Echocardiographic Study of the Cardiac Involvement in Rheumatoid Arthritis*

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Echocardiograms of high quality were obtained in 26 patients with classic or definite rheumatoid arthritis by the criteria of the American Rheumatism Association, but with no evidence of overt heart disease. The echocardiograms were analyzed for evidence of pericardial disease. The E-to-F slope was determined to assess the mitral valve, and multiple indices of left ventricular performance were determined. Only four patients with rheumatoid arthritis (15 percent) had evidence compatible with pericardial thickening or small effusion. None had significant abnormality of the E-to-F slope; the mean values of the E-to-F slope were not different from controls (101 ± 28 vs 100 ± 22 mm/sec). The mean velocity of circumferential fiber shortening (Vcf) was lower in the patients with rheumatoid arthritis as a group (1.13 ± 0.17 vs 1.34 ± 0.17 circumferences per second in controls; P < 0.001), but only two patients (8 percent) had a Vcf below the normal range. All other echocardiographic indices of left ventricular contractility in patients with rheumatoid arthritis were not significantly different from controls. The ratio of the prejection period over the left ventricular ejection time was higher in the patients with rheumatoid arthritis (0.35 ± 0.03 vs 0.30 ± 0.05 in controls; P < 0.001). Echocardiographic studies can unveil a small incidence of cardiac involvement in rheumatoid arthritis (pericardial disease and possibly myocardial depression); however, abnormalities of the mitral valve are either rare or undetectable by ultrasonic techniques.

Cardiac involvement in rheumatoid arthritis is well known. Rheumatoid granulomas, pericarditis, myocardial disease, arteritis, endocarditis, and valvular lesions have been described in material from autopsies; however, clinically overt rheumatoid heart disease, ie, heart disease secondary to demonstrable rheumatoid granuloma, is believed to be rather rare.5

The advent of echocardiography introduced a new dimension to the elucidation of cardiac disorders in patients with rheumatoid arthritis. The available reports concerning involvement of the mitral valve and pericardial disease presented conflicting results.6,8 There is no available information related to myocardial involvement. Therefore, this study was designed in order to reevaluate the incidence of different aspects of cardiac involvement that can be identified by ultrasound in patients with rheumatoid arthritis.

Materials and Methods

Thirty-three consecutive patients with classic or definite rheumatoid arthritis (by the criteria of the American Rheumatism Association) who were followed-up at the Rheumatology Clinic of the Houston Veterans Administration Hospital were studied by echocardiography. Of these, 26 patients, in whom echocardiograms of good quality were obtained, constituted our study group. Seventeen patients with no known cardiac illness who were hospitalized because of other disease had similar ultrasonic studies and were used as controls. All patients were examined by at least one of us, with special attention to the cardiovascular system. Patients with a history or physical findings of hypertension, angina, myocardial infarction, heart failure, or valvular disease were excluded.

The echocardiograms were recorded using a diagnostic ultrasonoscope (Smith-Kline Ekoline 20A) utilizing a C-12, 2.25-MHz focused transducer with a rate of repetition of 1,000 impulses per second. The output of the ultrasonoscope was recorded on a strip-chart recorder (Honeywell) at a paper speed of 50 mm/sec, with a simultaneously recorded electrocardiogram.

The patients were examined in the supine or left semilateral position. The left ventricle was studied according to previously described methods.10-14 In both groups (patients with rheumatoid arthritis and controls), the following measurements were made: E-to-F slope of the anterior leaflet of the mitral valve; thickness of the left ventricular posterior wall; interventricular septal thickness; end-diastolic and endsystolic dimensions; stroke volume; ejection fraction; normalized mean velocity of circumferential fiber shortening; nor-
ormalized mean velocity of the posterior wall; and normalized mean interventricular septal velocity. The ejection fraction (EF) was determined according to the following formula:11

\[
EF = \frac{Dd^3 - Ds^3}{Dd^3}
\]

where Dd is the end-diastolic dimension and Ds is the endsystolic dimension. The normalized mean velocity of circumferential fiber shortening (Vcf) in circumferences per second was determined using the following formula:13

\[
\text{Mean Vcf} = \frac{(Dd - Ds)}{\text{LVET} \times Dd}
\]

The left ventricular ejection time (LVET) was determined from the carotid pulse tracing.

