Ventricular Septal Defect and Ventricular Aneurysm Secondary to Acute Myocardial Infarction

Report of Four Cases with Successful Surgical Treatment

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SUMMARY

Four cases of ventricular septal defect and ventricular aneurysm after myocardial infarction are reported. The clinical findings and surgical results in these four cases and in eight previously reported patients with this combination of complications are summarized. Resection of the ventricular aneurysm and closure of the ventricular septal defect appear indicated, both on theoretical grounds and in view of the excellent results observed in six patients who underwent combined repair. In one of our patients, closure of the ventricular septal defect and resection of the ventricular aneurysm were possible with only a single suture line, as these defects were anatomically contiguous.

Additional Indexing Words:
Coronary artery disease  Myocardial infarction, complications  Open heart surgery

Better diagnostic techniques have led to increased recognition of two complications after myocardial infarction: ventricular septal perforation and ventricular aneurysm. Surgical repair has become more frequent as the risk involved with intracardiac surgery has decreased.

Nine patients with this combination of defects after acute myocardial infarction have been reported by other investigators.1-9 All of these patients underwent closure of the ventricular septal defect, and four underwent resection of the ventricular aneurysm as well.

This paper reports four additional cases of this combination of complications, and describes the first case (case 2, table 1) in which the ventricular septal defect and the ventricular aneurysm were anatomically contiguous. Because these defects were contiguous, we were able to repair both of them simultaneously, using only a single suture line (fig. 1).

Report of Cases

The pertinent data concerning the four patients we are reporting are shown in table 1. Roentgenograms of the thorax in all patients showed cardiomegaly and pulmonary vascular congestion, and one patient had right pleural effusion. Three patients underwent preoperative catheterization, but a fourth patient (case 1) was operated upon with a clinical diagnosis of mitral insufficiency, whereas a normal mitral apparatus and a ventricular aneurysm and ventricular septal defect (VSD) were actually discovered at operation. In all the catheterized patients the pulmonary flow was high and the pulmonary resistance was low; pulmonary hypertension was present.

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VSD AND VENTRICULAR ANEURYSM

(A and B) Inferior and anterior views, respectively, of heart, showing juxtaposed ventricular aneurysm and ventricular septal defect. (C) Aneurysmal ventricular wall has been resected. Margins are being reapproximated, with simultaneous obliteration of interventricular communication by a single row of simple interrupted sutures. Abbreviations: A = aorta; IVC = inferior vena cava; LA = left atrium; LV = left ventricle; RA = right atrium; RV = right ventricle; VSD = ventricular septal defect.

Discussion

Our four patients were similar in many respects to nine patients, previously reported by other investigators, who had a combination of ventricular septal defect and ventricular aneurysm after myocardial infarction. Because the clinical information about one of these patients was limited, we have not included him in the following discussion. The clinical presentation in the 12 patients with adequate description was related to the ventricular septal defect and the sudden development of congestive heart failure. While most patients developed symptoms early, one did not become symptomatic until 3 months after myocardial infarction.

Rapid deterioration in the clinical state was associated with the sudden appearance of a holosystolic murmur along the lower left sternal border in all 12 patients, and with a systolic thrill in the same location in 10 patients. The clinical differentiation of ventricular septal defect from mitral valve insufficiency is often difficult, as seen in our case 1, and right heart catheterization is, therefore, indicated for diagnosis prior to operation. In all patients the left-to-right shunt was large, with pulmonary/systemic flow ratios of 2.0 to 5.5. Mean pressure in the pulmonary artery was elevated in all but one patient.

The presence of a ventricular aneurysm was suspected clinically in only three patients preoperatively. Persistent ST-segment elevation is common early following uncomplicated myocardial infarction and does not necessarily indicate ventricular aneurysm formation. In six patients the ventricular aneurysm was first identified at operation, while in the remaining
three patients the diagnosis was made at autopsy; in these three patients the ventricular aneurysm was located on the inferior aspect of the left ventricle and may have been overlooked at operation.

There was good correlation between the electrocardiographic site of myocardial infarction and the location of the ventricular aneurysm as seen at operation. In seven patients the myocardial infarction and the ventricular aneurysm were located on the inferior wall, while in five patients they involved the anterior or anterolateral aspect of the left ventricle. The ventricular aneurysms varied in size from $3 \times 4$ cm to $6 \times 10$ cm.

