

Coexistence of discrete subaortic stenosis and unruptured sinus Valsalva aneurysm obstructing the right ventricular outflow tract

Sağ ventrikül çıkım yolunu tıkayan yırtılmamış Valsalva sinüsü anevrizması ile diskret subaortik stenozun birlikteliği

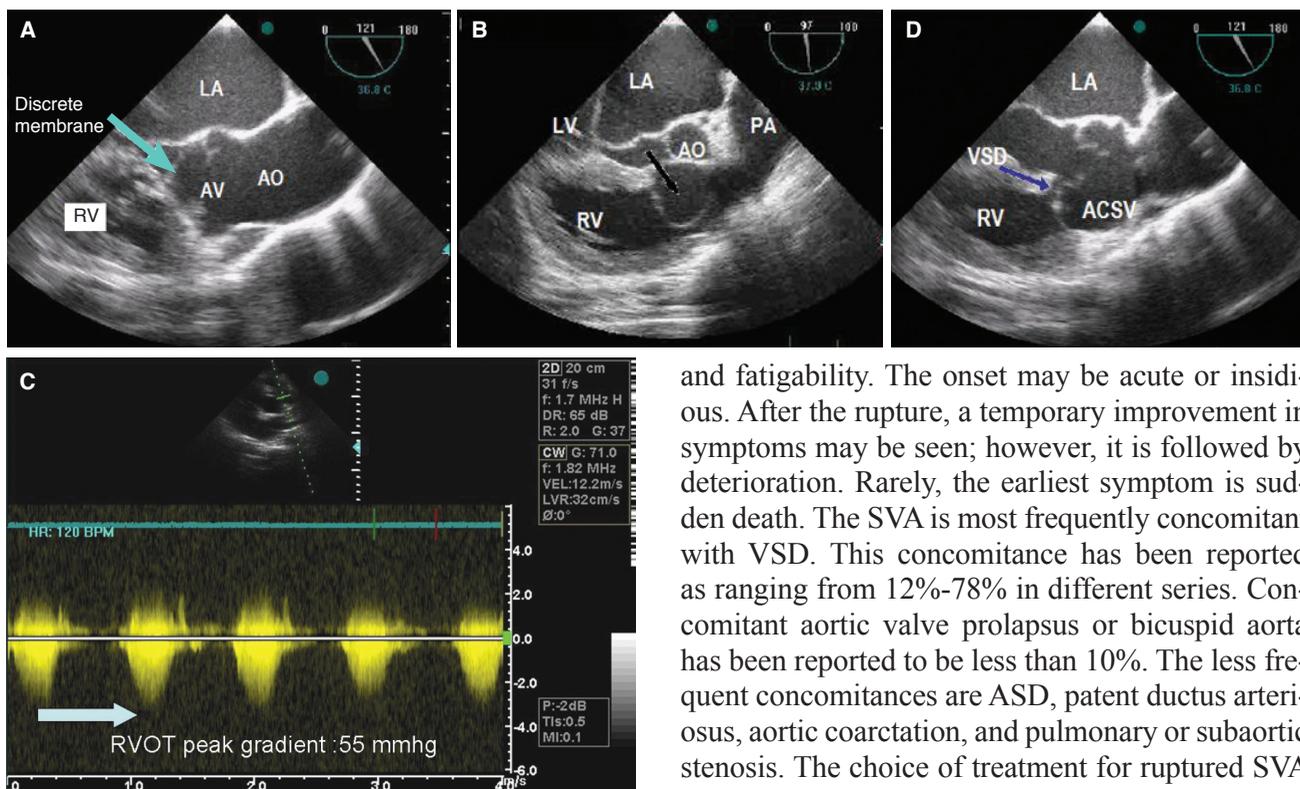
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A 19-year-old male patient was admitted to our department with complaints of exertional dyspnea. The physical examination revealed a grade 3/6 systolic murmur best heard at the mesocardiac area. The electrocardiography showed normal sinus rhythm with left ventricular hypertrophy.

The transthoracic echocardiography (TTE) revealed a biventricular hypertrophy, a discrete subaortic membrane (Fig. A), a 56 mmHg peak gradi-

ent through the left ventricular outflow tract, severe aortic regurgitation, a sinus of Valsalva aneurysm (SVA) protruding to the right ventricular outflow tract (RVOT) (Fig. B), a 55 mmHg peak gradient through the RVOT (Fig. C), and a ventricular septal defect (VSD) adjacent to the SVA (Fig. D). Transesophageal echocardiography confirmed the TTE findings. Sinus of Valsalva aneurysm (SVA) is a rare cardiac entity, occurring in 0.14%–0.96% of patients who have undergone open heart surgical procedures. It is most frequently associated with VSD, atrial septal defect (ASD), bicuspid aortic valve, and aortic coarctation. Unruptured SVA is usually asymptomatic. In ruptured SVA, the major symptoms are coughing, dyspnea, chest pain,



Figures— (A) TEE, parasternal long-axis view, revealing discrete subaortic membrane. (B) TEE showing a sinus of Valsalva aneurysm. (C) Continuous wave Doppler revealing a 55 mmHg peak gradient across the RVOT. (D) TEE showing a VSD adjacent to the sinus of Valsalva aneurysm. LA: Left atrium; RV: Right ventricle; AV: Aortic valve; Ao: Aorta; LV: Left ventricle; PA: Pulmonary artery.

and fatigability. The onset may be acute or insidious. After the rupture, a temporary improvement in symptoms may be seen; however, it is followed by deterioration. Rarely, the earliest symptom is sudden death. The SVA is most frequently concomitant with VSD. This concomitance has been reported as ranging from 12%–78% in different series. Concomitant aortic valve prolapsus or bicuspid aorta has been reported to be less than 10%. The less frequent concomitances are ASD, patent ductus arteriosus, aortic coarctation, and pulmonary or subaortic stenosis. The choice of treatment for ruptured SVA is surgery. If not ruptured, accompanying coronary ostial stenosis, outflow tract obstruction and/or infection are indications for surgical intervention. We recommended surgery to our patient but he declined.