Intrapericardial Abnormalities in Patients with Pericardial Effusion

Findings by Two-dimensional Echocardiography

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AND RICHARD L. POPP, M.D.

SUMMARY Ten patients with pericardial fluid who also had striking band-like intrapericardial echoes by two-dimensional ultrasonic sector scanning are reported. Four patients had prior mediastinal radiation and four patients had severe renal disease. One patient had purulent pericarditis and one patient had traumatic hemopericardium. The two-dimensional images permitted recognition of loculation of fluid and led to the suspicion of thickened pericardial membranes. Three postradiation patients had both tamponade and constriction; tamponade alone was present in four additional patients. These data are preliminary but suggest that such findings by two-dimensional echocardiography should alert us to consider an effusive-constrictive form of pericardial disease.

Echocardiography has proved extremely useful as a noninvasive method for diagnosing pericardial effusion. M-mode echocardiography can reliably establish the presence of pericardial effusion, but the narrow field of view and lack of spatial orientation of echoes make assessment of the size and distribution of the fluid difficult. Wide-angle, two-dimensional echocardiography provides dynamic images of the myocardium and pericardium in multiple cross-sectional planes, permitting assessment of the size, distribution and postural shift of the fluid within the pericardial space. In this article, we describe adhesive or fibrinous pericardial bands producing loculation of pericardial fluid, pericardial thickening, and a probable intrapericardial thrombus recognized during echocardiographic studies in 10 patients with suspected or proved pericardial effusions.

Methods

Patients

Ten patients studied over 30 months in the Stanford Medical Center Echocardiographic Laboratory for suspected pericardial effusions had echo-producing intrapericardial structures on the original examination (not seen in other patients with pericardial effusions). The 10 patients ranged in age from 17–56 years. Pericardial effusion was suspected on the basis of their clinical situation and chest x-ray. The 10 patients reported presented within three general clinical settings (table 1).

Four patients had oncologic processes and had received anterior mediastinal radiation, in excess of 5000 rads, from 1–2 years before the echocardiographic evaluation. Two patients had oat cell carcinoma and two patients had Hodgkin’s lymphoma. All four patients had attempted pericardiocentesis for suspected cardiac tamponade before their two-dimensional echocardiographic examinations. In three

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of the four patients the pericardiocentesis yielded a small amount of fluid, with minimal symptomatic relief. All three were sent to the echocardiographic laboratory for two-dimensional echocardiographic assessment of suspected loculated effusion after their pericardiocenteses. The fourth patient had multiple pericardiocenteses with recurrent accumulation of pericardial fluid. A second group of four patients had advanced diabetic renal disease with renal failure. Only one of these four patients had pericardiocentesis, but all patients were known to have had repeated bouts of pericarditis or pericardial effusion or both. The remaining two patients had acute processes: One had documented pneumococcal pericarditis and the other had a stab wound involving the left pleural and pericardial space. The patient with pneumococcal pericarditis had had pericardiocentesis for diagnosis and relief of symptoms, with a scant yield of fluid. The patient with chest trauma responded to insertion of a chest tube and did not require pericardiocentesis.

Study Procedure

Two-dimensional echocardiographic studies were obtained with a prototype or production model phased-array sector scanner (Varian V-3000) or a mechanical sector scanner (Smith Kline Instruments EkoSector I). The examination procedure was similar to that described by Kisslo et al. The transducer was initially placed in the parasternal position and the image plane oriented so that the viewed plane was approximately parallel with the long axis of the left ventricle. By rotating the transducer 90° clockwise, short-axis images of the heart and pericardial space were obtained sequentially from base to apex of the heart. Next, the transducer was placed over the cardiac apex with the patient in a steep left lateral decubitus position. The plane of the beam was initially oriented perpendicular to the interventricular septum so all four chambers of the heart were imaged simultaneously. Then, the transducer beam was angled toward the patient’s left hip until the apical portion of the pericardial space was imaged. By rotating the transducer 90° counterclockwise and parallel to the interventricular septum, the echocardiographic beam could be angled medially to visualize the posteromedial and anteromedial recesses of the pericardial space, and laterally to visualize the posterolateral and anterolateral recesses of the pericardial space. Finally, with the patient supine, the transducer was placed in a subxiphoid or right subcostal position for special attention to the pericardial space anterior to the right ventricular free wall. All patients were studied with the head elevated 30–90° from the horizontal plane. Specific attention was paid to the anatomy of the pericardial space, the distribution of the pericardial effusion around the heart, and the presence or absence of echo-producing structures within the pericardial space.

