

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 basi

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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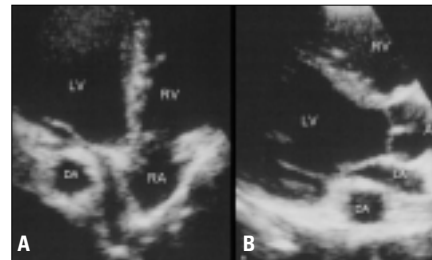


Figure 1. Two-dimensional echocardiography in the apical four-chamber (A) and parasternal long-axis (B) views shows that left atrium is compressed by the descending thoracic aorta

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