GIANT PULMONARY ARTERY ANEURYSM, CALCIFIC PULMONARY AND AORTIC STENOSIS AND ABNORMALLY ORIGINATING RIGHT CORONARY ARTERY ALL TOGETHER IN AN OLD WOMAN

Doç. Dr. İrfan BARUTÇU,
Uz. Dr. Hekim KARAPINAR*,
Uz. Dr. Mustafa Akçakoyun,**
Doç. Dr. Ali Metin ESEN,**
Doç. Dr. Mehmet ÖZKAN**

From:
Avicenna Hastanesi Kardiyoloji Kliniği, İstanbul
* Van Yüksek İhtisas Hastanesi Kardiyoloji Kliniği, Van
** Kartal Koşuyolu Yüksek İhtisas Eğitim ve Araştırma Hastanesi, Kardiyoloji Kliniği İstanbul

Address for reprints
Irfan BARUTCU MD,
Avicenna Hospital, Menderes mah.
Atışalanı Cad. 19. Sok No:2
Esenler/İstanbul_TURKEY
Telefon: 0212 611 51 52
Faks: 0212 611 51 11
e-posta: irfanbarutcu@yahoo.com

We report the case of a 63-year-old woman with giant pulmonary artery aneurysm, calcific pulmonary and aortic stenosis and abnormally originating right coronary artery all together.

Key Words: Giant pulmonary artery aneurysm, calcific pulmonary-aortic stenosis and abnormally originating right coronary artery

ÖZET

Dev Pulmoner Arter Anevrizması, Kalsifik Pulmoner ve Aort Darlığı ve Anormal Çıkışlı Sağ Koroner Arter Anamlilerinin Hepsinin Bir Arada Olduğu Yaşlı Bir Kadın Hasta

63 yaşında kadın hastada dev pulmoner arter anevrizması, kalsifik pulmoner ve aort darlığı ve anormal çıkışlı sağ koroner arter anamlilerinin hepsinin bir arada olduğu bir olguyu rapor ediyoruz.

Anahtar Kelimeler: Dev pulmoner arter anevrizması, kalsifik pulmoner-aort darlığı ve anormal çıkışlı sağ koroner arter

INTRODUCTION

Aneurysms of the pulmonary artery (PAA) are rare, and their natural history is not well understood and there is no definitive therapeutic approach. We report the case of a 63-year-old woman with giant pulmonary artery aneurysm, calcific pulmonary and aortic stenosis and abnormally originating right coronary artery all together.

CASE

A 63-year-old woman presented to our hospital with the history of exertion dyspnea and chest discomfort (NYHA class-II) for two years. Her medical history was marked by non-insulin dependent diabetes mellitus and hypertension for 20 years. She had undergone cardiac catheterization 35 years previously, but

there was no information about indication and results of catheterization. Blood pressure was 140/90 mm Hg, pulse rate 95 beats/min, and respiratory rate 16 breaths/min. Grade 3/6 systolic murmurs were heard at the left and right border of the sternum. S1 was normal, and S2 was soft heard. There was no stigma of connective tissue and infectious diseases. ECG revealed atrial fibrillation with rate of 110 beats/min, right bundle branch block and biventricular hypertrophy. On chest radiography, left hilar mass indicating dilation of the left pulmonary artery was detected (figure-1).

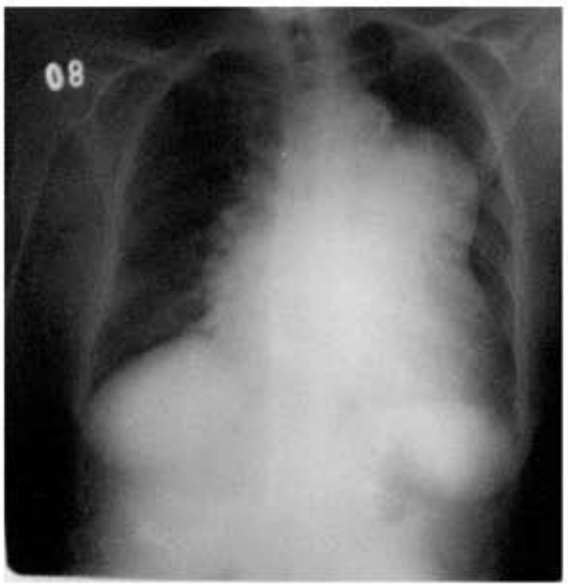

Figure-1: On chest radiography, left hilar mass indicating dilation of the left pulmonary artery was detected.