Six of the eight patients in whom both abnormalities were known to be present at operation underwent combined repair. In five of these patients the ventricular aneurysm was first excised; the ventricular septal defect was then closed by either direct suture or patch graft, and, finally, the left ventricle was reapproximated. In one patient (our case 2) it was possible both to close the defect in the inferior portion of the interventricular septum and to reapproximate the free wall of the right and left ventricles with a single suture line, as the defects were contiguous.

All six patients survived operation and were discharged from the hospital. Four of these patients were alive 22 weeks to 15 months postoperatively. The fifth patient died suddenly 7 months postoperatively, and the sixth died suddenly with severe chest pain 8 months after operation.

In contrast, surgical morbidity and late mortality rates were greater in the five patients in whom only the ventricular septal defect was closed. Four of these patients died late after operation, and only one was alive 32 weeks postoperatively. Of these four patients, one patient died in severe congestive heart failure 6 weeks postoperatively, and three patients died suddenly, 32 days, 12 weeks, and 8 months, respectively, after operation. Autopsy in these three patients revealed severe coronary artery disease without evidence of recent myocardial infarction. The presumed mechanism of death in each case was fatal arrhythmia. Ventricular tachyarrhythmias may be associated with ventricular aneurysms.11–14 Whether such a mechanism was operative in the three patients who died suddenly in this group is not known.

Table 1

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Age at operation</th>
<th>Sex</th>
<th>Events associated with, and after, myocardial infarction</th>
<th>Interval between infarction and operation</th>
<th>ECG findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>47</td>
<td>M</td>
<td>Multiple dysrhythmias, CHF; development of murmur on second day</td>
<td>10 weeks</td>
<td>Ant. infarction with ↑ ST, V₄ through V₆</td>
</tr>
<tr>
<td>2</td>
<td>64</td>
<td>M</td>
<td>Acute LV failure; systolic thrill; murmur at LLSB; palpable P₁ day of infarction</td>
<td>12 weeks</td>
<td>Infarction, inferior wall</td>
</tr>
<tr>
<td>3</td>
<td>60</td>
<td>M</td>
<td>Uncomplicated; development of CHF 3 weeks later when murmur and thrill noted</td>
<td>20 weeks</td>
<td>Anteroseptal infarction with ↑ ST precordial leads</td>
</tr>
<tr>
<td>4</td>
<td>70</td>
<td>M</td>
<td>Tachycardia with infarction; murmur noted 7 years later</td>
<td>8 years</td>
<td>Anteroseptal infarction with ↑ ST in V₁ through V₄</td>
</tr>
</tbody>
</table>

Abbreviations: CHF = congestive heart failure; ↑ ST = ST-segment elevation; VSD = ventricular septal defect; ant. = anterior; p.o. = postoperative; LV = left ventricular; LLSB = lower left sternal border; post. = posterior.
Resection of a ventricular aneurysm has been considered of benefit in three patients in controlling recurrent ventricular tachycardia. The energy-dissipating effect of areas of ventricular asynery or aneurysm formation warrants resection of such areas if identified at operation.

Surgical intervention in patients with post-myocardial infarction ventricular septal defects is justified in view of the high mortality figures (13% of 91 cases survived 2 months) in the medically treated group. Recurrent or persistent defects are common after closure of post-infarction ventricular septal defect, so surgical technique should be precise and secure. Use of a patch of prosthetic material is usually required, and, in fresh infarcts, the patch should overlap the margins of the defect by several millimeters. The overlap allows the sutures to be placed in tissue which is as firm as possible.

The ideal time for repair of a ventricular septal defect after myocardial infarction would appear to be 3.5 to 6 months on the basis of the work of Mallory and associates on myocardial wound healing. Collagen fibers reached a maximal density by 8 weeks after myocardial infarction, but were not sufficiently strong to hold suture lines consistently until after 3.5 to 6 months. Nevertheless, earlier operation may become mandatory in some patients because of clinical deterioration despite intensive medical therapy. Four of the 12 patients considered here were operated on during the first 2 months following myocardial infarction. Two of these died late after operation, and autopsy revealed small (1.5 to 2 mm) residual ventricular septal defects of little clinical significance. The other two patients had uneventful postoperative courses following repair of both the ventricular septal defect and the ventricular aneurysm.

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