SPONTANEOUS CLOSURE OF VENTRICULAR SEPTUM RUPTURE AFTER ACUTE MYOCARDIAL INFARCTION

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Spontaneous closure of ventricular septal defect due to acute anterzor myocardial infarction was demonstrated angiographically 20 months later in a 55-year-old female patient.

FECHAMENTO ESPONTÂNEO DE COMUNICAÇÃO INTERVENTRICULAR APÓS INFARTO AGUDO DO MIOCÁRDIO

Mulher de 55 anos apresentou infarto agudo do miocárdio de parede anterior e ruptura do septo interventricular, o qual fechou espontaneamente. O fechamento foi demonstrado angiograficamente depois de 20 meses do infarto.

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The rupture of the ventricular septum occurs in less than 1% of patients with fatal acute myocardial infarction[1]. The clinical outcome is directly related to the size of ventricular septal defect and to the impairment of right ventricular function that is responsible for the left-to-right shunt and hemodynamic compromise[2]. In general, patients with left intraventricular septum rupture usually present loud holosystolic murmur with a left sternal border thrill. The diagnosis is made with two-dimensional and Doppler echocardiography[3], and occasionally with Swan-Ganz catheterization that discloses a higher right ventricular oxygen content. The surgical intervention is the treatment of choice and the surgical result is better when correction is delayed a week or longer[4]. We present the case of a patient who had a spontaneous closure of ventricular septum defect after an acute anterior myocardial infarction probably related to a left ventricular thrombus.

CASE REPORT

A 55-year-old woman was admitted to the hospital with a 8 years history of atypical chest pain. There was a history of discrete systemic arterial hypertension and **diabetes mellitus** non-insulin-dependent type. On examination, the patient appeared well. The blood pressure was 160 x 100 mmHg, the pulse was 70 and the temperature was normal. The lungs were clear. The heart was normal and abdominal examination was negative. The liver and spleen were not felt. The electrocardiogram and the exercise tolerance test were normal. The glucose was 168 mg/dl, cholesterol/136 mg/dl and creatinine 0.9 mg/dl. The patient was kept on treatment of diabetes and hypertension. Three years later, on June 18, 1980, after a precordial pain of one hour duration, a cardiac catheterization was done. It disclosed normal left ventricular enddiastolic pressure and left ventricular angiographic examination showed muscular hypertrophy. The coronary angiography showed a proximal 40% stenosis in the left anterior descending artery. On August 26, 1985, the patient was admitted to the hospital with one hour chest pain. On examination, the patient appeared well. The blood pressure was 170 x 95 mmHg and the pulse was 76. Lungs were clear. The heart examination disclosed a thrill at left sternal border and a moderate holosystolic murmur. The electrocardiogram showed anterior myocardial infarction with ST segment elevation and T-wave inversion. On January 1, 1986, a cardiac catheterization disclosed a left ventricular end-diastolic pressure of 20 mmHg. The left ventricular angiography showed anterior dyskinesis with mural thrombus and a left-to-right shunt of small size through muscular septum (fig. 1). The coronary arteriography showed a 40% stenosis in left anterior descending artery. On June, 1986, the heart

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examination was normal, without systolic murmur. On April 30, 1987, after another prolonged typical chest pain, a cardiac catheterization was done and the left ventricular angiographic examination showed again the anterior dyskinesis with mural thrombus and closure of ventricular septal defect (fig. 2). The coronary arteriography was unchanged. On April 11, 1990, the heart examination was still normal, without systolic murmur.

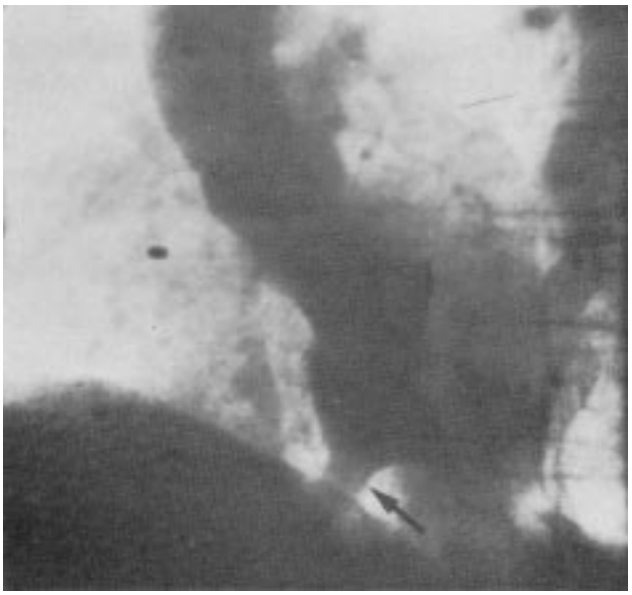

Fig. 1—Left ventricular anglography in the 60 degree left anterior oblique projection shows the ventricular septal defect (arrow).

Fig. 2—Left ventricular angiography in the 60 degree left anterior oblique projection without ventricular septal defect.

DISCUSSION

An intraventricular septum rupture is a mechanical complication that may occur after an acute myocardial infarction, specially in anterior infarction on elderly patients with systemic arterial hypertension. The size of the defect and the right ventricular function determine the extent of hemodynamic deterioration, and on patients with large size defects imediate surgical repair is indicated[5]. The use of aortic balloon pump is the first necessary step if patient condition permit before surgical treatment[6]. The small size defect can wait several days for surgical treatment so that healing to a certain degree may occur, but spontaneous closure of ventricular septal defect after an acute myocardial infarction is rare, and in this patient it has probably occurred after the interaction of at least three theoretical factors[7-9]: 1) a small and irregular rupture tend to reduce blood velocity across the defect and the blood stasis favors better conditions for coagulation system.; 2) the closure relation between ventricular septum defect and mural thrombus may permit progress of the thrombus within septal defect; 3) the left anterior dyskinesis, changing the left ventricular geometry, may produce an atenuation of pressure gradient between the compromised left ventricular segment and right ventricle, whitc is probably less that the relationship of left ventricle with normal contraction and the right ventricle. The interaction of these three factors may explain the spontaneous closure of ventricular septal defect after an acute myocardial infarction.

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