Results

Pericardial effusion was identified as a relatively echo-free space outside the myocardium in all 10 patients with the two-dimensional echocardiographic studies. The parietal pericardium was defined as an intense echo that remained after altering the transmitted ultrasonic energy level, as in the M-mode technique. Pericardial fluid volume was estimated as moderate (100–500 ml) to large (> 500 ml) in all 10 patients.

Table 1. Patient Data

<table>
<thead>
<tr>
<th>Pt</th>
<th>Presumed etiology</th>
<th>Tamponade</th>
<th>Constrictive hemodynamics</th>
<th>Pathology</th>
<th>Pleural effusion</th>
<th>Figure</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Radiation</td>
<td>+</td>
<td>+</td>
<td>NA</td>
<td>R, L</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Radiation</td>
<td>+</td>
<td>+</td>
<td>VP 2–4 mm</td>
<td>R, L</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>PP 8 mm</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Fibrous debris and fluid in sac*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Radiation</td>
<td>+</td>
<td>—</td>
<td>NA</td>
<td>R, L</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Radiation</td>
<td>+</td>
<td>+</td>
<td>Thick pericardial layers*; 500 ml fluid</td>
<td>R, L</td>
<td>1, 2</td>
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<tr>
<td>5</td>
<td>Renal disease</td>
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<td>NA</td>
<td>NA</td>
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<td></td>
</tr>
<tr>
<td>6</td>
<td>Renal disease</td>
<td>—</td>
<td>NA</td>
<td>NA</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Renal disease</td>
<td>—</td>
<td>NA</td>
<td>NA</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>Renal disease</td>
<td>+</td>
<td>—</td>
<td>NA</td>
<td>R</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>Purulent</td>
<td>+</td>
<td>—</td>
<td>VP fibrotic* PP 2–8 mm 700 ml fluid</td>
<td>R, L</td>
<td>3</td>
</tr>
<tr>
<td>10</td>
<td>Traumatic</td>
<td>+</td>
<td>—</td>
<td>NA</td>
<td>L</td>
<td>4</td>
</tr>
</tbody>
</table>

*Comment from surgery or pathology report.

Abbreviations: + = present; — = absent; R = right; L = left; NA = not available; VP = visceral pericardium; PP = parietal pericardium.
The four patients who had undergone anterior mediastinal radiation exhibited dense linear echoes traversing the pericardial space. These structures appeared connected to both the visceral and parietal pericardium and divided the pericardial space into compartments (fig. 1). One of these patients had a set of echoes in the most apical portion of the pericardial space. This structure had an undulating motion synchronous with the cardiac cycle and was not adherent to the visceral pericardium (fig. 2). This type of motion of the pericardial bands was commonly seen. Repeat recording in these four patients performed 2 or more minutes after the patients had assumed a sitting position showed no noticeable alteration in the distribution of the pericardial fluid compared with the supine position. The lack of fluid redistribution and pericardial segmentation by these bands of echoes suggested loculated effusion in these four patients. All four of these patients had bilateral pleural effusions as well.

The four patients with chronic renal failure also exhibited moderate-to-large pericardial effusions, as well as dense echoes bridging the pericardial space. There was no alteration in the apparent distribution of the pericardial effusion in these four patients when they were moved from a supine to a sitting position. Right-sided pleural effusion was present in only one of these patients.

The patient with the pneumococcal pericarditis had not only linear structures bridging the visceral and parietal pericardium, but also an apparent marked thickening of the visceral pericardium over the ventricular apex with multiple "shaggy" trailing edges (fig. 3). On the basis of the clinical setting and the two-dimensional echocardiographic evaluation, the patient underwent surgical pericardiectomy. At surgery, a friable, granular, fibrinous exudate was found on the visceral pericardium. Additionally, the parietal pericardium was 2–8 mm thick and multiple fibrinous bands extended into the pericardial space. The remaining patient had undergone a stab wound to the left lower thorax. Before a chest tube was inserted for pleural drainage, the patient had a moderate pericardial effusion and a homogeneous echo-producing structure in the diaphragmatic aspect of the pericardial space by two-dimensional echocardiography (fig. 4). Because of the patient's stable clinical status, pericardiocentesis was not performed. Subsequent imaging over 3 days showed no alteration in either the size of the pericardial effusion or in the intrapericardial echo. The latter was thought to represent an intrapericardial thrombus.

