

Case Report

Ventricular Special Defect in the Elderly: An Uncommon Clinical Entity

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Ventricular septal defect (VSD) is the most frequent congenital heart disease in adults, with equal distribution in both sexes, and has an incidence of about 20% in children. In adults congenital VSDs represent about 10% of all cases and the mortality at age 60 is around 75%. For elderly patients >80 years old only one living case has been reported previously. Here we describe an 86-year-old patient, totally asymptomatic, with a muscular type of VSD that showed interesting, canal-like echocardiographic images.

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Ventricular septal defect (VSD) is the most frequent congenital heart disease in adults, with equal distribution in both sexes, and has an incidence of about 20% in children.^{1,2} The most common type (70-80%) is a membranous defect located in the region of the membranous septum, while muscular ventricular defects are responsible for 5-20% of VSDs.^{1,3,4}

It is estimated that 25-40% of VSDs will close by the age of 2 years, while they are unlikely to persist after the age of 10 years.⁵ Thus, in adults congenital heart disease VSDs represent about 10% of the cases.^{1,2} However, the finding of a VSD in an elderly individual >80 years of age is extremely uncommon.

Case report

An 86-year-old patient without predisposing factors for coronary artery disease was referred to our outpatient clinic for evaluation of a systolic murmur. The patient reported having a systolic murmur since childhood with no further evaluation. He did not complain of shortness of breath and he worked as a farmer.

Clinical examination revealed a high-frequency holosystolic 4/6 murmur, mainly audible in the left parasternal region, which was accompanied by a palpable thrill.

Echocardiography showed mild concentric left ventricular (LV) hypertrophy and mild left atrial dilatation. A muscle type VSD, located in the region of the medial intraventricular septum, 10 mm in diameter was apparent (Figure 1) and continuous-wave Doppler showed a high-velocity (maximum 4.83 m/s) left to right signal. There was increased echogenic brightness at the VSD margins, suggesting that processes of fibrosis and calcification had taken place in the region, probably as a result of the increased high velocity blood flow through the VSD (Figure 2).

The pulmonary to systemic cardiac output was found to be Qp/Qs=1.2/1, while tricuspid regurgitation with a maximum velocity of 2.9 m/s was observed. A conservative follow-up was recommended, with prophylaxis against infective endocarditis.

Discussion

The natural history of VSDs depends on the size of the defect and on the pulmonary

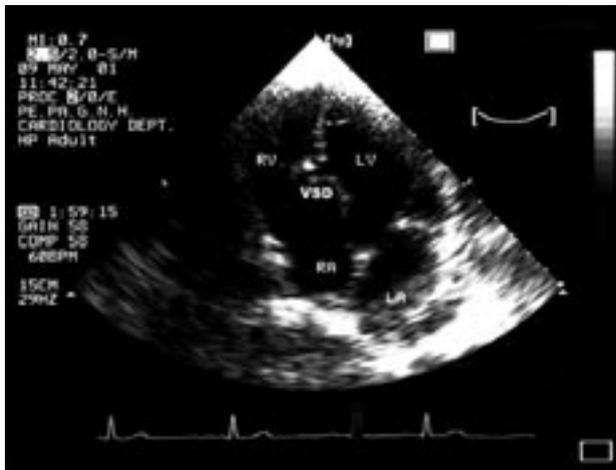


Figure 1. Four-chamber apical view showing a canal-like muscular ventricular septal defect located in the region of the medial intra-ventricular septum.

LV: left ventricle; RV: right ventricle; LA: left atrium; RA: right atrium; VSD: ventricular septal defect.

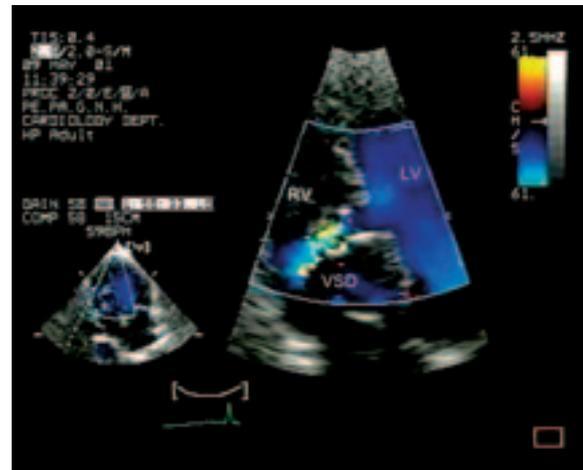


Figure 2. Color Doppler flow showing left-to-right ventricle communication through the muscular ventricular septal defect.

LV: left ventricle; RV: right ventricle; VSD: ventricular septal defect.

resistance. Thus, patients with large VSDs in adult life present with congestive heart failure or pulmonary hypertension and right heart failure, while adults with small defects are usually asymptomatic.

The survival of patients is about 85% at the second decade while the mortality at age 60 is about 75%.⁶ However, evidence in the medical literature concerning elderly patients with VSD is lacking, while for those over 80 years old only two cases^{7,8} have been reported, one as a finding on autopsy.⁷ The present case is the second reported in the medical literature that was discovered in a living patient as an accidental finding.

Also of interest in our case is the hyperechogenic appearance of the VSD boundaries, resembling a “canal”. The increased echogenic brightness of the VSD margins suggests that processes of fibrosis and calcification had taken place in the region, probably as a result of the increased high velocity blood flow through the VSD, although a chronic inflammatory process cannot be ruled out. Only one case has been reported in the literature with VSD calcification⁹ and that was in a 60-year-old patient.

The surgical indications for VSD in adults are large defects with pulmonary to systemic output $Q_p/Q_s > 1.5/1$, pulmonary hypertension > 50 mm Hg, progressive dilatation of the left atrium or the LV, reduced LV function, aortic regurgitation with perimembranous VSD and a history of endocarditis, especially recurrent.¹

Even though the natural history of a small VSD is considered benign, serious complications have been described in these patients, including infective endocarditis,^{10,11} congestive heart failure,¹¹ arrhythmias, and even sudden death.¹⁰⁻¹² In the present case, however, we present a benign form of a relatively small VSD with extended duration of life.

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