A Case of Atrial Septal Defect Complicating Myocardial Infarction

Severe Heart Failure Induced by Balloon Occlusion of ASD

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SUMMARY

A 58 year old man with an acute, extensive anterior myocardial infarction was found to also have an atrial septal defect secundum. A cardiac catheterization study showed a large left to right shunt (Q_p/Q_s : 6.0). Since other clinical findings were not consistent with the prolonged existence of a large shunt, we considered it to be secondary to the myocardial infarction. Balloon occlusion of the ASD was carried out to predict the hemodynamics after the surgical correction, which soon resulted in severe left ventricular failure. Therefore, an operation was not feasible in this case.

Additional Indexing Words:

Atrial septal defect secundum (ASD) Myocardial infarction
Balloon occlusion Left ventricular failure Bezold-Jarisch
reflex

AN atrial septal defect secundum (ASD) comprises about 10% of all congenital heart diseases, and the symptoms are not obvious until after the third decade.¹⁾ The prognosis of ASD is influenced mainly by organic obstructive changes in the pulmonary artery, reflected as the degree of the pulmonary hypertension.²⁾⁻⁵⁾ However, ASD patients are susceptible to the unfavourable complications which may result in abrupt hemodynamic changes. Myocardial infarctions in patients with ASD are not uncommon, but clinical reports concerning this combination are quite rare. This paper presents an ASD patient with a large left to right shunt, complicated by an extensive anterior myocardial infarction.

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CASE REPORT

A 58 year old man had been healthy until the morning of January 23, 1984 when he was admitted to a hospital due to an acute myocardial infarction. After 1 month, a bicycle ergometer stress ECG showed no significant ST-T changes at a 75 watt work load and he was discharged. He was readmitted to the hospital on March 13 with a second myocardial infarction. During the second admission, a left ventricular aneurysm and ASD were suspected by echocardiography. After 6 weeks, he was admitted to our hospital for further examination.

He was 156 cm tall, weighed 49 kg, his pulse was regular at a rate of 60/min and his blood pressure was 106/74 mmHg. The external jugular veins were slightly distended, but hepato-jugular reflux was not observed. Lungs were clear. While a grade 3 systolic murmur was maximal at the fifth intercostal space of the left sternal border, a grade 1 diastolic rumbling murmur was audible at the third and fourth intercostal space of the left sternal border. Neither hepato-splenomegaly nor edema was found. All deep tendon reflexes

Fig. 1. Electrocardiograms showing extensive anterior myocardial infarction with incomplete right bundle branch block. Note rsR pattern in V₂ lead in the first attack.

were intact and there were no pathological reflexes.

Urinalysis, blood cell counts and blood chemistry (including CPK) were normal. A chest X-ray showed a moderate cardiomegaly (60% in CTR). The main pulmonary artery was enlarged at its proximal portion and pulmonary were prominent in the upper lung fields. A resting electrocardiogram showed a regular sinus rhythm, IRBBB with the QRS axis of +135 degrees and an abnormal Q wave in I, aV_L and V₁-V₆ (Fig. 1). A phonocardiogram showed fixed splitting of the second sound with an accentuated pulmonary component. The jugular pulse tracing revealed a prominent peaked v wave. The PEP/ET was 0.446, indicating decreased cardiac performance. The echocardiogram showed dilation of the right atrium and ventricle and paradoxical motion of the interventricular septum. The transverse diameters of the left atrium, aortic root and right ventricle were 3.8, 3.2 and 3.3 cm, respectively. A negative jet during diastole in the right atrium was demonstrated by contrast echocardiography. Nuclear medical techniques supported the diagnoses of ASD and extensive anterior myocardial infarction.