Simultaneous recordings of the phonocardiogram, ECG, and carotid pulse tracings were made immediately before or after the echocardiographic recordings. The carotid pulse tracing was obtained using a transducer (Electronics for Medicine model PSA) held manually over the carotid artery and was displayed on a recorder (Electronics for Medicine DR12). The tracings were recorded at a paper speed of 100 mm/sec. The following values were calculated: corrected left ventricular ejection time; corrected preejection period; ratio of preejection period over left ventricular ejection time (PEP/LVET).15,16 The statistical analysis was performed using Student’s t-test.

**RESULTS**

**Clinical Data**

There were 23 men and three women in the group with rheumatoid arthritis; the age range was 33 to 63 years, with a mean of 53 years. The duration of disease ranged from 1 to 33 years, with a mean of ten years. None of the patients had a history or evidence of myocardial infarction, anginal or pericardial pain, heart failure, hypertension, or valvular disease. None was taking antihypertensive or cardiac drugs.

The ECG and cardiac size by chest x-ray film were within normal limits in all patients. Five patients with rheumatoid arthritis had localized short systolic murmurs (grade 1-2/6) at the apex or left sternal border. A gallop rhythm with a fourth heart sound was heard in two patients with rheumatoid arthritis.

The 17 control patients were all men. Their ages ranged between 28 and 61 years (mean, 49 years). None of these patients had a history or evidence of significant cardiac disease or hypertension. The ECG and cardiac size by chest x-ray film were normal in all patients. Three control patients had localized short systolic murmurs (grade 1-2/6) at the apex or left sternal border, and a gallop rhythm with a fourth heart sound could be heard in six control patients.

**Echocardiographic Data**

The mean value of the E-to-F slope was 101 ± 28 mm/sec (range, 55 to 180 mm/sec) in the group with rheumatoid arthritis and 100 ± 22 mm/sec (range, 75 to 145 mm/sec) in the control group. There was evidence of a small pericardial effusion or thickened pericardium17 in four (15 percent) of the 26 patients with rheumatoid arthritis and in none of the 17 control patients (Fig 1).

The left ventricular dimensions and indices of left ventricular function obtained during this study are summarized in Table 1. No significant differences

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**Table 1—Left Ventricular Dimensions and Indices of Left Ventricular Function**

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Controls</th>
<th>Rheumatoid Arthritis</th>
</tr>
</thead>
<tbody>
<tr>
<td>E-to-F slope of anterior leaflet of mitral valve, mm/sec</td>
<td>100 ± 22</td>
<td>101 ± 28</td>
</tr>
<tr>
<td>End-diastolic dimension, cm</td>
<td>4.6 ± 0.7</td>
<td>4.8 ± 0.8</td>
</tr>
<tr>
<td>End-systolic dimension, cm</td>
<td>2.9 ± 0.4</td>
<td>3.3 ± 0.7</td>
</tr>
<tr>
<td>End-diastolic volume, cu cm</td>
<td>105 ± 49</td>
<td>122 ± 63</td>
</tr>
<tr>
<td>Stroke volume, ml</td>
<td>79 ± 40</td>
<td>82 ± 38</td>
</tr>
<tr>
<td>Ejection fraction, percent</td>
<td>73 ± 9</td>
<td>60 ± 6</td>
</tr>
<tr>
<td>Mean velocity of circumferential fiber shortening, circumferences per second</td>
<td>1.34 ± 0.17**</td>
<td>1.13 ± 0.17**</td>
</tr>
<tr>
<td>Mean velocity of posterior wall, sec⁻¹</td>
<td>0.91 ± 0.29</td>
<td>0.77 ± 0.17</td>
</tr>
<tr>
<td>Mean interventricular septal velocity, sec⁻¹</td>
<td>0.51 ± 0.20</td>
<td>0.48 ± 0.13</td>
</tr>
<tr>
<td>PEP/LVET</td>
<td>0.30 ± 0.05**</td>
<td>0.35 ± 0.03**</td>
</tr>
</tbody>
</table>

*All values represent mean ± SD.

