non-specified cardiovascular exploration while in an emergency care unit, he was referred to an external cardiologist. Initial workup studies included a transthoracic echocardiogram (TTE) which revealed concentric left ventricle (LV) hypertrophy and also a computed tomography pulmonary angiography (CPTA) that found multiple bilateral pulmonary aneurysms. At a tertiary assistance center, his case was reframed. Initial cardiovascular examination revealed tachycardia, a left superior parasternal heave along a harsh, machine-like, continuous murmur at the pulmonic area irradiated to the ipsilateral dorsum; the rest of the physical examination was unremarkable. A thoracic chest roentgenogram showed significant distal focal right pulmonary dilation (Fig. 1A, white arrow), and a 12-lead electrocardiogram showed LV hypertrophy and diastolic overload. A new TTE examination found an aliasing artifact at the level of the pulmonary trunk, highly suggestive of patent ductus arteriosus (PDA) presence (Fig. 1B) with a left to right shunt (peak gradient 56 mmHg), and in addition, hyperechoic sessile images (vegetations) no longer of 5 mm, attached to the pulmonic and mitral valves, confirmed through transesophageal

echocardiography (TEE) (Figs. 1C and D, white arrows). Infective endocarditis (IE) was suspected; therefore, he was admitted to the coronary care unit, a first set of peripheral blood cultures was drawn, and empiric antibiotic treatment with third-generation cephalosporin (ceftriaxone) was initiated. Multiple splenic infarctions as well as bilateral pulmonary infarctions were found through computed tomography, along with several mycotic aneurysms, the widest up to 4 cm (Fig. 2A, white arrows) and a PDA connected to the left pulmonary artery, Krichenko type A (Fig. 2B, white arrow). After exhaustive interrogation, no immunosuppression was found, and in addition, the patient recalled an early dental procedure months ago. *S. viridians* was detected in retrieved blood cultures; henceforth, cephalosporin was continued during hospitalization. The heart team meeting recommended surgical treatment: PDA excretion, allogeneous mitral valve replacement, pulmonic valve checking, and expectant management respecting pulmonic vasculature affection. After a complete post-surgical recovery, ambulatory follow-up ensued, including oral antibiotic treatment for up to 6 weeks after the surgical procedure, with an adequate evolution.

Discussion

The ductus arteriosus (DA) is a vascular structure that bridges the proximal descending aorta to the pulmonary

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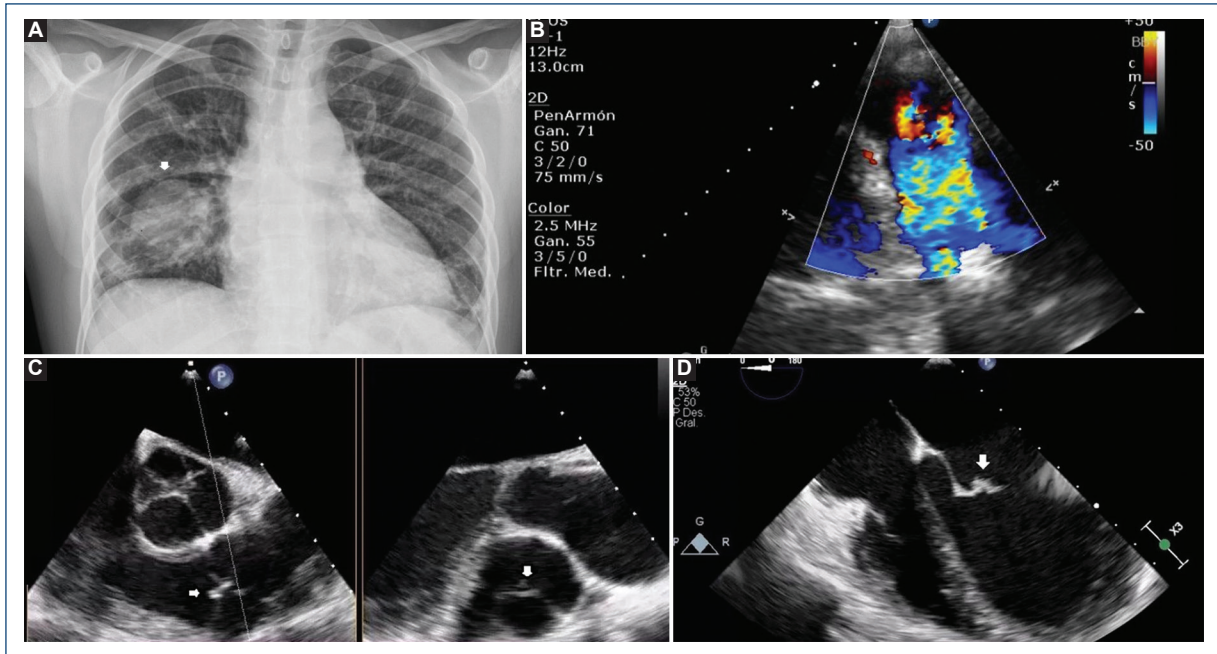


