



### **A Case of Adult PDA with Eisenmenger**

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#### **Introduction**

PDA is an uncommon clinical finding in adult patients. The incidence of PDA is 0.2 to 0.4 % in term infants. However depending on estimated gestational age the incidence in preterm neonates varies from 20 to 60% on the third day of life. PDA accounts for 6 to 11% of all the congenital heart diseases.[1,2,3]

Although most cases of PDA seem to occur sporadically, multifactorial inheritance is believed to underlie many cases. These people are thought to possess a genetic predisposition acted on by an environmental trigger that occurs at an unknown but vulnerable time during pregnancy. [4]

PDA is seen twice more often in females than in male.[5]

#### **Case report**

A 34 year old female with no underlying comorbidity presented with chief complaint of shortness of breath for two to three weeks with significant past history of unilateral headache (right side), recurrent oral ulcers, flushing, blurring of vision, rash on face and arthralgias for the same duration. The patient had two children both delivered by normal vaginal delivery with uneventful gestational course. There was history of hysterectomy done three years back for DUB, however biopsy was normal.

On examination the patient was clubbed with grade 3 clubbing in both upper and lower limbs, facial plethora (a rash in malar distribution), no joint swelling or tenderness with a systolic murmur in mitral area and rest examination was unremarkable.

On routine lab investigations the patient had a hemoglobin of 24 gm% with hematocrit of 84 %, thrombocytopenia, proteinuria, hypoxemia, hypocalcemia with calcium of 7.35 and hyperuricemia with uric acid of 8.5. The coagulogram was normal, triple serology was negative, autoimmune profile was negative, serum erythropoietin was within normal range (16). The ECG had signs of pulmonary hypertension with p pulmonale and CXR was consistent with PAH. The USG abdomen showed a congenital atrophic kidney on right side with left normal kidney. Echo revealed EF of 55%, RVSP of 123 + rap, with PDA with reversal of shunt. CT Angio was suggestive of PDA with reversal of shunt with TR and lung fields sequestered with b/l congenital diaphragmatic hernia.

All clinical symptoms and findings were explained by secondary polycythemia presenting in an adult female due to cyanotic heart disease that is PDA in this case.

The patient was managed by phlebotomies done at weekly intervals with sufficient volume replacement by normal saline and after two such phlebotomies the HB dropped to

23 and HCT to 73.5 and 21 and 65 respectively . The patient is on follow up and on ambresartan and tadalafil kit with diltiazem for rate control.

### Discussion

PDA accounts for 6 to 8 % of all adult congenital heart diseases and is third most common after ASD and VSD.

The ductus arteriosus is a physiological structure allowing shunt of blood from the pulmonary artery to the descending aorta during fetal life[ unventilated lung.[ 5]

After birth with commencement of pulmonary blood flow and a two ventricle circulation , a variety of physiological and biochemical signals normally result in complete closure of the ductus. Persistent patency of the ductus may impair systemic cardiac output and result in deleterious effects on cvs and lungs.[6]

After age 20 patients with PDA develop ph or advanced heart failure and 20% die by the age of 30 while 60% die by the age of 60.

The most common symptoms in adult PDA are shortness of breath upon exertion and respiratory distress.other symptoms include palpitations,chest pain,fainting, repeated respiratory infections.[ 7]

The hemodynamic consequences of PDA can be categorized by the degree of left to right shunting based upon the pulmonary to systemic flow ratio (Qp:Qs)

Small –Qp:Qs <1.5-1

Moderate –Qp:Qsbetween 1.5 and 2.2 to 1

Large –Qp:Qs .2.2 to 1.[8]

Individuals with pda have increased mortality and morbidity primarily due to heart failure and rarely infective endocarditis.[9]

Our patient had no symptoms until 30 years of age and as such was a case worth presenting with severe ph, atrophic kidney, secondary polycythemia with shunt reversal .

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