Hemodynamic Features of Prolapsing and Nonprolapsing Left Atrial Myxoma

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SUMMARY

In the course of the evaluation of five patients with left atrial myxoma, it was noted that the movement of the myxoma was related to specific changes in left atrial hemodynamics. Prolapsing tumors, Type I, move from the left ventricle to the left atrium in early systole and from the left atrium to the left ventricle in early diastole, thereby causing prominent c and v waves accompanied by a rapid y descent. Nonprolapsing tumors, Type II, remain in the left atrium during the entire cardiac cycle, impeding flow across the mitral valve. In these latter cases, the y descent is slow and indistinguishable from that caused by mitral valvular stenosis. The cineangiocardiograms and echocardiograms corroborate these two types of hemodynamic observations. The particular value of direct echocardiographic examination of the left atrium prior to cardiac catheterization was evident in two of the three patients with nonprolapsing tumors. Since the hemodynamic pattern of nonprolapsing left atrial myxoma resembles that of mitral valvular stenosis, it is stressed that echocardiography should have an important place in precatheterization assessment of patients with mitral valve disease. If left atrial myxoma is suspected clinically or on the basis of echocardiographic findings, regardless of the pressure curve contours, transseptal cardiac catheterization should be avoided and the left atrium visualized by pulmonary angiography levophase.

Additional Indexing Words:
Cardiac catheterization
Echocardiography
Cardiac tumors
Suprasternal ultrasonography
Cineangiocardiography

INCREASING CLINICAL AWARENESS of intracardiac tumors has been stimulated by the improved diagnostic techniques of cardiac catheterization and the more recent advent of echocardiography. Nevertheless, left atrial myxoma continues to be a diagnostic challenge because of its variable clinical presentation.

Several excellent reports dealing with the unique hemodynamic events and phonocardiographic features of left atrial myxoma have appeared in the literature.1-6 A characteristic left atrial and/or pulmonary wedge pressure pulse consisting of prominent c and v waves with a rapid y descent has been considered to be diagnostic of the presence of left atrial myxoma.5,7,9 Our experience in five patients with this disease has enabled us to classify two distinct alterations in left atrial hemodynamics with cine-angiocardiographic and echocardiographic correlation. The purpose of this paper is to define the underlying mechanisms of the hemodynamic alterations and to correlate these physiological changes with the mobility variations found in left atrial myxoma.

Materials and Methods

Between 1967 and 1973, five patients with left atrial myxoma were encountered at Jackson Memorial Hospital. All were female, four Caucasian and one Negroid (case 4). Their ages ranged from 37 to 66 years. Their clinical profiles are summarized in table 1. One patient (case 1) has been reported fully elsewhere.5 The diagnosis of left atrial myxoma was suspected clinically prior to cardiac catheterization in two patients (cases 4 and 5). The other three patients were initially suspected to have bacterial endocarditis (case 1) and rheumatic mitral valvular stenosis (cases 2 and 3). In each case, the tumor was surgically removed. At surgery, the mitral valve was found to be normal in all except one (case 3) whose mitral valve was thickened and fibrotic but without microscopic evidence of a rheumatic process. The operative findings are included in table 1. All patients survived surgery except for one (case 3) in whom a fatal cerebral vascular accident occurred in the immediate postoperative period.

Right and left heart catheterization was performed in all patients. The left atrium was entered via transseptal technique in one patient (case 2), and inadvertently by a retrograde catheter from the left ventricle in two patients (cases 3 and 5). Simultaneous pressures were recorded using
### Table 1