Discussion

M-mode echocardiography is unquestionably a useful technique for detecting pericardial effusions, but wide-angle, two-dimensional echocardiography
allows better recognition of the distribution of fluid and other structures within the pericardial sac. We do not attempt to compare M-mode and two-dimensional echocardiography in this paper, as two-dimensional methods were preferentially used in these patients due to their clinical situations. Pericardial thickening and dense intrapericardial echoes are recognizable on M-mode records taken from the base of the heart in the standard way. However, it is difficult to distinguish fluid loculation or intrapericardial bands with confidence on M-mode tracings. Recognition of intrapericardial bands may be important clinically in patients with a history of chronic or recurrent pericarditis in whom pericardiocentesis is a mainstay of management. Two-dimensional echocardiography may detect pericardial loculation before pericardiocentesis in this group of patients. In the presence of multiple intrapericardial bands, absence of positional fluid shifts may suggest fluid loculation. Placing the ultrasound transducer in the parasternal, subxiphoid and apical positions provides optimal assessment of possible compartmentalization of the pericardial fluid and may also aid in planning needle placement for pericardiocentesis to achieve maximum fluid yield. Patients suspected of having large pericardial effusions in whom initial pericardiocentesis yields little fluid may benefit from two-dimensional echocardiographic identification of pericardial loculation due to multiple bridging bands.

The majority of patients described in this report had either oncologic disease that required anterior mediastinal radiation, or renal failure, with multiple episodes suggesting pericarditis or pericardial effusion. This implies that chronic and recurrent pericardial processes are most likely to develop adhesive or fibrinous pericardial bands. We are interested in the high prevalence of pericardial constriction and tamponade in these patients (table 1), because this may be important for prospective recognition of these hemodynamic states, and adds to the increasing appreciation of an effusive-constrictive form of pericardial disease. The combination of thickened pericardial membranes and enclosed fluid around the heart noted by Hancock is marked in this group of patients diagnosed by echocardiography. Pericardial effusions are often seen in patients with heart failure due to valvular heart disease or coronary artery disease, but we have not seen pericardial bands as described here in such patients. Two-dimensional echocardiography may be useful in evaluating patients with pericardial trauma in whom hemopericardium is suspected and in assessing possible complications of cardiac catheterizations, suspected coronary artery disease during pericardiocentesis and in patients seen after chest trauma.

A potential problem of interpretation should be mentioned. Pleural effusion was common in our patients (table 1). Patients with both pericardial and left pleural effusion may present an echocardiographic picture in which the parietal pericardium appears to be a band-like membrane spanning the fluid (fig. 1).
This may be distinguished from intrapericardial bands if the parietal pericardium is thin and fixed, because most of the pericardial bands in our patients had an undulating motion during the cardiac cycle.

Awareness of the pericardial abnormalities described here may allow correlation with specific pathologic findings by investigators in the future. For the present, these findings may improve understanding and management of patients with chronic recurrent pericardial disease.

References

Abnormal Blood Pressure Response and Marked Ischemic ST-segment Depression as Predictors of Severe Coronary Artery Disease

MIGUEL E. SANMARCO, M.D., STEVEN PONTIUS, M.D., AND RONALD H. SELVESTER, M.D.

SUMMARY The usefulness of an abnormal blood pressure response and a marked ischemic ST-segment depression during exercise testing as predictors of severe coronary artery disease was assessed in 378 consecutive patients who had a maximal symptom-limited exercise test before coronary arteriography. An abnormal blood pressure response occurred in 90 patients. The sensitivity of this response for three-vessel or left main disease was 38.6%, the specificity 87.4% and the predictive value 70%. A marked ischemic ST-segment abnormality (MIST) appeared in 85 patients. The sensitivity of MIST for three-vessel or left main disease was 38.6%, the specificity 89.8% and the predictive value 74.1%. One hundred thirty-eight patients had either an abnormal blood pressure response or a marked ST-segment change. The sensitivity of either response for three-vessel or left main disease was 56.4%, the specificity 78.6%, and the predictive value 66.7%. Exercise duration and ejection fraction were not significantly different in patients with normal or abnormal blood pressure. We conclude that abnormal blood pressure and marked ischemic ST-segment depression during exercise testing are helpful in identifying a subset of patients with advanced coronary artery disease. The physiologic mechanism for these responses is probably exercise-induced ischemia.

EXERCISE TESTING is probably the most widely used method for identifying patients with probable coronary artery disease. Numerous parameters of stress testing may predict not only the presence of coronary artery disease, but also its functional significance and risk for the patient. The predictive value of ST-segment changes and its dependency on the type and degree of ST-segment abnormality have been reported. In addition to ST depression, the presence of exercise-induced ventricular arrhythmias, a change in the amplitude of the R wave and a fall in blood pressure below resting levels appear to be reliable predictors of severe coronary disease. The significance of decreasing systolic blood pressure during exercise has been the subject of three recent reports. Thomson and Kelemen and more recently, Morris and co-workers have suggested that exercise-induced hypotension is highly specific for multiple-vessel coronary disease. However, Levites et

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