Traumatic Rupture of Interventricular Septum Proved by Cardiac Catheterization

By Scott R. Inkley, M.D., and Frank M. Barry, M.D.

A rare complication of trauma to the heart is rupture of the interventricular septum. The diagnosis of an acquired lesion of the septum can be reasonably made from history and cardiac catheterization and early recognition is of importance if immediate surgical correction is indicated.

Rupture of the interventricular septum due to trauma is a rare phenomenon, according to Pollock, Markelz and Shuey, who reviewed all the reported cases up to 1952 and found a total of 12 proven traumatic interventricular defects. This report concerns a patient who developed an interventricular septal defect following severe trauma to the chest that was proven by cardiac catheterization.

Case Report

A 38-year-old white man was admitted to the University Hospitals of Cleveland after the milk delivery truck that he was driving was struck by another car. The patient had been crushed against the chest-high center platform of his truck by cases of milk falling from a shelf behind him. The blow was sudden and compressed the left chest anteroposteriorly.

On his admission, the pulse was 104, the respirations were 24 and the blood pressure was 104/68; he was in obvious respiratory distress from multiple rib fractures of the left chest. The right chest was clear to percussion and auscultation, and the left chest revealed hyperresonance to percussion, decreased breath sounds and tactile fremitus. Subcutaneous emphysema was present in the left axilla and neck. Except for multiple lacerations and abrasions of the face and extremities, no other serious injuries were noted.

The patient's early hospital course was marked by chest pain, dyspnea and cyanosis, but by the fifth day in the hospital there was marked improvement and on the eighteenth day the patient was discharged. Adequate auscultation of the left chest anteriorly was not possible, because of pain and a traumatic pneumothorax, until the patient returned to his surgeon in a follow-up visit, at which time a loud systolic murmur was noted over the precordium. The heart was not enlarged to percussion, and a loud, blowing systolic murmur was heard best in the fourth interspace just to the left of the sternum. The murmur was transmitted to the cardiac apex and base.

A tentative diagnosis of traumatic interventricular septal defect was made, and the patient was admitted to the hospital for cardiac catheterization. The results of catheterization are seen in table 1; they show a pulmonary artery pressure at the upper limits of normal and definite evidence of a left-to-right shunt. This was considered confirmatory evidence of an interventricular septal defect. In establishing the development of the defect as secondary to the trauma sustained in the automobile accident, the results of 2 pre-employment physical examinations and of a selective service physical examination made prior to the accident were considered. In each instance, the heart was described as normal. It is improbable that a murmur of such intensity as the present one would be missed during even the most cursory examination. Following his discharge from the hospital the patient complained of some dyspnea on exertion, and chest pain associated with breathing. These symptoms gradually disappeared during the course of the next 6 months, and approximately 9 months after his injury he returned to work as a milkman in a rural community. His activity is somewhat restricted by fatigue, but he has done extremely well. He complains of no dyspnea with normal exertion. Electrocardiographic studies 18 months after the injury showed no significant change and a chest x-ray taken at the same time showed no evidence of cardiac enlargement. The auscultatory findings remained essentially unchanged during this period.

Discussion

Experimental studies on laboratory animals with the use of blunt force on the heart have
TRAUTOMATIC RUPTURE OF INTERVENTRICAL SEPTUM

Table 1.—Cardiac Catheterization Studies

<table>
<thead>
<tr>
<th>Location</th>
<th>Pressure (mm Hg)</th>
<th>Oxygen content (vol. %)</th>
<th>Cardiac output (L/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulmonary artery</td>
<td>30/15 mean 24</td>
<td>13.0</td>
<td></td>
</tr>
<tr>
<td>Right ventricle</td>
<td>30/0 mean 15</td>
<td>14.0</td>
<td>7.3</td>
</tr>
<tr>
<td>Right atrium</td>
<td>7/3</td>
<td>11.7</td>
<td></td>
</tr>
<tr>
<td>Inferior vena cava</td>
<td>11.7</td>
<td>11.6</td>
<td></td>
</tr>
<tr>
<td>Superior vena cava</td>
<td></td>
<td>16.1</td>
<td></td>
</tr>
<tr>
<td>Femoral artery</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left ventricle</td>
<td></td>
<td>4.4</td>
<td></td>
</tr>
<tr>
<td>Right-to-left shunt</td>
<td></td>
<td>2.9</td>
<td></td>
</tr>
</tbody>
</table>

Oxygen consumption 220 ml/min.

shown that rupture is most likely if the heart is struck late in diastole or early in systole.2 The simple mechanics of rupture dictate that it would be most likely to occur when the ventricles are full and when the intraventricular pressure would rise highest with sudden compression of the ventricular walls. If the mitral and tricuspid valves are in the closed position, there is less chance for relief of pressure into the atria and the great veins. Presumably, the impact in this patient occurred when the ventricles were full, and the sudden elevation of pressure caused a rent in the septum that has been maintained by the normal pressure differential between the right and left ventricle. Other injuries that are likely with nonpenetrating trauma to the heart are rupture of the ventricular wall into the pericardial sac, fracture of mitral or tricuspid valves, or tearing of the chordae tendineae. Contusion of the ventricular muscle may also cause changes similar to myocardial infarction.3 Electrocardiographic studies after injury may reveal evidence of muscle damage or pericardial irritation. In this instance electrocardiographic studies were not made during the acute stage of the illness. No significant limitation of physical activity has yet been necessary in this patient, and there has been no objective evidence of cardiac enlargement by x-ray or by electrocardiogram. Operative repair of the defect is contemplated, but the absence of symptoms, the relatively low pulmonary artery pressure and the normal electrocardiogram make it reasonable to postpone surgery for the present.

Traumatic rupture of the septum has been rarely reported but will be likely to appear more commonly as the result of more frequent use of the cardiac catheter. Since Pollock and co-workers4 report of 12 cases in the literature in 1952, 1 case proven by cardiac catheterization,4 1 by autopsy5 and 1 by surgical repair have been noted. Recognition of this defect is important because of the availability of surgical correction in those cases where a large shunt may be responsible for pulmonary vascular disease and cardiac failure.

Summary

A case of traumatic rupture of the interventricular septum proved by cardiac catheterization is presented. The patient has returned to his occupation of milkman without symptoms or signs of cardiac embarrassment. Surgical repair of this defect will be considered in the future.

SUMMARIO IN INTERLINGUA

Es presentate un caso de ruptura traumatic del septo interventricular, demonstrate per catheterismo cardiac. Le patiente ha retornate a su occupation de lactero, sin symptomas o signos de embarasso cardiac. Le possibilitate de reparar le defecto per intervention chirurgic va esser considerate in le futuro.

REFERENCES


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