Figure 1. **A:** postero-anterior chest X-ray projection: significant distal dilatation of right pulmonary artery can be seen (white arrow), along discrete cardiomegaly, CTI 0.52 secondary to left chamber enlargement. **B:** transthoracic echocardiography, suprasternal view: pulmonary artery trunk is visualized, aliasing is seen due to left to right shunt occurrence, probably due to a patent ductus arteriosus (not appreciated). **C:** transesophageal echocardiography, upper esophageal view: a hyperechoic image is seen attached to pulmonary valves, corresponding to a vegetation (white arrows). **D:** transesophageal echocardiography, mid esophageal view: mitral valve is also affected, specifically the anterior valve (white arrows).

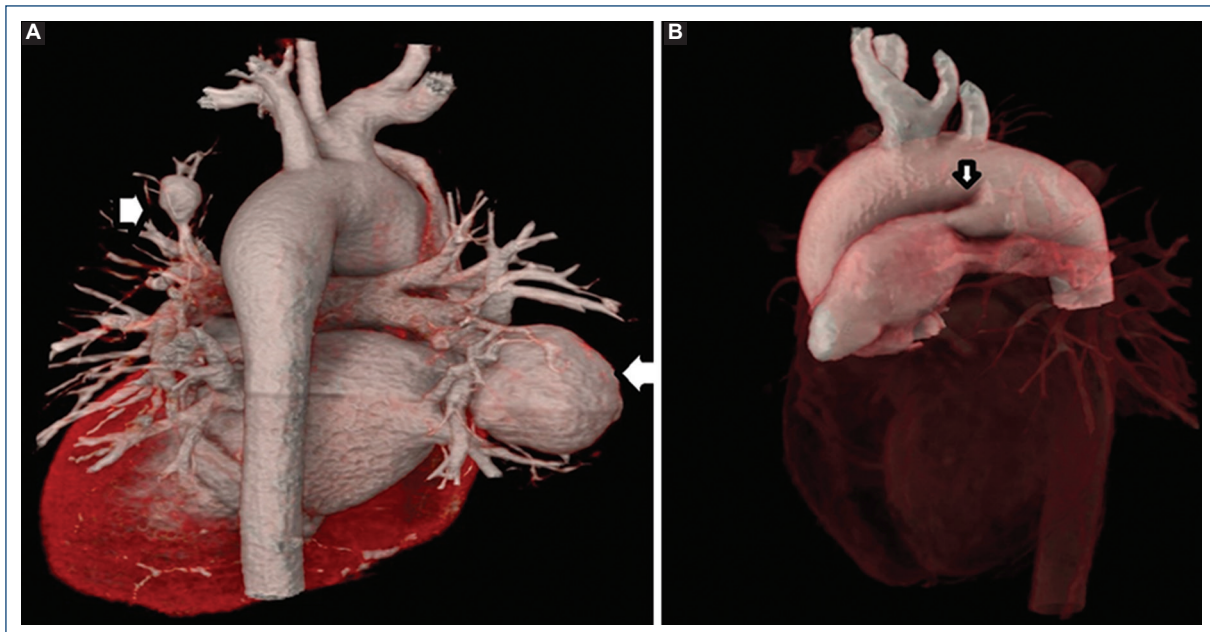


Figure 2. **A:** computed angiography, 3D reconstruction: bilateral pulmonary aneurysm can be seen, the greatest of them localized in the right pulmonary artery (left side of the screen, white arrows). **B:** computed angiography, 3D reconstruction: persistent ductus arteriosus between the left pulmonary artery and descending aorta is visualized (white arrow).

artery near the origin of the left branch pulmonary artery. Essential during intrauterine life, it diverts cardiac output away from the highly resistant and poorly functional pulmonary vascular bed, toward systemic circulation, and finally, to the placenta. After birth, DA suffers a first functional closure at 24-96 h old age, and after 2-4 weeks, an anatomical and definitive closure^{1,2}. If DA persists, especially in preterm and some newborns, it endures no longer than a year². PDA might account for 5-10% of congenital heart diseases (CHD). Small PDA might pass underdiagnosed; however, significant PDA, defined as a left-to-right shunt with Qp: Qs > 1.5, carries an increased pulmonary overflow and left-sided heart cavity overload altogether; and lately, to pulmonary vasculature remodeling and PH, an ultimate and irreversible complication. Therefore, the diagnosis of significant PDA precludes percutaneous or surgical closure, as long as pulmonary vascular resistance does not exceed 5 Wood Units (WU)³.

Even though nowadays it is a quite uncommon complication (< 1% of PDA cases), PDA natural history might lead to infective endarteritis (IEa) and most commonly IE since altered hemodynamics beget persistent stress and vascular injury⁴. Both predisposing conditions and infection sources should exist to develop IE besides endocardium disruption and later fibrin deposition along platelet aggregation^{4,5}. IE might develop in an acute and aggressive fashion, or alternatively, with a subacute presentation. Fever, chills, malaise, and general status deterioration are the most common symptoms. Other clinical manifestations such as cardiac murmur, conduction disturbance, heart failure, and distal alterations might depend on local heart affectation and embolic and immunological phenomena, respectively.

