



Spontaneous Closure of Patent Ductus Arteriosus in an Adult Patient: A Case Report

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Authors' contributions

This work was carried out in collaboration between all authors. Author ST wrote the draft of the manuscript. Author ST managed the literature searches. Author CTL designed the figures, managed literature searches and contributed to the correction of the draft. Author CSW provided the case, the figures and supervised the work. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

Patent Ductus Arteriosus (PDA) is a well-described congenital heart disease in infants and children but is less commonly reported in adults. Mechanical, surgical and spontaneous closure of PDAs in infants and fetuses are common, however spontaneous closure of PDA in adults is uncommon and has not been reported in the recent literature. We present a case report of spontaneous closure of a PDA in a 49 years old patient demonstrated on cardiac computed tomography (CT).

Keywords: Patent ductus arteriosus; PDA; spontaneous closure.

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1. INTRODUCTION

Patent Ductus Arteriosus (PDA) is common congenital heart disease of childhood. Although PDA closure in infant is well recognized, the spontaneous closure of PDA in adult rarely has been published. Our case report shows spontaneous closure of a PDA in an adult patient documented on cardiac computed tomography (CT).

2. CASE PRESENTATION

A 49-year-old African American man presented with the chief complaint "my heart is on the wrong side". The patient has initially presented to the hospital due to a motor vehicle accident 8 years earlier. At the time, a chest radiograph showed a small right lung and rightward mediastinal shift (Fig. 1). These findings raised the possibility of a complex congenital venolobar syndrome consisting of a hypoplastic lung, an abnormal arterial supply and an abnormal venous drainage [1]. Chest CT with contrast demonstrated no chest trauma or aortic injury or findings of congenital venolobar syndrome. A small connection between aortic arch and pulmonary artery was incidentally found and diagnosed as a PDA (Fig. 2A). The patient has no respiratory or cardiac symptoms during that visit.

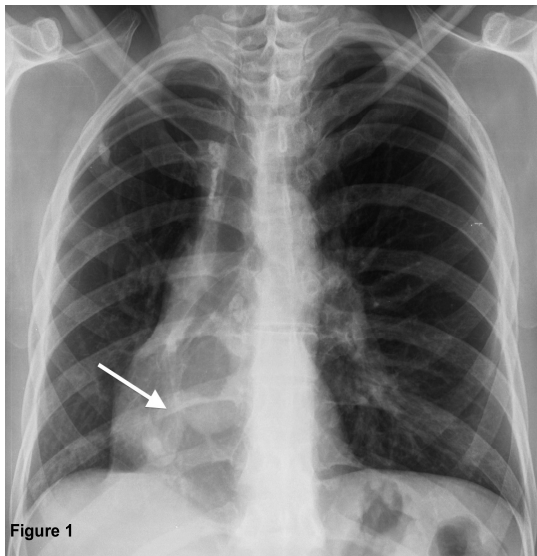


Figure 1

Fig. 1. A PA chest radiograph demonstrates a small right lung and prominent right lower lobe vessels (arrow) concerning for congenital venolobar syndrome

The patient returned to the hospital 5 years later complaining of chest discomfort. Echocardiography performed at that time showed abnormal cardiac orientation within the chest, normal left and right ventricles, normal right atrium, a mildly dilated left atrium, mild aortic tricuspid and pulmonic regurgitation. The diastolic flow into the distal pulmonary artery was not well evaluated and was believed to represent either a PDA or fistula.

The patient had complained of worsening dyspnea, chest discomfort and right chest pain for 6 months before the current visit. Physical examination revealed a BP 118/86 mmHg, HR 83 beats per minute, and a non-significant general, chest and abdominal physical examination. Cardiovascular auscultation showed regular rate and rhythm, no rubs or gallops, and no diastolic murmur. A recent stress echocardiogram was normal.

Coronary CT angiogram (CTA) was requested by the Cardiology and Interventional Radiology services to rule out coronary artery disease and possible coronary anomaly. No coronary artery disease or coronary artery anomaly was detected. The previously noted PDA was no longer evident and there was a new calcific structure abutting the aortic arch at the aortic side of PDA (Fig. 2B).

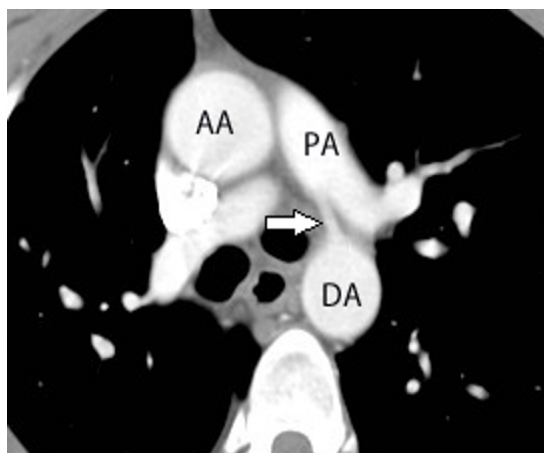
3. DISCUSSION

PDA is commonly found in infants and children with 2:1 female to male ratio. The treatment is essential for symptomatic patients with significant left to right shunt but is not mandatory for asymptomatic patients with a small PDA [2].