**Summary of the Five Cases of Left Atrial Myxoma**

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Sex</th>
<th>Age</th>
<th>Previous cardiovascular disease</th>
<th>Presenting symptomatology</th>
<th>Positive physical findings</th>
<th>Pertinent laboratory data</th>
<th>Operative findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F</td>
<td>66</td>
<td>None</td>
<td>Anorexia, weight loss, intermittent low grade fever, and orthopnea for six months duration, transient right hemiparesis.</td>
<td>Anemic, systolic and diastolic murmurs of variable intensity, accentuated first heart sound and presence of a diastolic sound.</td>
<td>Hb 9.7, Hct 32, Ret 6, Sed rate 71, Coombs test positive, elevated gamma globulin.</td>
<td>Normal mitral valve. A pedunculated, irregularly shaped mass of 5 x 3 x 3.5 cm in size attached to the lower part of the left atrial septum.</td>
</tr>
<tr>
<td>2</td>
<td>F</td>
<td>53</td>
<td>None</td>
<td>Transient left hemiparesis, acute onset of congestive heart failure.</td>
<td>Slightly accentuated first and second heart sounds. Presence of a diastolic sound.</td>
<td>Hb 13.0, Hct 35, Ret 1.5, Sed rate 40, Coombs test negative, elevated gamma globulin.</td>
<td>Normal mitral valve. A multilobar mass of 5 x 4 x 4 cm in size with a short pedicle attached to the left atrial wall near the entry of right pulmonary vein.</td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>57</td>
<td>None</td>
<td>Profound weakness, intermittent atrial fibrillation for 20 years, transient left facial paresis and dysarthria.</td>
<td>Atrial fibrillation with a Grade 3/6 systolic murmur at the apex.</td>
<td>Hb 14.7, Hct 42, Ret 1.2, Sed rate 20, normal serum protein electrophoresis.</td>
<td>Mitral valve fibrotic and thickened without commissural fusion. A 5 x 4 x 3 cm roughly round, largely calcified mass with a short pedicle attached to the left atrial wall.</td>
</tr>
<tr>
<td>4</td>
<td>F</td>
<td>37</td>
<td>None</td>
<td>Repeated pulmonary infection, recent episode of intractable heart failure.</td>
<td>Intermittent pansystolic murmur and diastolic rumble at the apex.</td>
<td>Hb 10.8, Hct 32.5, Ret 3, Sed rate 35, Coombs test negative, elevated gamma globulin.</td>
<td>Normal mitral valve. A huge gelatinous mucoid mass of 6 x 4 x 3.5 cm in size with a pedicle arising from the anterior inferior portion of the left atrial septum.</td>
</tr>
<tr>
<td>5</td>
<td>F</td>
<td>60</td>
<td>Hypertension for 20 years</td>
<td>Episodes of dizziness and syncope with fluctuation of blood pressure related to postural changes.</td>
<td>Slightly accentuated first heart sound and intermittent diastolic sound.</td>
<td>Hb 14.8, Hct 44, Ret 1.3, Sed rate 14, elevated gamma globulin.</td>
<td>Normal mitral valve. A calcified tumor of 4 x 4 x 3 cm in size with a very short pedicle attached to the left atrial wall.</td>
</tr>
</tbody>
</table>

Abbreviations: Hb = hemoglobin (gm %); Hct = hematocrit (%); ret = reticulocyte count (%); Sed rate = erythrocyte sedimentation rate (mm/hr).
a multichannel recorder (Electronics for Medicine, Model DR-8 or DR-16) with Statham P-23-A strain gauges.

Selective cineangiography, with injection of contrast medium into the left ventricle, ascending aorta or pulmonary artery was performed on all patients.

Echocardiographic analysis was obtained on two patients (cases 4 and 5). It was performed by using a Unirad Ultrasonoscope and an unfocused transducer, employing piezoelectric crystal 9.5 mm in diameter with a primary resonant frequency of 2.25 MHz. The technique of recording was the same as described by Feigenbaum. Because of the unusual echocardiographic patterns obtained in the conventional approach, direct examination of the left atrium was performed using the suprasternal method described by Goldberg.

Results

Two patients (cases 3 and 4) had atrial fibrillation at the time of study, the others were in normal sinus rhythm. A moderate degree of pulmonary hypertension was noted in two patients (cases 1 and 4). The resting Fick cardiac output and cardiac index were normal in two patients (cases 1 and 2), moderately reduced in two patients (cases 3 and 5), and severely reduced in one patient (case 4). A mitral diastolic gradient was present in all of the patients and a minimal to moderate degree of mitral regurgitation was noted in all but one patient (case 5). Simultaneous recording of the left ventricular pressure and pulmonary wedge or left atrial pressure revealed two distinct hemodynamic features. In Type I (cases 1 and 2) (fig. 1), there are prominent c and v waves accompanied by a rapid y descent in the pulmonary wedge or left atrial pressure pulse, and a notch is present in the ascending limb of the left ventricular pressure pulse. In Type II (cases 3, 4, and 5) (fig. 2), although the v waves may appear prominent, the y descent is slow and there is no notch in the ascending limb of the left ventricular pressure pulse. The hemodynamic data are tabulated in table 2.
Table 2

