An unusual severe pulmonic stenosis case without significant electrocardiographic changes

Belirgin elektrokardiyografik değişikliği olmayan nadir bir ciddi pulmoner stenoz olgusu

A 21-year-old man was admitted with the complaints of dyspnea on exertion and light-headedness. A 3/6 grade systolic ejection murmur was heard in the third left intercostal space. Continuous wave Doppler revealed 200 mmHg pressure gradient across the stenotic pulmonary valve associated with secondary hypertrophic subpulmonary stenosis (Fig. 1). Although the severity of pulmonic stenosis is closely related to R wave amplitude in lead V1, tall P waves and monophasic R wave were not observed on the electrocardiogram (Fig. 2). During right heart catheterization, 203 mmHg pressure gradient was found between main pulmonary artery and right ventricle (Fig. 3).

A severe coarctation of the aorta incidentally diagnosed during cardiac catheterization of a 40-year-old male patient presenting acute coronary syndrome

Akut koroner sendrom nedeni ile başvuran 40 yaşında erkek hastada kardiyak kateterizasyon sırasında tanı konulan ciddi aort koartasyonu

Coarctation is defined as a narrowing of the lumen of the aorta that obstructs flow. Typically, it is located at the insertion of the ductus or ligamentum arteriosum. It accounts for 5% to 10% of congenital heart disease and occurs more frequently in Caucasian and males. The disorder is typically diagnosed in childhood but may go undetected well into adulthood. Most patients develop persistent systemic hypertension and are at risk for premature coronary artery disease.

A 40-year-old man was referred to our hospital with the diagnosis of non-ST elevation myocardial infarction. There was no any cardiovascular risk factors except for systemic hypertension. A systolic ejection murmur at the left upper sternal border and upper extremity hypertension in conjunction with diminished femoral pulsations was found in cardiovascular physical examination. Electrocardiogram showed lateral ischemia. Coronary angiography was planned through the right femoral artery. However, the guidewire did not move forward due to aortic obstruction (Fig. 1, Video 1,2. See corresponding video images at www.anakarder.com). Therefore, coronary angiography was performed through the left brachial artery. Left anterior descending artery and left circumflex artery lesions were found on coronary angiography (Fig. 2). Arcus aortography and computed tomography-angiography showed coarctation of the aorta distal to the left subclavian artery origin (Fig. 3). The patient was referred to the department of cardiovascular surgery for the surgical management of coarctation of aorta and coronary artery disease.

Figure 1. Continuous wave (CW) Doppler shows severe mobile dome-shaped pulmonary stenosis and secondary hypertrophic subpulmonary stenosis. The velocity across the stenotic pulmonary valve was 7.1 cm, reflecting a peak instantaneous gradient of 200 mmHg. The asymmetric profile within the envelope of CW Doppler signal represents the gradient across the zone of secondary hypertrophy.

Figure 2. The absence of tall P waves in the inferior leads and monophasic R wave in lead V1 associated with right axis deviation is seen on electrocardiogram.

Figure 3. Right heart catheterization reveals 203 mmHg pressure gradient between pulmonary artery (PA) and right ventricle (RV).
Calcified giant congenital non-coronary sinus Valsalva aneurysm ruptured into the left ventricular outflow tract

Sol ventrikül çıkış yoluına rüptüre olan konjenital sinus Valsalva anevrizma olgusu

A 42-year-old man with a six-month history of congestive heart failure (NYHA II) was referred to our hospital for evaluation. A calcified giant sinus Valsalva aneurysm (ASV) was detected with transesophageal echocardiography (TEE), computed tomography (CT) and angiography (Fig.1-2, Video 1. See corresponding video/movie images at www.anakarder.com). The aneurysm extended for 8.5 cm in length including the ascending tract, the arch and the descending tract of the aorta. Moreover, severe aortic valve regurgitation and mild mitral and tricuspid regurgitations have been noticed.

During the operation (Fig.3), a 0.3x1.0 cm defect was found in the non-coronary sinus communicated with aneurysm. Degeneration of non-coronary leaflet, presumably caused by aortic regurgitation, was seen. A Gore-Tex patch was used to close the outlet of the aneurysm at the non-coronary sinus. A metallic aortic valve was replaced. Before the weaning of the cardiopulmonary bypass, a severe mitral regurgitation was noticed on the control TEE. Therefore, a metallic

References


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