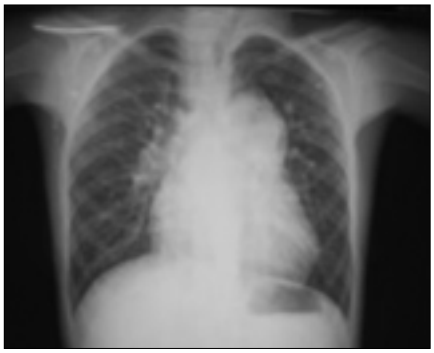
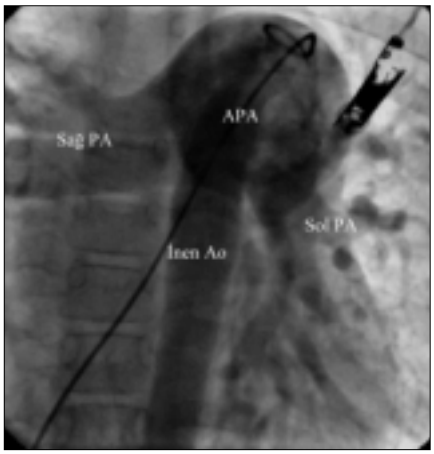
ww.anakarder.com'da izlenebilir). Yapılan sağ ventrikül anjiyografisinde, kontrast maddenin büyük bir VSD aracılığıyla sol ventriküle geçtiği görüldü. Kardiyak basınç ölçümlerinde ise pulmoner arter basıncı 95 mmHg olduğu ve sol ventrikül ile eşitlendiği saptandı.

Halsizlik, çabuk yorulma, taşikardi, takipne ile karşımıza çıkan bu klinik durumda alt ekstremitede oksijen satürasyonu daha düşüktür ve diferansiyel siyanoz izlenir. Eğer tedavi edilmezse %90 hasta ilk bir yıl içerisinde kaybedilir. Tip A aortik enterüpsiyon (IAA) yetişkinlerde nadir karşılaşılan bir durumdur ve hasta asemptomatik olabileceği gibi şiddetli pulmoner hipertansiyon ile Eisenmenger sendromu bulgularıyla karşımıza çıkabilir. Sonuç olarak; IAA oldukça nadir bir anomali olup sıklıkla diğer anomalilerle birlikte seyreder. Erken tanı ve tedavi bu hasta grubunda hayat kurtarıcıdır.

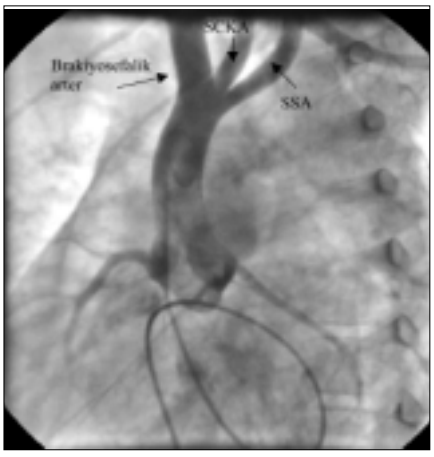


Resim 1. Tip A aortik enterüpsiyona eşlik eden büyük ventriküler septal defekt ve patent duktus arteriyozus olan hastanın teleradyografik görüntüsü



Resim 2. Ana pulmoner arter ve inen aorta devamlılığı

Ao - aorta, APA - ana pulmoner arter, PA- pulmoner arter



Resim 3. Çıkan aorta anjiyografisi

SCKA- sol common karotis arter, SSA- sol subklaviyan arter

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Hypertrophic cardiomyopathy with systolic anterior motion of the posterior mitral leaflet

Hipertrofik kardiyomiyopatide mitral kapağın arka yaprakçığının sistolik ön hareketi

A 63-year-old man with diagnosed hypertrophic cardiomyopathy (HCM) was referred for sudden death risk assessment. The diagnosis was established one year ago in another hospital, where the patient was admitted with dyspnea on exertion. When the patient came to our attention, he complained of mild exertional dyspnea (New York Heart Association functional class II), but without syncope or chest pain. There was no family history of HCM or sudden death. He had neither history of arterial hypertension nor any other systemic disease. The patient's blood pressure was 130/70 mmHg and a grade 3-4/6 systolic ejection murmur was present at the apex with radiation to the lower left parasternal border. The murmur was exaggerated during Valsalva maneuver. Electrocardiogram (ECG) showed a left bundle branch block pattern. A two-dimensional echocardiogram (Fig. 1, Video 1. See corresponding video/movie images at www.anakarder.com) revealed: an asymmetrical type of left ventricular (LV) hypertrophy with maximum wall thickness of 24 mm measured at the anterior ventricular septum; LV end diastolic dimension of 55 mm; left atrium size of 45 mm; and LV outflow obstruction due to systolic anterior motion (SAM) of the posterior mitral leaflet (PML) (Doppler echocardiography estimated 39 mm Hg gradient at rest and 65 mmHg after Valsalva maneuver). The mitral annulus had severe calcification with elongated PML (37 mm) with mild mitral regurgitation. The patient had a normal blood pressure response on exercise test, no episodes of non-sustained ventricular tachycardia in 24-hour ECG recording, and absence of late