Hemodynamic Findings of the Five Cases of Left Atrial Myxoma

<table>
<thead>
<tr>
<th>Case no.</th>
<th>RA (mm Hg)</th>
<th>RV (mean)</th>
<th>PA (mean)</th>
<th>PAW (mean)</th>
<th>LA (mean)</th>
<th>LV (mean)</th>
<th>Ao (mean)</th>
<th>LA-LV diastolic gradient (mean)</th>
<th>CI</th>
<th>CO</th>
<th>PAWc</th>
<th>PAWv</th>
<th>COv</th>
<th>COc</th>
<th>COa</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>10</td>
<td>66/24</td>
<td>66/24</td>
<td>16</td>
<td>44</td>
<td>28</td>
<td>100/9</td>
<td>98/50</td>
<td>15</td>
<td>6.3</td>
<td>3.3</td>
<td>3.3</td>
<td>230</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>3</td>
<td>32/3</td>
<td>30/19</td>
<td>28</td>
<td>30</td>
<td>20</td>
<td>27</td>
<td>25</td>
<td>21</td>
<td>5.7</td>
<td>3.5</td>
<td>2.0</td>
<td>98</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>12</td>
<td>37/12</td>
<td>35/21</td>
<td>—</td>
<td>16</td>
<td>20</td>
<td>130/15</td>
<td>132/88</td>
<td>3</td>
<td>3.6</td>
<td>2.0</td>
<td>3.6</td>
<td>222</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>9</td>
<td>85/7</td>
<td>85/38</td>
<td>—</td>
<td>45</td>
<td>32</td>
<td>112/9</td>
<td>112/78</td>
<td>24</td>
<td>2.4</td>
<td>1.4</td>
<td>1.4</td>
<td>800</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>5</td>
<td>34/10</td>
<td>32/12</td>
<td>20</td>
<td>16</td>
<td>14</td>
<td>168/16</td>
<td>172/80</td>
<td>0-2</td>
<td>3.5</td>
<td>2.2</td>
<td>2.2</td>
<td>115</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: RA = right atrium (mean); RV = right ventricle; PA = pulmonary artery; PAW = pulmonary artery wedge pressure; LA = left atrium; LV = left ventricle; Ao = aorta; CO = cardiac output (L/min); CI = cardiac index (L/min/M²); PVR = pulmonary vascular resistance (dynes/sec-cm⁻⁵).

The cineangiograms of those patients with Type I left atrial hemodynamics (cases 1 and 2) demonstrated that the tumor moved from the left ventricle to the left atrium in early systole and moved from the left atrium to the left ventricle in early diastole (fig. 3). On the other hand, in those patients with Type II left atrial hemodynamics (cases 3, 4, and 5), the tumor remained in the left atrium during the entire cardiac cycle (fig. 4).

Echocardiography was available in only two patients (cases 4 and 5) with Type II left atrial hemodynamics. Dense abnormal echoes were demonstrated behind the aortic root within the left atrium in both patients. These echoes remained within the left atrium throughout the cardiac cycle. Direct examination of the left atrium via the suprasternal approach also demonstrated large mass lesions which remained within the left atrial cavity through systole and diastole (fig. 5). In one patient (case 4) (fig. 6), these abnormal echoes were present beneath the proximal portion of the anterior mitral leaflet during diastole, but they were not seen beneath the more distal portions of the leaflet as the sound beam was directed inferiorly along the major left ventricular axis. This mass, therefore, did not appear to enter the left ventricular cavity. The diastolic slope velocity of the anterior mitral leaflet was reduced in this patient, reflecting the decreased flow across the mitral orifice. Mitral valve movement was normal in the other patient (case 5) (fig. 7).

Discussion

Clinically, left atrial myxoma has been described to masquerade as mitral valvular disease, bacterial en-
docarditis, or connective tissue disorders. Adams et al. reported that over 90% of the cases with this disease were initially diagnosed as having rheumatic mitral valvular stenosis. The clinical distinction of this disease is of considerable importance because it may be lethal if left untreated, but represents a curable form of disease with rare recurrence if treated surgically.

