Extrinsic Nonvalvular Mitral Obstruction due to Large Epicardial Hematoma*

Arvind Kumar Shah, M.D.; David Melarkey; and Vincent T. Ness, M.D.

A case of extrinsic nonvalvular mitral obstruction due to a large epicardial hematoma in a patient with rheumatoid constrictive pericarditis is described. The patient had longstanding rheumatoid arthritis and a mitral diastolic murmur developed. Mitral obstruction was confirmed by a hemodynamic study. Coronary angiography and left ventricular angiography showed severely diseased coronary arteries and a distorted left ventricular cavity. Autopsy demonstrated rheumatoid arthritis, a normal mitral valve, thickened pericardium, and epicardial hematoma surrounding both ventricles at the atrioventricular junctional level.

(W)hile many causes of nonvalvular mitral obstruction are reported in the literature,1-3 obstruction of the mitral orifice with normal mitral valve leaflets due to epicardial hematoma pressing on the atrioventricular ring and left ventricle has not been described (to our knowledge). The following case report illustrates such a nonvalvular mitral obstruction in a patient with rheumatoid constrictive pericarditis.

Case Report

This 57-year-old, retired quality controller for a grinding shop was admitted to Reno VA Medical Center on August 1, 1989 for increasing fatigue and hip pain of 6 months' duration. He had been under treatment at the medical center for 23 years for crippling rheumatoid arthritis, receiving aspirin, nonsteroidal anti-inflammatory analgesics, gold, and steroids at different times. Other treatments included digoxin, furosemide, and potassium chloride supplements. He underwent multiple surgical procedures to improve the function of his joints, including bilateral hip arthroplasty, synovectomy of both wrists and knees, and Swanson prosthesis for proximal interphalangeal joints of the left fingers after removing nodules. Pathologic studies of synovial tissues confirmed the diagnosis of rheumatoid arthritis. His personal and family histories were unremarkable.

Clinical examination revealed a well-built, well-nourished man with multiple deformities of peripheral joints and deformed shape of his chest. He had a regular pulse of 65/min, blood pressure of 160/60 mm Hg, temperature of 36.8°C and respiratory rate of 18/min. Results of examination of lungs, abdomen, and nervous system were normal. He had no visible or palpable apical impulse. The first and second heart sounds were normal; a mitral opening snap and a localized mitral diastolic rumble were heard. The complete blood cell count included a hematocrit of 51.2, hemoglobin of 16.0 g/dl, WBC count of 6,200/cm3 and platelet count of 168,000/cm3. Chemistry was normal except for mild hypokalemia. The chest deformity prevented the interpretation of the chest radiograph except for possible cardiomegaly and clear lung fields. The electrocardiogram showed sinus rhythm with a heart rate of 64/min, right axis deviation, relatively low-voltage QRS, and nonspecific T-wave.

*From the Departments of Medicine (Dr. Shah) and Anatomy and Pathology (Dr. Melarkey), University of Nevada School of Medicine, Reno, and the University of California, San Francisco, and VA Medical Center, Fresno, Calif (Dr. Ness).

Reprint requests: Dr. Shah, VA Medical Center, Reno, Nevada 89520

Figure 1. Left ventricular angiogram in right anterior oblique view. Abnormalities in all leads. His echocardiogram was uninterpretable. The patient underwent cardiac catheterization on October 15, 1989 for more complete evaluation, as a part of preoperative workup for total hip replacement. The mean right atrial pressure was 17 mm Hg with A and V wave pressures of 22 and 24 mm Hg, respectively. Right ventricular and pulmonary arterial pressures were 36/20 and 36/20 (mean, 26) mm Hg respectively. The mean pulmonary capillary pressure was 25 mm Hg. Aortic and left ventricular pressures were 120/70 (mean, 86) and 120/22 mm Hg, respectively. There was a gradient of 8 mm Hg across the mitral valve with cardiac index of 2.64 L/mm/m2. The calculated mitral valve area was 1.6 cm2. There was no gradient across aortic, tricuspid, and pulmonary valves or detectable evidence of left to right shunt. Hemodynamic interpretation was consistent with mitral obstruction and uneven constrictive pericarditis or restrictive cardiomyopathy. The coronary cineangiograms showed 100 percent occlusion of the right coronary artery and 75 percent obstruction of an oblique marginal branch of the circumflex artery. The left ventricular cineangiograms showed a bullerina shape of the cavity and ejection fraction of 85 percent (Fig 1). Cineortogram showed mild aortic regurgitation. Total hip replacement was considered undesirable in view of the probable high mortality. The patient died 3 months after hospital admission.

Figure 2. Epicardial hematoma below right and left ventricle. Artificial cleft is seen on right side of hematoma.
Orifice. Hemodynamic study was consistent with uneven pericardial constriction (Table 1). Even though diastolic pressures of all chambers and pulmonary artery were elevated, equalization of pressures of left- and right-sided chambers and among pulmonary artery, right atrium, and right ventricle was not seen. The pressure waveform of the right ventricle did not show typical features of constrictive pericarditis. It is very probable that fibrocalcific pericardium localized the hematoma and allowed the pressures to be transmitted through the mitral annulus and obstructed mitral orifice. While the cause of a hematoma is unclear, vasculitis associated with a malignant type of rheumatoid arthritis and platelet dysfunction due to aspirin therapy may have contributory roles. No other mechanism can explain the findings in our case.

To our knowledge, there has been no report documenting findings similar to our case. In a large series of constrictive pericarditis from South Africa, cases of constrictive pericarditis with localized fibrotic annular constriction, producing valvular obstruction of one or more of the four valvular orifices, including the mitral, have been documented. Even though echocardiographic and autopsy studies of rheumatoid arthritis have shown frequent involvement of pericardium, its clinical recognition is infrequent. Constrictive pericarditis with cardiac tamponade due to hemopericardium, obstruction of the tricuspid orifice due to localized hematoma, and valvular obstructions due to rheumatoid valvular nodules have been reported. The diastolic mitral murmur in a patient with rheumatoid constrictive pericarditis can be due to mitral annular constriction, mitral valvular nodules, and an epicardial hematoma compressing the left ventricle at the atroventricular level as in our patient.

**References**


**Table 1—Cardiac Pressures**

<table>
<thead>
<tr>
<th>Pressures, mm Hg</th>
<th>Mean</th>
<th>Systolic</th>
<th>Diastolic</th>
<th>A wave</th>
<th>V wave</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right atrium</td>
<td>17</td>
<td>. . .</td>
<td>22</td>
<td>24</td>
<td></td>
</tr>
<tr>
<td>Right ventricle</td>
<td>0</td>
<td>.</td>
<td>.</td>
<td>36</td>
<td>20</td>
</tr>
<tr>
<td>Pulmonary artery</td>
<td>26</td>
<td>36</td>
<td>20</td>
<td>. .</td>
<td>.</td>
</tr>
<tr>
<td>Pulmonary capillary (wedged)</td>
<td>25</td>
<td>. . .</td>
<td>.</td>
<td>.</td>
<td>.</td>
</tr>
<tr>
<td>Aorta</td>
<td>86</td>
<td>120</td>
<td>70</td>
<td>. .</td>
<td>.</td>
</tr>
<tr>
<td>Left ventricle</td>
<td>. .</td>
<td>120</td>
<td>20</td>
<td>. .</td>
<td>.</td>
</tr>
</tbody>
</table>