

A rare presentation of patent ductus arteriosus in an adult patient with normal pulmonary hypertension and limb edema

Bahram Pishgoo⁽¹⁾, Amin Saburi⁽²⁾, Arezoo Khosravi⁽³⁾

Case Report

Abstract

BACKGROUND: Patent ductus arteriosus (PDA) at childhood is one of the five major and frequent congenital abnormalities, but it can be rarely seen in adults. Pulmonary hypertension (PHTN) and other presentations such as heart failure and edema are the identified complications of longstanding PDA, but adult case with no permanent heart symptoms and PHTN was rare. We reported a rare case of with an obvious PDA and normal pulmonary pressure.

CASE REPORT: A 61-year-old woman presented with dyspnea (New York Heart Association class 2), chest pain, and lower limb edema. Echocardiogram showed; normal left ventricular chamber size and function, normal size of both atria. Furthermore, an obvious PDA (diameter = 6-7 mm) connecting the aortic arch to the pulmonary artery was reported in echocardiography. No lung congestion and evidence for PHTN was reported by computed tomographic angiography [Pulmonary capillary wedge pressure (PCWP) = 30 mmHg]. The patient was treated with antihypertensive drugs and after 1 and 3 months follow-up, edema and other symptoms were resolved.

CONCLUSION: Finally, we conclude that PDA in adulthood can present with nonspecific cardiovascular symptoms, and it seems that PHTN is not a fixed echocardiographic finding in these patients.

Keywords: Adults, Edema, Patent Ductus Arteriosus eri, Pulmonary Hypertension

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Introduction

Patent ductus arteriosus (PDA) at childhood is one of the five major and frequent congenital abnormalities that its frequency was reported as 10-14.4% of all congenital defects.^{1,2} "PDA normally closes soon after birth, but in some newborns it does not close spontaneously, and there is continuous flow from the aorta to the pulmonary artery (i.e., left-to-right shunting)".² Rarely, PDA maybe presented in adulthood but most adult cases reported with pulmonary hypertension (PHTN). PHTN and other presentations such as heart failure and edema are the identified complications of longstanding PDA, but adult case with no permanent heart symptoms and PHTN was rare. We reported a rare case of with an obvious PDA and normal pulmonary pressure.

Case Report

A 61-year-old woman presented with dyspnea (New York Heart Association class 2), chest pain and lower limb edema. In past medical history, she was under treatment for hypertension, hyperlipidemia, and acute coronary syndrome (ACS) and she had a history of hospital admission twice for chest pain and ACS. Despite the hospital admissions, cardiopulmonary assessment was not perfectly performed to diagnose the main cause of her chest pain. At the first visit, initial blood pressure was 110/70 mmHg, heart rate: 68/min, respiratory rate in normal range, and body temperature was 37 °C. She did not have any complaint of productive or dry cough, fever, nocturnal dyspnea or orthopnea.

Her physical examination revealed an obvious systolic machinery murmur, which was best-heard at the second left intercostal space, and the second heart

1- Associate Professor, Department of Cardiovascular Diseases, School of Medicine AND Cardiovascular Research Center, Baqiyatallah University of Medical Sciences, Tehran, Iran

2- Researcher, Chemical Injuries Research Center, Baqiyatallah University of Medical Sciences, Tehran, Iran

3- Assistant Professor, Cardiovascular Research Center AND Department of Cardiovascular Diseases, School of Medicine, Baqiyatallah University of Medical Sciences, Tehran, Iran

Correspondence to: Arezoo Khosravi, Email: arekhosravi@yahoo.com

sound was physiologically split suggesting an absence of significant PHTN. Pulses and diastolic murmurs were near to normal. Pulmonary rates and wheezing could not find in any of both lungs, but bilateral pretibial pitting edema was obviously seen. Echocardiogram showed; normal left ventricular chamber size and function, normal size of both atria. Furthermore, an obvious PDA (diameter = 6-7 mm) connecting the proximal of descending aorta to the left to the pulmonary artery was reported in echocardiography (Figures 1 and 2).

