

Large Patent Ductus Arteriosus in an Adult

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Abstract: PDA is usually diagnosed and treated shortly after birth, it is rarely seen in adults. Few symptomatic cases of more than 40 years old with uncorrected PDA were reported in the literature. The natural history of adult PDA is characterized by a rising annual death rate from heart failure and bacterial endocarditis. Eisenmenger's physiology is less often associated with PDA than with large atrial or ventricular septal defects. We present the case of a 42 years old patient who was first diagnosed with PDA during adulthood.

Keywords: Patent Ductus Arteriosus, PDA, Grown-Up Congenital Heart Disease, GUCH

1. Introduction

Patent ductus arteriosus (PDA) is a rare diagnosis in adults, since symptoms and signs usually occur in infancy and most cases are treated shortly after diagnosis. We present the case of a patient who was first diagnosed with PDA during adulthood.

2. Case Report

A 42-year-old female patient, born to a first-degree consanguineous marriage, was referred to our hospital for surgical treatment of a pulmonary stenosis and a double-chambered right ventricle.

She presented with a grade IV progressive dyspnea, that first appeared at the age of 39 years old. Physical examination revealed mild tachypnea at rest, BP=110/50 mmHg, SaO₂ at 85%, cyanosis, differential clubbing and conjunctival hyperemia. A fixed split second heart sound on auscultation with no murmur and peripheral signs of right heart failure.

In room air, she had hypoxemia (PaO₂: 52mmHg) and normocarbica (PaCO₂: 39mmHg) with SaO₂ of 87%. Hemoglobin was 20 g/dL, and hematocrit was 62%.

EKG showed regular sinus rhythm at 90 beats/minute with right heart axis deviation, right ventricular hypertrophy, and negative anteroseptal and biphasic inferior T waves. Chest X-ray demonstrated cardiomegaly (CI=0,62), elevated cardiac apex, enlarged right atrium, prominent pulmonary outflow tract, enlarged pulmonary arteries with mild pruning of peripheral pulmonary vessels.

TTE showed biventricular hypertrophy predominantly right-sided, with a dynamic stenosis, no obstacles in the outflow tracts, a moderately dilated right atrium, an enlarged pulmonary trunk, trivial tricuspid and pulmonary regurgitations. A 10 mm patent ductus arteriosus was identified with a left-to-right shunt.

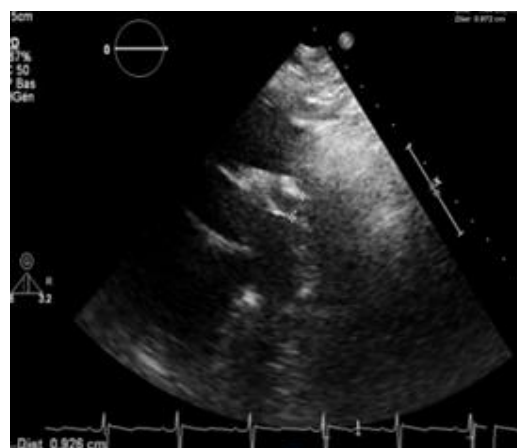
Right and left heart catheterization revealed a left-to-right shunt through the defect and severe isosystemic pulmonary hypertension (PAP= 131/73 (101 mmHg)), with increased pulmonary vascular resistance. Aortic root injection confirmed the large PDA.

Oxygen and high dose aspirin (300mg) were promptly started. On the second day of the treatment, SaO₂ was 90%

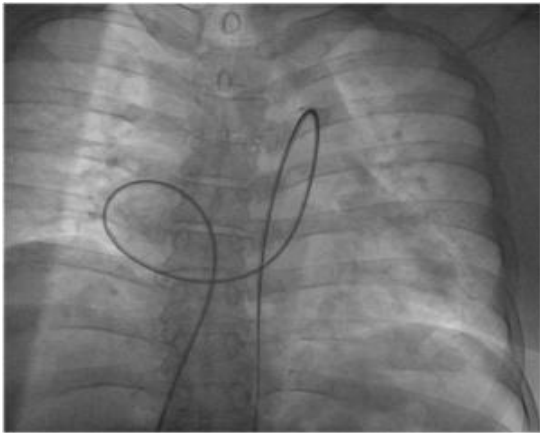
in room air. Due to the lack of financial means, calcium channel blockers were discussed with the referring physician. The presence of Eisenmenger syndrome contraindicated the PDA's closure. At follow-up, she was asymptomatic at rest; however she had dyspnea and became desaturated at minimal exertion.



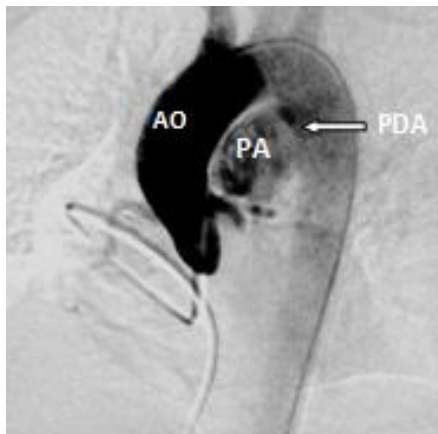
A : Parasternal short axis view shows biventricular hypertrophy predominantly right sided



B: Upper parasternal plane 2D image shows the large PDA



C: Right catheterization: The catheter advanced from the PA to the descending aorta through the PDA



D: Lateral aortogram showing the PDA

4. Conclusion

Uncorrected PDA is associated with high mortality rates. Few symptomatic cases of more than 40 years old with uncorrected PDA were reported in the literature. Our patient reached adulthood with an untreated, significant PDA. This very unusual natural history seems to merit to be reported.

References

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3. Discussion

PDA is usually diagnosed and treated shortly after birth; it is rarely seen in adults. The natural history of adult PDA is characterized by a rising annual death rate from heart failure and bacterial endocarditis. Eisenmenger's physiology is less often associated with a PDA than with a large atrial or ventricular septal defect.

Clinical findings can vary depending on the size of the PDA and associated defects. EKG and Chest X-ray findings are non-specific. The diagnosis rests upon TTE, but can prove difficult and sometimes impossible in adults due to interference of the lung and the increased size of the thoracic cage. It shows the presence of an abnormal turbulent mosaic flow pattern within the pulmonary artery that must be proven to be originating from the descending aorta; because several other disorders also cause this flow pattern such as aortopulmonary septal defect and pulmonary stenosis. Thus, a multimodal approach (TTE, TOE, CT, MRI, heart catheterization) in assessing adult PDA remains important.

Closure of a PDA is only feasible when irreversible pulmonary hypertension has not yet developed. In adults, percutaneous closure with a closure device or coil is the preferred treatment. PDA patients who are beyond surgical or percutaneous closure are treated according to the guidelines for Eisenmenger syndrome.