Blood cultures are a cornerstone of the diagnostic approach, with up to 90% of cases having positive results; most cases are related to staphylococci and streptococci infection⁶. The current diagnostic gold standard is based on modified Duke criteria^{4,5}, a composite of clinical, microbiological, and imaging criteria, which are both sensitive and specific for IE. Both TTE and TEE constitute the second cornerstone study, with up to 70-90% sensitivity capacity and 90% specificity⁷. Right-sided IE is uncommon, especially in pulmonic valves, with less than 2% of IE total cases. When embolization occurs, pulmonary parenchyma and its vasculature might be compromised, typically resulting in (IEa) and some cases even mycotic aneurysm since both focal arterial alteration and concomitant infectious invasion might occur, due to bacteremia or contiguous spreading⁸. Like IE, blood cultures in IEa might be positive in up to 85% of cases⁹.

Proper IE treatment includes parenteral and purported antibiotic administration. In up to 50% of cases, prompt surgical treatment is needed^{4,5,10}. Factors

associated with poor prognosis are persistent infection and embolic phenomena despite appropriate antibiotic treatment, worsening heart failure, valvular dysfunction, large vegetation size, and multivalvular involvement⁶.

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Conflicts of interest

None.

Ethical disclosures

Protection of human and animal subjects. The authors declare that no experiments were performed on humans or animals for this study.

Confidentiality of data. The authors declare that they have followed the protocols of their work center on the publication of patient data.

Right to privacy and informed consent. The authors have obtained approval from the Ethics Committee for the analysis and publication of routinely acquired clinical data, and informed consent was not required for this retrospective observational study.

Use of artificial intelligence for generating text. The authors declare that they have not used any type of generative artificial intelligence for the writing of this manuscript, nor for the creation of images, graphics, tables, or their corresponding captions.

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