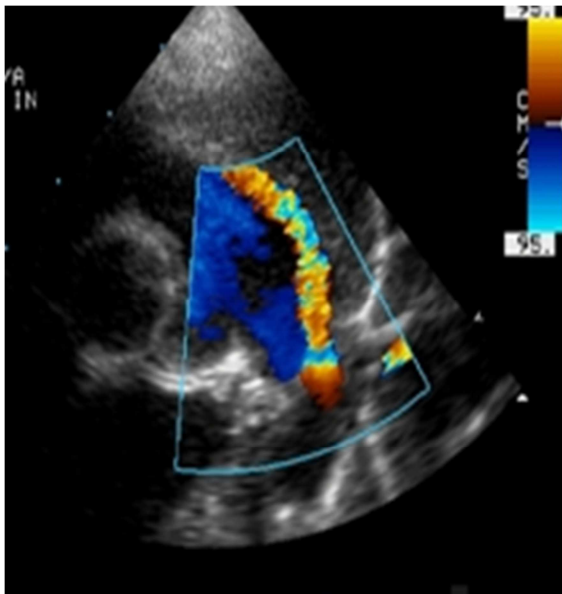


Figure 1. Patent ductus arteriosus (PDA) in Transthoracic echocardiography

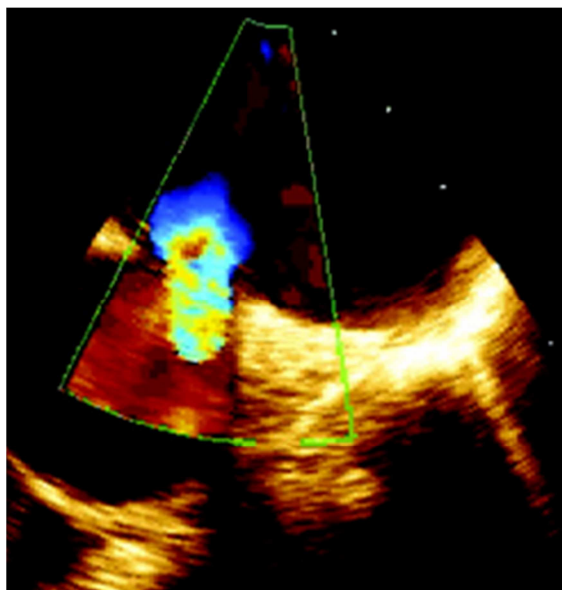


Figure 2: Patent ductus arteriosus (PDA) in Transesophageal echocardiography

For more accurate evaluation, a transthoracic echocardiogram (TTE) was performed, and it showed visualized jet flow at main pulmonary artery, indicating the presence of PDA. TTE also showed mild mitral and tricuspid regurgitation and showed mild diastolic dysfunction but preserved left ventricular function (ejection fraction of the left ventricle = 55%). Patients referred for cardiac catheterization and computed tomographic (CT) angiography. CT angiography was revealed a PDA with 5.5-7 mm luminal width interposed between the roof of left pulmonary artery and descending aorta (both at origins) (Figure 3).



Figure 3. Computed tomographic angiographic findings

No lung congestion and evidence for PHTN was reported by CT angiography (pulmonary capillary wedge pressure: 30 mmHg). Saturation study showed a step up from right pulmonary artery to the right ventricle (72-60 %). Measured pulmonary to systemic blood flow ratio (Q_p/Q_s) through oxygen saturation was 1.26. Renal dysfunction, thyroid imbalance, and other etiology of edema were ruled out. The patient was treated with aspirin (80 mg/day), Lasix (tablet, 40 mg/day), losartan (tablet, 0.5 twice a day), atorvastatin (tablet, 20 mg/day), nitrocontin (tablet, 2.6 twice a day), and atenolol (tablet, 50 mg daily). After 1 and 3 months follow-up, edema and other symptoms were resolved.

Discussion

Presentation of PDA in adulthood often associated with congestive heart failure, pulmonary arterial

hypertension, atrial fibrillation, recurrent pneumonia, sign of volume overload, endocarditis and also may be silent.³ We reported a case of adult PDA that present with general symptoms of cardiovascular disorders. Due to the recent improvement in the quality of medical care services, the survival of premature infants increases. On the other hand, PDA is mostly seen in preterm newborns. Therefore, it is expected that the prevalence of incidental PDA in adult's increase. Thus, primary care physicians need to be alert to the clinical situations suggesting a previously undiagnosed PDA.³

There are few similar reports in the literature review that most of them had presented with PHTN and heart failure.^{4,5} PDA with increase of pulmonary blood flow or pulmonary vascular resistance cause increase of pulmonary pressure and cause PHTN.⁶ Occurrence of symptoms such as Eisenmenger syndrome in systemic to pulmonary shunt are related to location, size and also magnitude of the shunt. It was previously reported that if the size of the defect is large (more than 2.5 mm), occurrence of PHTN and Eisenmenger syndrome will increase.^{7,8} The normal pulmonary artery pressure in our patient despite a moderate defect could be because of the potential ability of pulmonary vascular bed for tolerating longstanding high volume of blood from the childhood.⁹

Surgical treatment is the standard and recommended method for treating these patients but we showed that accurate medical therapy with antihypertensive drugs can help for managing their symptoms although it is not the definite treatment.¹⁰ Recently, percutaneous trans catheter occlusive devices was effectively and safely used in both children and adults and it seems this new interventions can remove the need to surgical ligation in the near future.³ Moreover, Moller and Anderson were revealed that: "In patients with ventricular septal defect, atrial septal defect, and PDA, the Kaplan–Meier survival curves followed a normal curve. When these conditions were present with another malformation, the curves were significantly lower than normal, and showed a marked variation."¹¹ Therefore, it can be a useful clue that pure adult PDA maybe need no further intervention except routine drug therapy but in complicated adult PDA, the treatment protocol may be deferent.

Finally, we conclude that PDA in adulthood can present with nonspecific cardiovascular symptoms, and it seems that PHTN is not a fixed

echocardiographic finding in these patients.

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Conflict of Interests

Authors have no conflict of interests.

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