Case Reports

Large Patent Ductus Arteriosus in a 44-Year-Old Woman

Leading to Calcium Deposition in the Left Atrium and Mitral and Aortic Valves

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This report describes unusual autopsy findings in a 44-year-old woman who had a large, calcified patent ductus arteriosus that produced substantial left-to-right shunting. The patient died in 1962, 7 days after patch closure of the aortic orifice of the ductus. Numerous calcific deposits were present in the mural left atrial endocardium, the mitral valve leaflets and annulus, and the aortic valve cusps. The cause of the left-sided calcific deposits was perhaps related to the patient's several-decades-old giant aortopulmonary shunt, causing a major increase in the volume of blood passing through the left-sided cardiac chambers in comparison with the volume in the right side. To our knowledge, such findings in a patient with patent ductus arteriosus have not been reported previously. ***(Tex Heart Inst J 2015;42(3):262-4)***

Key words: *Adult; calcinosis/pathology; cardiomegaly/etiology/radiography; ductus arteriosus, patent/surgery; heart atria/pathology; heart diseases/physiopathology*

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A large patent ductus arteriosus (PDA) is now rarely seen in adults in the Western world; however, this was not necessarily the case 50 years ago. We describe noteworthy autopsy findings in a woman who had presented with symptoms after several decades of aortopulmonary shunting through a large PDA.

Case Report

A childless housewife was born in 1917 and died in 1962, at 44 years of age. A precordial murmur had been present since childhood, when she was diagnosed with rheumatic heart disease. She was asymptomatic until 40 years of age, when peripheral edema, abdominal swelling, and exertional dyspnea developed. The patient was seen initially in November 1959 at the National Heart Institute (Bethesda, Md), at 41 years of age. She was cachectic and in severe right-sided heart failure. Her neck veins were markedly distended. On auscultation, the following were heard: rales in the lung bases, an apical grade 4/6 blowing systolic murmur in association with a thrill that radiated into the left axilla, a diastolic rumble at the apex, a crescendo diastolic grade 3/6 blowing murmur along the left sternal border, and an accentuated P_2. A chest radiograph showed a greatly enlarged cardiac silhouette. An electrocardiogram, measured with normal 10-mm standardization (10 mm=1 mV), showed a total 12-lead QRS voltage of 315 mm (consistent with combined right and left ventricular hypertrophy); the rhythm was atrial fibrillation (Fig. 1).[1] Cardiac catheterization via the transbronchial route (involving endotracheal intubation, with the pressure-recording catheter inserted into the left atrium from the trachea) provided only the left-sided cardiac pressures (Table I). These findings were interpreted as showing "predominant mitral regurgitation." The patient's blood hematocrit was 34%. During her 6-week hospitalization, she was placed on a low-salt diet, chlorothiazide, and acetazolamide, and she lost 40 pounds from her already cachectic frame.

In June 1962, she was readmitted because of similar symptoms and signs. Table I also shows the findings from right-sided and transseptal left-sided cardiac catheterization. The ^{85}Kr and indocyanine green dye curves showed predominant left-to-right shunting (Fig. 2). The catheter was passed from the aorta into the major pulmonary arteries through a PDA, which a chest radiograph showed to contain calcific deposits. The patient's right brachial and femoral arterial oxygen saturations were 92% and 89%, respectively. After a course of diuretic therapy, her peripheral edema and ascites

completely resolved. In July 1962, the PDA was closed via the surgical placement of a Teflon patch at the aortic entrance of the PDA, with use of extracorporeal circulation during the procedure.[2] Postoperative bleeding necessitated the evacuation of a left hemothorax the next day, at which time the right and left ventricular peak systolic pressures were 105 and 80 mmHg, respectively. Hypotension and renal, hepatic, respiratory, and cerebral failure developed thereafter, and the patient died 7 days after the PDA closure operation. (This patient would not have been a candidate for PDA closure today.)

At necropsy, the heart weighed approximately 520 g. The PDA was 1.5 cm in diameter, and its wall was heavily calcified. All 4 cardiac chambers were dilated and had thicker walls than normal: both the right and left ventricular free walls were approximately 1.3 cm thick. The left atrium was dilated much more than the right atrium. The pulmonary trunk and main right and left pulmonary arteries were very dilated.

Figure 3 shows the locations of calcific deposits in several structures: sprinkled within the mural left atrial endocardium, focal within the mitral and aortic valves (with more in the aortic cusps than in the mitral cusps), and in the mitral annular area behind the posterior mitral leaflet.

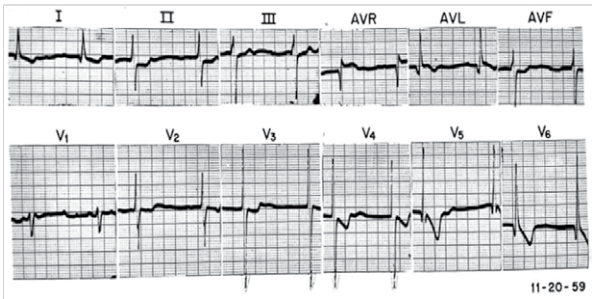

Fig. 1 *Electrocardiogram of the patient at 41 years of age shows atrial fibrillation and excessive QRS voltage consistent with both right and left ventricular hypertrophy.*

TABLE I. Cardiac Hemodynamic Values in the Patient

Pressure Variable (mmHg)	**December 1959**	**May 1962**
Pulmonary artery	—	118/52
Right ventricle	—	118/52
Right atrium		
Mean	—	21
V wave	—	25
Right brachial artery	—	144/60
Femoral artery	150/70	—
Left atrium		
Mean	22	18
V wave	32	27

