LARGE LEFT-TO-RIGHT SHUNT THROUGH A SMALL
ATRIAL SEPTAL DEFECT PRODUCED BY PROGRESSIVE
AORTIC STENOSIS IN THE ELDERLY

— A Case Report —

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We present a 74-year-old female who showed a large left-to-right shunt through a small atrial septal defect presumably due to diastolic dysfunction of the left ventricle caused by the progression of aortic stenosis. Accordingly, elderly patients with even small atrial septal defect should be followed carefully, since diastolic dysfunction of the acquired left ventricular disorder could increase the left-to-right shunt.

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Atrial fibrillation (Af) and tricuspid regurgitation (TR) are two major factors that can aggravate congestive heart failure in patients with atrial septal defect (ASD) of more than moderate size. On the other hand, although a small ASD or a patient foramen ovale are usually not enough to induce congestive heart failure and may not affect life expectancy, they do have some clinical implications; a reverse shunt can develop in acute cor pulmonale, and paradoxical cerebral emboli can occur in young patients who lack risk factors for cerebrovascular accident.

We present a patient who developed a large left-to-right shunt through a small ASD presumably due to the progression of aortic stenosis. Our purpose in this paper is to point out that changes in left ventricular geometry in an elderly patient associated with diastolic dysfunction can influence the severity of left-to-right shunt at the atrial level.

CASE REPORT

A 74-year-old woman was admitted to our hospital with dyspnea on effort which had progressed over the past 2 years. The pulse rate was 70/min with Af, and blood pressure was 100/70 mmHg. The jugular vein was distended with CV wave predominance and the carotid upstroke was delayed. A grade 2/6 mid-systolic murmur was heard along the aortic area and the second heart sound was widely split. There was no diastolic murmur. A large left-to-right shunt pattern was seen on the radiocardiogram. Two-dimensional echocardiography showed marked enlargement of both the right atrium and the right ventricle. The left ventricular wall was diffusely thickened without impairment of wall motion; left ventricular end-diastolic dimension was 35 mm, fractional shortening was 31%, the wall thickness of the left ventricu-
Large Shunt Through Small ASD with AS

Fig. 1. Transthoracic (right) and transesophageal (left) two-dimensional echocardiography each showed a small atrial septal defect with a diameter of about 1 cm. Note that both the right atrium and the right ventricle were markedly enlarged in the four-chamber view obtained by transthoracic echocardiography.

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<th>Pressure Sat.</th>
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Qp:6.2L, Qp/Q:2.6L, Qp/Q=2.6
AVF=135ml, MPG=58mmHg, A/A=0.4cm²

Fig. 2 Data obtained on cardiac catheterization. Upper panel shows the pressure and oxygen saturation ratio of each chamber (AVF=aortic valve flow, MPG=mean pressure gradient, A/A=aortic valve area). Blood pressures in both the left ventricle (LV) and the ascending aorta (Ao) were recorded simultaneously at a paper speed of 10 cm/sec. Note that the left ventricular diastolic decay was depressed.

Lar posterior wall was 11 mm, and the wall thickness of the left ventricular septum was 11 mm. The aortic valves were thickened with marked restriction of movement during systole. The peak velocity of TR was 3 m/sec, and that of the ascending aorta was 5 m/sec. However, a small ASD, maximally about 1 cm in diameter, was seen in both transthoracic and transesophageal color Doppler echocardiograms (Fig. 1).

Cardiac catheterization was performed after diuretics were administered to control congestive heart failure (Fig. 2). Cardiac output (Qs) decreased to 2.6 L/min, and pulmonary flow (Qp) increased to 6.2 L/min. Oxygen-step up was seen at the atrial level and Qp/Qs was 2.6. Levophase pulmonary arteriography revealed that four pulmonary veins normally drained into the left atrium. A catheter could be inserted into the left atrium and left ventricle through the ASD, and the pressures in both the left ventricle and the ascending aorta were measured simultaneously; the aortic valve area was 0.4 cm². Pressure decay in the left ventricle was markedly prolonged, but left atrial pressure and left ventricular end-diastolic pressure were not increased markedly.

We performed a direct closure of an ASD
(10×8 mm) of the central fossa ovalis type, and replaced the aortic valve. Postoperatively, the radiocardiogram revealed no shunt pattern and the size of the right atrium and the right ventricle diminished markedly.

**DISCUSSION**

In this case, Qp/Qs, as calculated by oximetry, was 2.6 at the atrial level although the ASD was small. Four pulmonary veins were observed to drain into the left atrium normally by levophase pulmonary arteriography and postoperative radiocardiography verified that the shunt had disappeared. Therefore, this left-to-right shunt had occurred through this small ASD. Severe aortic stenosis was present; the mean pressure gradient across the aortic valve was 58 mmHg in the presence of low output status. The calculated area of the aortic valve was 0.4 cm². However, the left atrial and left ventricular end-diastolic pressures were not significantly increased. If this small ASD were to be closed by a balloon, the left atrial and left ventricular end-diastolic pressures might have increased, since a large volume of left atrial blood may have entered the left ventricle directly instead of entering the right ventricle.

When ASD is complicated by mitral stenosis in Lutembacher syndrome, a left-to-right shunt can increase relative to the size of the hole because left atrial pressure can rise according to the severity of mitral stenosis. In this case, left atrial pressure was 5 mmHg, which was insufficient to account for this amount of shunt.

Left ventricular diastolic function can be impaired with the preservation of systolic function when the left ventricular wall becomes thickened. This diastolic dysfunction may be expressed by a prolongation of isovolumic relaxation time and a delayed deceleration of the rapid filling phase in the transmitral flow velocity pattern, which means that the left ventricle becomes less compliant. In this case, a thickened left ventricular wall was observed on echocardiography and a marked depression of diastolic decay was seen on the left ventricular pressure tracing. However, left atrial pressure was within normal limits, presumably due to the low output status. In addition, the right ventricular end-diastolic pressure was decreased to as low as 1 mmHg. Therefore, a large volume of left atrial blood traveled to the more compliant right ventricle via this small ASD.

Aging per se can stiffen the left ventricle. Moreover, chronic hypertension and/or the progression of aortic stenosis can lead to thickening of the left ventricle, causing diastolic dysfunction.

We conclude that elderly patients with ASD should be followed carefully, even if the defect is small, since a left ventricular disorder can develop concomitantly, resulting in an increase in the left-to-right shunt.

**REFERENCES**

2. CASE RECORD OF MASSACHUSETTS GENERAL HOSPITAL. N Engl J Med 1985; 313; 1003–1012
3. HAUSMANN D, MUGGE A, BECHT I, DANIEL WG: Diagnosis of patient foramen ovale by transesophageal echocardiography and association with cerebral and peripheral embolic events. Am J Cardiol 1992; 70; 668–672