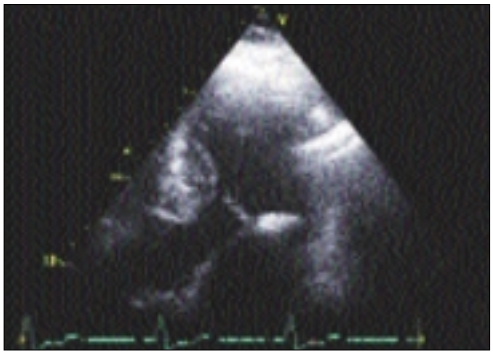

Figure 1. Apical 5-chamber view at mid-systole displaying contact of the posterior mitral leaflet with the anterior septum

hyperenhancement in cardiac magnetic resonance imaging. He was put on β-blockers therapy and after a 3-month follow up period he remained with mild symptoms and no significant drop in LV outflow tract gradient.

Systolic anterior motion selectively of the PML with posterior leaflet-septal contact is not exceptional in generating LV outflow tract obstruction in patients with HCM being identifiable in about 10% of previously studied series of patients (1-3). It is difficult to isolate one causative factor for SAM, given the complex interplay of mechanical and flow factors contributing to it. The initial mechanism proposed for the etiology of SAM was the increased flow velocity and decreased pressure above the valve caused by the hypertrophied interventricular septum, (the Venturi effect) (4). The more recently investigated mechanism concerns the decrease in effective posterior restraint (increased leaflet slack) caused by anterior redirection of papillary muscle tension; increased length of the residual leaflet, which is relatively free to move anteriorly, unlike the coapted leaflet bodies; and interposition of the leaflets into the path of outflow with the potential to cause drag forces (pushing forces of flow) (5, 6).

The present case reinforces the notion that SAM is mostly due to the drag flow phenomenon and not to the Venturi effect. According to Sherrid et al. (7) Venturi flow in the outflow tract cannot be lifting the posterior leaflet because there is little or no area of this leaflet exposed to outflow tract flow and also the PML is shielded and separated from outflow tract flow by the cowl of the anterior leaflet (7). Finally, it should be emphasized that patients with obstructive HCM often have primary structural abnormalities of the mitral apparatus, including displacement of the papillary muscles anteriorly and toward one another with a concomitant anterior shift of the mitral valve, as well as elongated and slack leaflets with altered coaptation (5, 7). These findings suggest that HCM is a disease not only of the muscle, but also of the mitral valve and reinforce the hypothesis that primary changes in the mitral apparatus and, in particular, papillary muscle displacement, can be a primary cause of SAM, independent of septal hypertrophy.

In conclusion, SAM selectively of the posterior mitral leaflet with posterior leaflet-septal contact is not exceptional in generating LV outflow tract obstruction in patients with HCM. Furthermore, it suggests that the drag forces are more important than Venturi effect for causing SAM.

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Unusual late cardiac complication of left pneumonectomy: left atrial compression

Sol pnömonektomiye bağlı gelişen nadir bir komplikasyon: Sol atriyal bası

Postpneumonectomy syndrome is a well-known problem and includes excessive mediastinal shift to the ipsilateral side with bronchovascular compromise and decreased pulmonary reserve due to postoperative hyperinflation of the remaining lung (1). Although left atrial compression caused by thoracic aortic aneurysm has been described previously (2), left-sided pneumonectomy has been reported rarely as etiologic factor for this entity (3).

A 20-year-old man was referred to our hospital for investigation of his telecardiographic changes detected at a military health check-up. He had only mild exertion dyspnea and had undergone a left-sided pneumonectomy as treatment for unilateral bronchiectasis ten years ago. Chest X-ray demonstrated the usual post-pneumonectomy changes of fibrous-tissue-filled opaque with a leftward rotation of the heart axis; the right lung was normal. His pulmonary function was good for a person with one lung. Transthoracic echocardiography showed extrinsic compression of the left atrium by descending aorta in apical four-chamber (Fig. 1A) and parasternal long-axis views (Fig. 1B). Although the mediastinal repositioning with the use of prostheses in some is the therapy of choice, we prefer the conservative approach due to the patient's good functional status. At present, this patient is being followed at the outpatient clinic.

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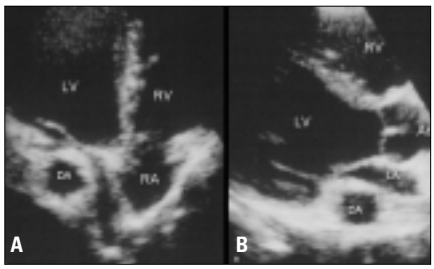

Figure 1. Two-dimensional echocardiography in the apical four-chamber (A) and parasternal long-axis (B) views shows that left atrium is compressed by the descending thoracic aorta

Ao- ascending aorta, DA- descending aorta, LA- left atrium, LV- left ventricle, RA- right atrium, RV- right ventricle