**P < 0.001 for comparison of controls and patients with rheumatoid arthritis.

**CARDIAC INVOLVEMENT IN RHEUMATOID ARTHRITIS**
were observed between the group with rheumatoid arthritis and the control group regarding the end-diastolic dimension, end-systolic dimension, end-diastolic volume, stroke volume, ejection fraction, normalized mean velocity of the posterior wall, and normalized mean interventricular septal velocity. The mean (normalized) velocity of circumferential fiber shortening, although within the normal range (1.0 to 1.7 circumferences per second) was reduced in the group with rheumatoid arthritis (1.13 ± 0.17 circumferences per second), compared to the control group (1.34 ± 0.17 circumferences per second; P < 0.001). In only two patients with rheumatoid arthritis was the mean velocity of circumferential fiber shortening abnormal (0.74 and 0.87 circumferences per second), suggesting possibly significantly depressed myocardial function. Also, the PEP/LVET was significantly higher in the group with rheumatoid arthritis, supporting the possibility of some depression of myocardial function.

D I S C U S S I O N

The nature and extent of cardiac involvement in rheumatoid arthritis has long been of interest. Most of the available reports discuss the histopathologic findings at necropsy. The lesions that have been most commonly found are pericarditis, rheumatoid granulomata, and vasculitis. Valvular regurgitation, pericarditis with effusion, tamponade or chronic constriction, myocardial disease, heart failure, defects in conduction, and coronary arteritis with myocardial infarction have also been reported; however, the manifestations are usually subtle and antemortem diagnosis of rheumatoid heart disease is seldom made in the vast majority of cases. A method of early identification of those patients with rheumatoid arthritis and cardiac involvement is highly desirable, since later stages may become serious enough to be life-threatening.

Echocardiography is now a well-established technique for the noninvasive evaluation of heart disease. Recent studies have proven reliable in the assessment of left ventricular performance in the absence of asynergy. A few echocardiographic studies have been reported on patients with rheumatoid arthritis. These studies dealt with motion of the mitral valve and pericardial disease, but no information is available regarding myocardial function, although myocardial disease has been described at autopsy.

Two recent echocardiographic studies reported a high incidence of abnormalities of the mitral valve (based on the observation of decreased E-to-F slope) and pericardial involvement in patients with rheumatoid arthritis. The mean E-to-F slope of the anterior leaflet of the mitral valve in our group with rheumatoid arthritis was within normal limits (101 mm/sec) and not different from the control group. Only one patient in the group with rheumatoid arthritis showed a borderline E-to-F slope (55 mm/sec). Our results support the observations of Davia et al that echocardiographic abnormalities of the anterior leaflet of the mitral valve rarely, if ever, occur in patients with rheumatoid arthritis, provided meticulous attention to the recording technique is observed. The discrepancy between our results and those of Nomeir et al and Prakash et al is not explained, but it may be due to inclusion in their studies of patients with left ventricular hypertrophy consequent to hypertension or other causes that may result in a decreased E-to-F slope secondary to diminished compliance of the left ventricle.

The incidence of pericardial abnormalities (minimal effusion or thick pericardium) was 15 percent (four of 26 patients) in the group with rheumatoid arthritis (Fig 1). This incidence also appears to be less than those previously reported, and the discrepancies can most probably be ascribed to technical factors. In our study, only patients with echocardiograms of high quality were included.

Of the indices of left ventricular function measured in our study, the values for the mean velocity of circumferential fiber shortening and the PEP/LVET were suggestive of depressed myocardial function in the patients with rheumatoid arthritis, compared to control patients. The mean values of these indices in the group with rheumatoid arthritis, although within normal limits, were significantly less than in the control group. Only two (8 percent) of the patients with rheumatoid arthritis had an abnormally depressed mean velocity of circumferential fiber shortening. Neither of these two patients had any other clinical evidence of heart disease. Although the clinical implications of an isolated finding of a reduced value for the mean velocity of circumferential fiber shortening are not established, it is believed to be a sensitive index for detecting abnormalities of left ventricular performance.

Whether the depressed mean velocity of circumferential fiber shortening and the increased PEP/LVET represent definite myocardial involvement by the rheumatoid process is difficult to ascertain, and further studies are necessary to clarify this possibility.

We conclude that echocardiographic studies can detect a small incidence of cardiac involvement in patients with rheumatoid arthritis but with no overt clinical evidence of heart disease, ie, pericardial disease in 15 percent and probable myocardial involvement in 8 percent; however, abnormalities of the
mitral valve in this population are either rare or undetectable by ultrasonic techniques.

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