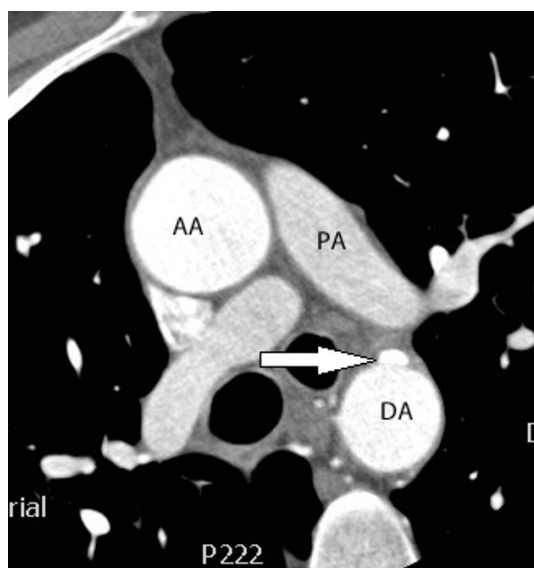
Untreated PDA in adults may lead to severe complications such as pulmonary complications, pulmonary hypertension, bacterial endocarditis and congestive heart failure [3].

Anatomically, the ductus arteriosus is a large channel connecting the main pulmonary artery and the descending aorta, located 5-10 mm distal to the origin of the left subclavian artery in a full-term infant. It diverts the major proportion of the right ventricular output away from the high-resistance pulmonary vascular bed into the low-resistance umbilical-placental circulation. After birth, the systemic vascular resistance rises while the pulmonary vascular resistance falls. The alteration in arterial pO₂, circulating PGE₂ and blood pressure within the lumen of the ductus

promotes functional closure (within 24 hours) followed by anatomical closure of the ductus arteriosus (within a few days or weeks) [3]. Interruption of this normal process leads to a PDA.



(A)



(B)

Fig. 2A-B. An enhancing slit-like structure between the aortic arch and the pulmonary artery compatible with PDA was seen on the same chest CT (arrow in Fig. 2A). Coronary CTA performed 8 years later shows a new coarse calcification at the aortic end of PDA. A patent PDA is no longer seen (arrow in Fig. 2B)

Current treatment approaches for PDA include pharmacological treatment of PDA in preterm infants by using Indomethacin or Ibuprofen, as well as transcatheter PDA occlusion and surgical clipping/ligation of the PDA [4]. Spontaneous closure of the PDA and ductus arteriosus aneurysm in infants has been reported [5]. There is a recent report of spontaneous closure of PDA in a 16 months old Taiwanese girl following Kawasaki's disease. The closure was presumably due to prolonged inflammation from generalized vasculitis [6].

The prevalence of PDA in adults is not well established. Patients may present with congestive heart failure, pulmonary edema [7], and non-cardiac related symptoms [8], however asymptomatic cases are not uncommon [8]. The oldest adult with PDA ever reported is a 92 year-old Japanese woman who presented with infective endocarditis [9].

Spontaneous closure of PDA in adults has not been report in the CT era. However, there are scattered case reports of this phenomenon in the older literature. The diagnosis in these instances was based on physical examination or disappearance of the cardiac murmur. One 44 years old man presented with disappearance of his continuous murmur which was detected when he was in his teen's [10]. Spontaneous closure may be a sequela of infection or thrombosis [11].

The PDA found in our patient was incidental and small. Although the echocardiogram performed 3 years before his last visit showed diastolic flow into the distal pulmonary artery which may have represented a PDA or fistula. There was no significant chest or respiratory symptoms promoting the patient to seek medical attention until 6 months before his last visit. The most recent stress echocardiogram and cardiovascular evaluation were normal which are consistent with PDA closure as seen on coronary CTA.

Vascular calcification is a well-known marker for cardiovascular disease and is caused by a number of different mechanisms. It is a complex process occurring at the molecular level [10]. With advanced age, vascular inflammation, hypertension, and certain metabolic disorders, calcium can accumulate in the arterial system [12].

Development of new calcification at the aortic end of PDA in our patient simulating mechanical

closure was presumably due to infection, inflammation or atherosclerosis.

4. CONCLUSION

Radiologists often encounter coarse calcification abutting the aortic arch on routine chest CT in healthy adults. This may indicate the possibility of unrecognized congenital heart disease, especially a PDA, therefore the prevalence of PDA in the general population may be higher than reported.

CONSENT

Not applicable.

ETHICAL APPROVAL

Not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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