Perforation of the Interventricular Septum in a 71-Year-Old Woman; Successful Repair Nine Days following Acute Myocardial Infarction

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We report a 71-year-old woman who underwent successful repair of a perforation of the interventricular septum while in shock and pulmonary edema, nine days following acute myocardial infarction. This case illustrates that a bold surgical approach during the acute phase of myocardial infarction, even in the elderly patient in severe pump failure, is the treatment of choice in this condition.

Since the first case of surgical repair of perforation of the interventricular septum following myocardial infarction was described by Cooley et al in 1957, numerous additional cases have been reported in the literature.2-12 Only a small number of these cases have been operated on during the acute stage of myocardial infarction, and of these, most have not survived the immediate postoperative period. Patients above the age of 70 operated on for this complication have rarely been reported. The purpose of this report is to describe the case of a 71-year-old woman who underwent successful repair of perforation of the interventricular septum while in shock and pulmonary edema, nine days following acute myocardial infarction.

CASE REPORT

This 71-year-old housewife had been well except for a duodenal ulcer 20 years previously. Ten months prior to this admission, she underwent a cataract extraction and her electrocardiogram was normal at that time. She had always been physically active and habitually performed vigorous morning exercises. On the morning of her admission, she felt pain in the epigastrium, but performed her usual exercises in spite of this and then walked to the market where she suddenly collapsed. She was brought to the emergency room of Shaare Zedek Hospital unconscious, with blood pressure 80 mm Hg systolic and pulse rate 75 per minute. Electrocardiogram showed signs of an acute anteroseptal infarction (Fig 1). Examination after transfer to the medical ward revealed a grade 3/6, somewhat coarse systolic murmur at the upper left sternal border and in the aortic area, and a few rales over the left lung base. Her blood pressure fell to 60 mm Hg systolic, with central venous pressure 17 cm water. She was treated with an intravenous infusion of norepinephrine initially; her blood pressure rose and she became conscious. Subsequent treatment included intravenous digitalis and hydrocortisone in large doses (a total of 4 grams during a 12-hour period) and signs of shock gradually disappeared, although she remained anuric for 24 hours. Serum enzymes the next morning showed lactic dehydrogenase level over 2000 units, glutamic oxaloacetic transaminase 50 units and glutamic pyruvic transaminase 65 units. These levels gradually fell over the next few days. The blood urea rose to 71 mg percent on the third day, but subsequently returned to normal. Serial electrocardiograms confirmed the presence of acute anteroseptal infarction.

From the second to the sixth day after admission, her condition was good; there was no sign of congestive failure and after the second day she excreted adequate amounts of urine. On the sixth day, the systolic murmur, which had been present since admission, was noted to be louder and coarser, was maximal at the second to third left intercostal space, and a systolic thrill appeared in the same area. During the next 24

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Figure 1. Electrocardiogram on admission.

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Pulmonary artery

day after admission the patient was in frank pulmonary edema, and in that condition she was transferred to the Hadassah University Hospital with the presumptive diagnosis of perforated interventricular septum.

The patient was taken immediately to the catheterization laboratory where right and left heart catheterization were performed. The findings (Table 1) indicated the presence of a left-to-right shunt at the ventricular level and biventricular failure with pulmonary hypertension. Left ventricular injection of contrast medium showed moderately good left ventricular contractility. A high ventricular septal defect and a small aneurysm of the outflow tract of the right ventricle were visualized.

Following catheterization, the patient's blood pressure fell to 60 mm Hg systolic (direct intra-arterial reading), there were signs of poor peripheral perfusion, mental confusion and gasping respiration. Isoproterenol infusion produced only partial improvement and raised the systolic pressure to 80-90 mm Hg. It was obvious that emergency closure of the septal defect was her only chance of survival, and in this condition she was brought to the operating room. Under general anesthesia the chest was rapidly opened via midsternotomy incision. On opening the pericardium, a marked thrill was palpable over the right ventricle. The myocardium was grey-blue in color, consistent with infarcted muscle, and bulged outwards prominently with each ventricular contraction. A large fibrotic scar was noted over the apex of the left ventricle. Severe hypotension and bradycardia supervened at this stage necessitating rapid cannulation of the heart and commencement of the cardiopulmonary bypass. Extracorporeal circulation was maintained by the Rygg-Kvysgaard disposable bubble oxygenator* and moderate hypothermia. Following crossclamping of the ascending aorta, the outflow tract of the right ventricle was opened longitudinally. The muscle in this area was extremely thin and of the consistency of wet blotting paper. A defect of the upper part of the ventricular septum very close to the anterior surface of the heart was apparent. It appeared as if the ventricular septum in this region had burst open causing an oblique tunnel through the septum, extending from fairly low down in the left ventricle to its termination, high and anterior in the right ventricle not far from the pulmonary valve (Fig 2). The muscle surrounding the defect was friable and of yellowish hue. The defect was closed in two layers by interrupted mattress sutures of 3-0 silk tied over Teilon felt buttresses (Fig 3). The incision in the right ventricle was closed. No thrill was now palpable over the right ventricular outflow tract. The heart beat in sinus rhythm throughout and maintained an adequate pressure following cessation of the extracorporeal circulation, without the addition of cardiotonics. The patient was fully awake at the completion of the procedure. The endotracheal tube was left in place and controlled intermittent positive pressure respiration was continued for 18 hours after the operation.

