Wide splitting of the second heart sound and systolic murmur: Not what you think

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A 20-year-old woman with recent fatigue, weakness of the hands and eyelids, and change in the character of her voice was referred to us for surgical removal of a thymic tumor revealed by means of chest computed tomography. This examination (Figure 1) disclosed a cystic mass measuring 12×5 cm in the anterior mediastinum without hilar, mediastinal, or axillary adenopathy. The acetylcholine receptor antibody level was 14.4 pmol/mL (normal, 0-0.5 pmol/mL). There was no conduction abnormality on the electrocardiogram. Physical examination on admission to our service included auscultation of the heart. The second heart sound (S_2) was widely split, and a significant midsystolic murmur was heard over the base of the heart, especially over the upper left sternal border. These findings prompted an immediate echocardiogram that excluded any heart defect. Surgical intervention was performed through a submammary skin incision and median sternotomy. A well-encapsulated and rigid mass occupying the entire anterior mediastinum was removed. Pathologic examination documented a multicystic mass weighing 465 g, which is consistent with grade B1 cystic thymoma. The operative course was uneventful, and 4 days later, the patient went home. On her outpatient visit 3 weeks later, she reported improvement in her myasthenic symptoms. S_2 was normally splitting, and no murmur was heard.

Figure 1. Chest computed tomographic scan showing a large anterior mediastinal mass (thymoma) compressing the main pulmonary artery at the bifurcation.

Wide splitting of S_2 is caused either by a delay in the pulmonic component of S_2 or by shortening of left ventricular systole with earlier occurrence of A_2 . A survey of the content of 10 different physical examination books revealed the following causes for wide splitting of S_2 : pulmonic stenosis, right bundle branch block, mitral valve regurgitation, and ventricular septal defect. It is not always easy to tell wide splitting from fixed splitting, which is the auscultatory hallmark of atrial septal defect.

Echocardiography performed in our patient excluded conditions associated with wide (or fixed) splitting of S_2 and a systolic murmur, such as valvular pathology (pulmonic stenosis and mitral valve regurgitation) or septal defect (ventricular septal defect or atrial septal defect).

The absence of right bundle branch block and the postoperative disappearance of the auscultatory findings in our patient suggest another mechanism for these findings, namely external compression of the right ventricular outflow tract creating impedance to right ventricular emptying, delaying P_2 , and narrowing the main pulmonary artery, causing a systolic murmur.

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