Discussion

This case is unusual in several aspects. The PDA had been confused with valvular disease for more than 4 decades. The patient's first cardiac catheterization, in 1959, was via the transbronchial route and yielded pressures only in the left side of the heart; these data did not enable a diagnosis of PDA but were consistent with the admitting diagnosis of mitral regurgitation. The right-sided pressures were not determined until 2 years later, and at that time no echocardiography was available for making estimates. The wall of the PDA was heavily calcified at the time of diagnosis, when the patient was 44 years of age. Although the peak systolic pressures were similar in the major pulmonary arteries and in the brachial artery, the shunting through the PDA

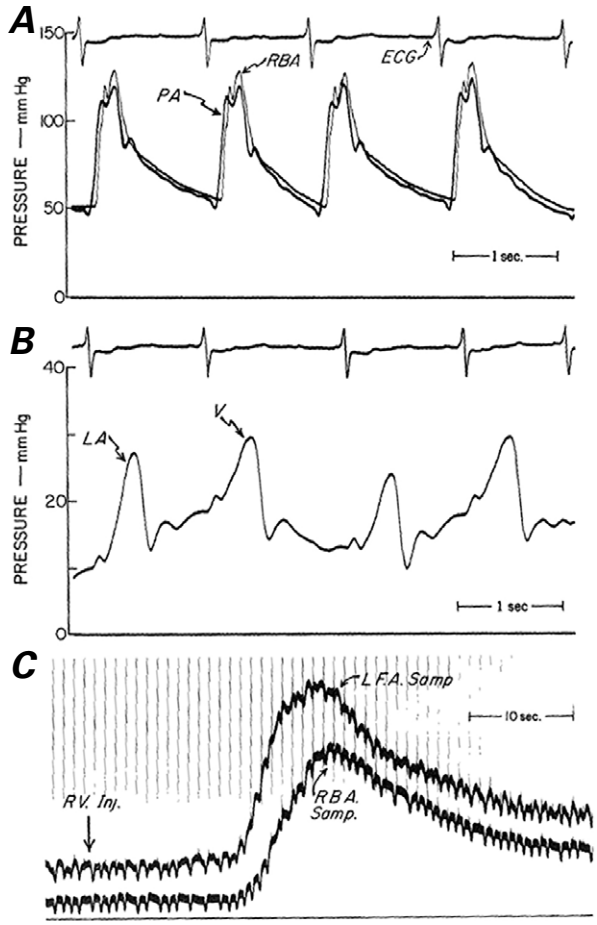

Fig. 2 *Diagrams show findings in June 1962, approximately one month before closure of the ductus.* ***A****) Simultaneous pressure tracings of the pulmonary artery (PA) and right brachial artery (RBA) show similar pressures in each vessel.* ***B****) Left atrial (LA) pressure tracing shows tall V waves.* ***C****) Drawing shows indocyanine green dye curves upon right ventricular injection (RV. Inj.) and sampling (Samp.) at simultaneous times in the left femoral artery (L.F.A.) and right brachial artery (R.B.A.).*

ECG = electrocardiogram

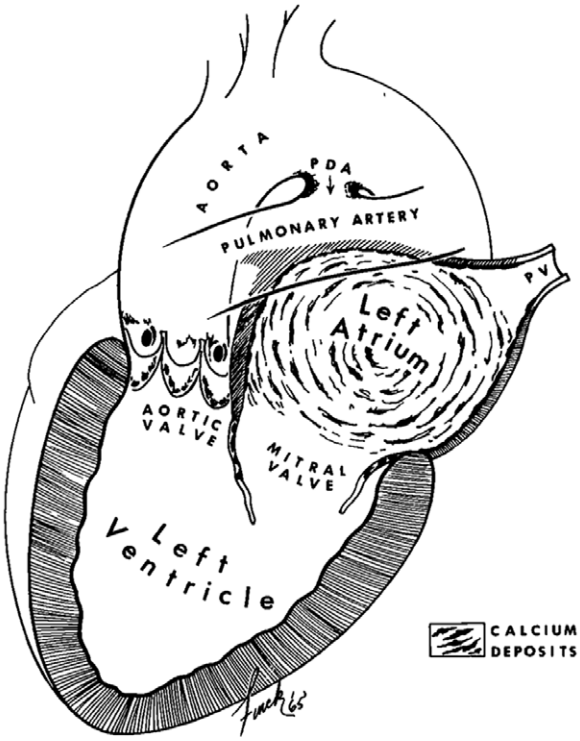

Fig. 3 *Diagram shows locations of calcific deposits in the patent ductus arteriosus (PDA), mitral valve, aortic valve, and left atrial endocardium.*

was overwhelmingly from left to right. Nevertheless, the heart failure was mainly right-sided. The method of closing the calcified PDA was new at the time: inserting a Teflon patch over the aortic entrance into the PDA, with use of extracorporeal circulation.[2] Finally, calcific deposits in the left atrial wall and in the leaflets of the mitral and aortic valves have not to our knowledge been reported previously in patients with PDA.

The reason for the deposits in the left atrial endocardium and in the mitral and aortic valves is unclear. The phenomenon might be explained by the massive volume overload on the left side of the heart—caused by the left-to-right shunting through the PDA—while much smaller volumes of blood passed through the right side of the heart. The increased volume and therefore increased work of the left side of the heart might have caused rapid aging, such that the condition of that side more resembled typical findings in an 85-year-old patient than those expected in a 44-year-old. The calcific deposits in the left atrial endocardium might have resulted in part from the considerable stretching of that chamber due to increased volume and pressure, and from the superimposed mitral regurgitation. In patients with mitral stenosis, calcific deposits have been seen in the left atrial endocardium when a thrombus has organized into calcific plaques; however, this patient never had evidence of left atrial thrombus.[3] Her PDA was heavily calcified, a finding in older patients with a large PDA.

References

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