The postoperative course was surprisingly smooth. The pulmonary edema cleared rapidly as did all other signs of congestive cardiac failure. The coarse systolic murmur over the heart heard previously had disappeared. The blood urea levels gradually returned to normal. On follow-up examination three months after the operation, the patient was well, there were no signs of congestive heart failure, and only a short, grade 2/6 ejection murmur was heard over the aortic area.

**DISCUSSION**

Perforation of the interventricular septum accounts for 1-2 percent of deaths following acute myocardial infarction and approximately 20 percent of all perforations of the heart. In the vast majority of cases it is a lethal complication unless surgically repaired. The first reported case of such a surgical repair was by Cooley in 1957, the operation being performed five weeks after the acute infarction. Since then an increasing number of patients have undergone surgery for this complication. To date, a total of 98 patients have been reported in the English literature who have been operated on for a post-

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**Table 1—Cardiac Catheterisation Findings**

<table>
<thead>
<tr>
<th>Pressure</th>
<th>O₂ Saturation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Superior vena cava</td>
<td>59, 63</td>
</tr>
<tr>
<td>Right atrium</td>
<td>v=15, m=10</td>
</tr>
<tr>
<td>Right ventricle</td>
<td>65/15</td>
</tr>
<tr>
<td>Pulmonary artery</td>
<td>60/20</td>
</tr>
<tr>
<td>Pulmonary capillaries</td>
<td>v=13, m=8</td>
</tr>
<tr>
<td>Left ventricle</td>
<td>130/15-20</td>
</tr>
<tr>
<td>Aorta</td>
<td>140/85</td>
</tr>
</tbody>
</table>

Pulmonary: Systemic blood flow = 1.5:1

*Polystan, Copenhagen, Denmark.*

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**SCHEMATIC DIAGRAM OF OPERATIVE FINDINGS**

Figure 2. Schematic diagram of operative findings.
have been reported who have undergone surgical repair of a perforated interventricular septum. To our knowledge only two patients over 70 have been operated on during the first 15 days after the acute infarction, and neither of these survived. This case thus further illustrates that a bold surgical approach to this complication during the acute phase of myocardial infarction, even in the elderly patient in severe pump failure, can be successful.

An additional feature of this case worth noting is the unusual location of the perforation of the septum. Only 4 percent of perforations of the interventricular septum are located in the upper portion of the septum. This location was responsible for the unusual site of the murmur and thrill, which were maximal in the second to third left intercostal spaces in this patient. It also dictated the surgical approach via the outflow tract of the right ventricle, in preference to left ventriculotomy which is the approach currently recommended for the more common type of perforation of the septum.

REFERENCES

Recurrent Pleural Effusion Associated with Dysgamma Globulinemia*

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A 45-year-old man presented with recurrent bilateral pleural effusions of undetermined etiology. This was associated with absence of IgM and a decreased IgA fraction. Possible associations are discussed.

A variety of pathologic conditions, particularly of the respiratory tract, have been described in association with specific immunoglobulin abnormalities. These include pneumonia, sinusitis, bronchitis, bronchiectasis, unilateral hyperlucent lung and emphysema. To date, however, there has been no reported instance of a pleural effusion in association with an immunoglobulin deficiency alone. A patient is presented who developed recurrent pleural effusions in association with absence of IgM and a reduction in the IgA fraction.

Case Report

This 45-year-old man was admitted to the Naval Hospital, Portsmouth, Virginia in February 1969 for evaluation of a right pleural effusion noted on an annual chest roentgenogram. Several weeks previously he had experienced an acute episode of "influenza" characterized by chills, fever, myalgia, and a productive cough. Results of intermediate strength tuberculin and histoplasmin skin tests were negative. Thoracentesis yielded 300 ml of straw-colored fluid, and cytology for malignant cells was negative. Specific cell counts were not obtained. Cultures of the pleural fluid for routine pathogens and mycobacteria were negative. Bronchoscopy provided normal findings. A tube thoracostomy was performed because of reaccumulation of the effusion. In April, 1969, a diagnostic right thoracotomy with decortication of the right lower lobe was performed. Pleural cultures revealed a subacute fibrous pleuritis. Histologic examination revealed the very porous sequestrum, the pleura, and the abnormal changes in the serous membrane, the pleura, and the abnormal changes in capillary dynamics. Increased capillary permeability, increased capillary pressure, decreased plasma colloid osmotic pressure, or blockage of the lymphatics may play the etiologic role in producing a pleural effusion. Diseased states associated with abnormal immunoglobulins are well recognized as causative factors of pleural reac-

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