Transthoracic echocardiography revealed calcific severe pulmonary valve stenosis (maximum gradient: 97 mmHg, mean gradient: 47 mmHg), 8 cm in diameter aneurysm involving the entire main pulmonary artery and the proximal branches (figure-2)
severe aortic valve stenosis(maximum gradient:95 mmHg, mean gradient:66 mmHg), left ventricular concentric hypertrophy (thickness of interventricular septum in diastole:14 mm, thickness of posterior wall in diastole:13 mm), normal left and right ventricular sizes, with preserved systolic functions. Aortic valve was

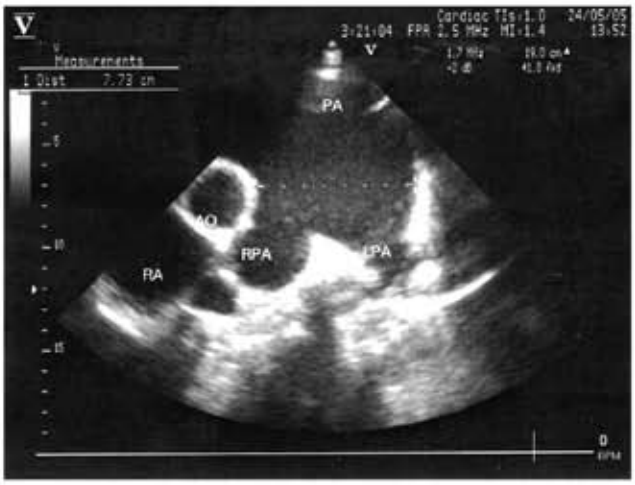

Figure-2: On TEE aneurysm involving the entire main pulmonary artery and the proximal branches.

tricuspid and calcific. Magnetic resonance angiography with gadolinium enhancement confirmed the presence of a giant pulmonary artery aneurysm (with 8 cm transverse diameter, 11.5 cm antero-posterior diameter), and normal diameter of aorta (figure-3).

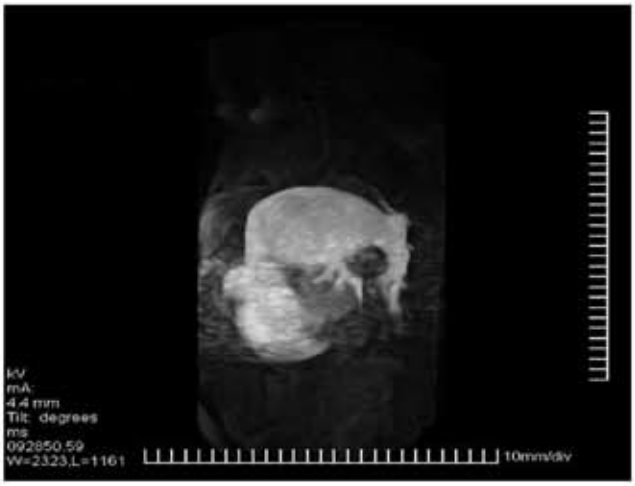

Figure-3: Magnetic resonance angiography with gadolinium enhancement confirmed the presence of a giant pulmonary artery aneurysm (with 8 cm transverse diameter, 11.5 cm antero-posterior diameter), and normal diameter of aorta

Cardiac catheterization revealed a 43 mmHg peak-to-peak gradient across the pulmonary valve and 55 mmHg systolic pulmonary artery

pressure. Oximetric study revealed no left-to-right shunt. Coronary angiogram revealed that right coronary artery originated from left sinus valsalva, with only non-critical plaques. We suggested operation because of symptomatic severe aortic and pulmonary stenosis, but patient refused operation. She remained alive at four-month follow-up and asymptomatic except exertion dyspnea.

DISCUSSION

Aneurysms of the pulmonary artery are uncommon. Deterling and Clagett reported only eight cases in a review of 109.571 necropsies [1]. Pulmonary arterial aneurysm has been described in patients with persistent ductus arteriosus, ventricular septal defect, atrial septal defect, and transposition of the great arteries with ventricular septal defect, where the pulmonary circulation may have considerable volume and pressure overload [1]. It is also associated with connective tissue disorders such as Marfan syndrome, Ehlers-Danlos syndrome, systemic vasculitides including Behcet's disease and giant cell arteritis [2,3]. Other causes are atherosclerosis, degenerative changes of the elastic media, especially cystic medial necrosis, vasculitis, hypertension, trauma, arterio-venous communication, and pulmonary valve stenosis and infection including syphilis, bacterial endocarditis, tuberculosis [4,5]. Due to lack of systemic and infectious disorder, we think that pulmonary valve stenosis may be possible reason of PAA.

The natural history of low pressure PAA is unknown yet. Progression to dissection or rupture in the setting of severe pulmonary arterial hypertension, and congenital left to right shunts was documented [1,6]. Similarly, evidence of progression to arterial wall dissection and rupture has been documented in patients with connective tissue disorders [7]. Wall stress, the most important determinant of progression to rupture, is directly proportional to the pressure and radius of the vessel wall and is inversely proportional to the wall thickness according the Laplace law [8]. Our patient reached 63 years old in despite of severe pulmonary valve stenosis, significant pulmonary hypertension, and giant diameters of PAA. This case confirmed why the line between surgical and medical treatment is indefiniteness.

We could find no case like ours in literature, and this may be the first case of calcific aortic and pulmonary valves stenosis, and giant PAA, and coronary artery anomaly all together in an old patient.

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