Hemodynamically, left atrial myxoma is usually associated with a diastolic pressure gradient across the mitral valve. However, it has been claimed that the contour of the left atrial and/or pulmonary wedge pressure pulse can be used to distinguish left atrial myxoma from mitral valvular stenosis. In the former, prominent c and v waves with a rapid y descent are present. With cineangiograms, we, and others previously, have demonstrated the mechanisms responsible for these hemodynamic changes referred to herein as Type I left atrial hemodynamics for left atrial myxoma. The tumors move from the left ventricle into the left atrium in early systole, abruptly decreasing the left ventricular volume and causing a notch in the ascending limb of the left ventricular pressure pulse. The concomitant sudden increase in the left atrial volume produces prominent c and subsequently, dominant v waves. In early diastole, the tumors enter the left ventricle, abruptly decreasing the left atrial volume. Because of this sudden decompression of the left atrium, the y descent is rapid and deep in character. Two of our patients (cases 1 and 2) demonstrated these hemodynamic changes (fig. 1) with cineangiographic correlation (fig. 3). We believe that these features are characteristic of prolapsing left atrial myxomas and it is their movement back and forth across the mitral orifice which results in the observed hemodynamic effects.

In contrast, the other three patients (cases 3, 4, and 5) had hemodynamic features indistinguishable from that of mitral valvular stenosis (fig. 2), in which there is no notch present in the ascending limb of the left ventricular pressure pulse and the y descent is relatively slow. In these cases, the cineangiograms revealed that the tumors remained in the left atrium during the entire cardiac cycle (fig. 4). Accordingly, flow across the mitral valve was impeded throughout diastole, causing a relatively slow y descent. These features are referred to herein as Type II left atrial hemodynamics of nonprolapsing left atrial myxomas. At surgery, none of these cases had mitral valvular stenosis, and the mitral valves were normal in all patients except for one (case 3), whose mitral valve was thickened and fibrotic. In this patient, thickening and fibrosis of the mitral valve was felt to have
resulted from longstanding trauma due to the motion of a calcified atrial myxoma, since there was neither commissural fusion nor microscopic evidence of rheumatic process.

The echocardiograms of the two patients (cases 4 and 5) with nonprolapsing left atrial myxoma (figs. 5, 6, and 7) were consistent with the observed hemodynamic events. The tumor echoes were identified in the left atrium but not within the left ventricle during diastole in contrast to the usual echocardiographic findings that have been described for left atrial myxoma. The motion of the anterior mitral leaflet was completely normal in one patient (case 5). Both of these patients also illustrate the particular value of direct echocardiographic examination of the left atrium when a nonprolapsing tumor is present. Such direct examination of the left atrium along the long axis of the left ventricle in the presence of suspected nonprolapsing atrial myxoma was first suggested by Popp and Harrison. We found the suprasternal approach proposed by Goldberg an effective means of examining the left atrium directly (fig. 6).

Features similar to our Type II left atrial hemodynamics have been reported in a single patient with left atrial myxoma as an “unusual” finding. However, three of our five patients demonstrated these changes. The importance of Type II left atrial hemodynamics in the presence of left atrial myxoma has not been emphasized previously. Although because of the small number of cases included in this report, statistically one cannot draw a conclusion, we do, however, believe that these changes, related to nonprolapsing tumors, occur more frequently than is generally realized. Failure to recognize this phenomenon may lead to unnecessary and potentially hazardous transeptal left heart catheterization in patients with left atrial myxoma.

In conclusion, two variations in left atrial hemodynamics may occur in left atrial myxoma. In Type I, or prolapsing myxoma, there is a notch in the upstroke of the left ventricular pressure pulse and
prominent c and v waves with a rapid y descent are present in the pulmonary wedge or left atrial pressure pulse. In Type II, nonprolapsing myxoma, there is no notch in the upstroke of the left ventricular pressure pulse and the y descent of the pulmonary wedge or left atrial pressure pulse is slow and indistinguishable from that of mitral valvular stenosis. The important contribution and value of well performed echocardiography in the precatheterization assessment of patients with suspected mitral valve disease is emphasized. It is stressed that irrespective of the left atrial hemodynamic findings, pulmonary angiography with observation of the levophase should be performed in any patient with clinical or echocardiographic evidence suggestive of left atrial myxoma.

Acknowledgment
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LEFT ATRIAL MYXOMAS

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