Cardiac catheterization confirmed the ASD and revealed an extraordinarily massive left-to-right shunt (Q_p/Q_s : 6.0) and large pulmonary flow (Q_p : 17.5 L/min). The pulmonary to systemic vascular resistance ratio (R_p/R_s) was as low as 0.03, implying that there were only minimal organic changes in pulmonary vasculature. The coronary angiogram showed 99% stenosis at the proximal portion of the left anterior descending artery, which was supposed to be responsible for the myocardial infarction. The left ventriculogram showed akinesia of the antero-lateral wall of the left ventricle. Balloon occlusion of the atrial septal defect was performed to predict the hemodynamic changes during surgical correction of the ASD. A 22 Fr. Forgaty balloon catheter was inflated with a normal saline solution containing contrast medium. Complete closure of the defect required 40 ml of normal saline. From this volume, the diameter of ASD was estimated to be around 4.2 cm. Following the occlusion, the left ventricular end-diastolic pressure (LVEDP) increased from 11 mmHg to 40 mmHg and the mean pulmonary arterial pressure (mPAP) from 19 mmHg to 42 mmHg (Fig. 2). However, cardiac output increased from 2.92 L/min to 4.52 L/min. An atrial kick, which was not seen before occlusion, appeared on the left ventricular pressure tracing after occlusion. Because the patient complained of intolerable chest oppression and dyspnea 5 min after occlusion, the balloon was deflated and the symptoms disappeared. Bradycardia (rate: 33/min) appeared soon after occlusion, but the heart rate recovered gradually to 40/min. From these results, surgical correction of the atrial septum was considered to be contraindicated. Thus, medical therapy with diuretics, long acting nitrates and Ca-

Fig. 2. Simultaneous pressure tracing of left ventricle and pulmonary artery before and immediately after the balloon occlusion of atrial septal defect. Note the elevation of LVEDP, PAP and mPAP after occlusion. An atrial kick appeared after occlusion, suggesting a difficulty with flow into the left ventricle. LV=left ventricle; PAP=pulmonary arterial pressure; LVEDP=left ventricular end-diastolic pressure; mPAP=mean pulmonary arterial pressure.

antagonists was continued.

DISCUSSION

Most symptoms of atrial septal defect (ASD) may be due to volume overload of the right side of the heart, which usually evolves into pulmonary hypertension and right heart failure.¹⁾⁻³⁾ Factors influencing the compliance and/or contractility of both ventricles should alter the hemodynamics of the ASD and eventually change its prognosis.^{6), 7)} Clinical reports on the occurrence of myocardial infarctions in patients with ASD are rare.⁸⁾ In this case, cardiac catheterization showed low vascular resistance, suggesting that the pulmonary vasculature was not affected by the organic changes. Given the patient's age of 58 years and the large left to right shunt rate, it is strange that the ECGs showed a regular sinus rhythm and that the pulmonary resistance was within normal limits. Therefore, this high pulmonary flow may be a secondary change due to left ventricular failure after the myocardial infarction. Since the left ventricle cannot pump as much blood in the systemic circulation as previously, a certain amount of blood will be shunted into the right atrium during diastole. Balloon occlusion of an atrial septal defect

has been applied to select ASD and hypoplastic heart syndrome patients for operations.^{9),10)} This procedure in the present case was soon associated with dyspnea, chest pain and oppression. In 5 min, he could no longer stand the examination, and all the hemodynamic parameters except cardiac output worsened. We tried to decrease the venous return by tilting the bed by 30 degrees, but the result of the test was almost the same. The closure of ASD would have improved the hemodynamics of this patient, provided the blood volume had been optimized by venesection. But there was not enough time to control the blood volume before the development of the hemodynamic derangement. Thus, it was concluded that surgical correction of ASD was not feasible. The patient showed bradycardia during occlusion, while the systemic blood pressure fell slightly. It was presumed that blood flow into the left ventricle after the occlusion may have expanded the ventricular wall, especially the posterior wall, resulting in the phenomenon called the Bezold-Jarisch reflex.¹¹⁾

An increasing number of patients suffering from myocardial infarction may inevitably involve ASD. However, few reports of this complication have been published. This is probably the first report of a clinical case of myocardial infarction which occurred in a patient with ASD; however, 2 autopsied cases have been reported.⁸⁾ In this present case, balloon occlusion of ASD was performed to predict the hemodynamic changes after surgery and immediately resulted in severe left heart failure. Thus, the ramifications of surgical closure of ASD complicated by myocardial infarction should be carefully assessed.

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