Echocardiographic abnormalities in ankylosing spondylitis

T W O’Neill, G King, I M Graham, J Molony, B Bresnihan

Abstract
Twenty four patients with ankylosing spondylitis of 10 or more years’ duration were assessed for evidence of cardiac disease. Seven patients (29%) had evidence of cardiac disease, including one patient with a pericardial effusion, three with conduction abnormalities, and two with aortic incompetence. Aortic incompetence in one patient was clinically silent and was detected only with Doppler echocardiography. This patient had, in addition, thickening of the posterior aortic wall, an echocardiographic feature not previously described in ankylosing spondylitis. There was no evidence of aortic valve disease in a control group matched for age and sex. Patients with ankylosing spondylitis and cardiac abnormalities were older, had a longer disease duration, and more peripheral joint disease than those without cardiac abnormalities. Doppler echocardiography is a useful technique in the assessment of cardiac disease in ankylosing spondylitis and may detect aortic valve disease at an early preclinical stage.

Ankylosing spondylitis is a chronic inflammatory disease, affecting predominantly young men, with a predilection for the sacroiliac joints and spine. Cardiac abnormalities are well recognised. The most important lesions are aortic valve incompetence and heart block. Other lesions include mitral insufficiency, pericarditis, and cardiomyopathy. The development of non-invasive cardiac imaging techniques, particularly Doppler echocardiography, gives increased sensitivity in the detection of valvular regurgitation.

We evaluated a group of patients with ankylosing spondylitis using M mode, two dimensional, and Doppler echocardiography, looking for evidence of cardiac abnormalities and in particular for evidence of early aortic valve disease.

Patients and methods
Twenty four patients with ankylosing spondylitis of 10 or more years duration who attended our rheumatology clinic were recruited for this study. The reason for selecting patients with longer than 10 years duration of disease was to increase the chance of finding such abnormalities, as their frequency is known to increase with age. All patients fulfilled the New York classification for AS. Patients were excluded if they had psoriasis, Reiter’s syndrome, or if they had a history of hypertension, rheumatic, or ischaemic heart disease. Twenty four controls matched for age and sex were selected from the same source. The control subjects had either soft tissue or degenerative rheumatic disorders.

A full history, clinical examination, standard 12 lead electrocardiographic, two dimensional, M mode, and Doppler echocardiographic examinations were performed on all subjects. Lumbar flexion was measured using the modified Schober test and chest expansion was measured at the fourth costochondral junction. Echocardiography was performed using a Hewlett Packard 77 OZOA system rev K with a selection of electronic focused transducers ranging from a 3.5 to 5 mHz probe. The system had colour flow mapping facilities as well as standard pulsed and continuous wave Doppler ultrasound. Patients were placed in a semi-erect right anterior oblique position. Standard long axis, short axis, and apical views were obtained. Quantitative Doppler echocardiographic assessments were complemented by the addition of colour flow mapping.

Electrocardiograms and echocardiograms were read blindly at the end of the study by an independent observer. Student’s t test and Fisher’s exact probability test were used to assess whether there was an association between the clinical features of patients with ankylosing spondylitis and the presence of cardiac abnormalities.

Results
There were 18 men and six women in each group. Table 1 summarises their age and, for the patients with ankylosing spondylitis, disease duration, occurrence of peripheral arthritis, uveitis, and degree of limitation of chest expansion and lumbar flexion. The electrocardiographic and echocardiographic features of those patients with ankylosing spondylitis and control subjects with cardiac abnormalities are presented in tables 2 and 3 respectively.

Aortic incompetence was present in two patients with ankylosing spondylitis. This was
Table 2  Cardiac abnormalities in patients with ankylosing spondylitis

<table>
<thead>
<tr>
<th>Patient No</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Disease duration (years)</th>
<th>Echocardiographic findings*</th>
<th>Electrocardiographic findings*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>45</td>
<td>M</td>
<td>21</td>
<td>AI; thickened posterior, aortic wall</td>
<td>Q waves in inferior leads</td>
</tr>
<tr>
<td>2</td>
<td>53</td>
<td>M</td>
<td>21</td>
<td>AI</td>
<td>LVH</td>
</tr>
<tr>
<td>3</td>
<td>39</td>
<td>F</td>
<td>18</td>
<td>Small pericardial effusion</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>41</td>
<td>F</td>
<td>15</td>
<td>MVP</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>35</td>
<td>M</td>
<td>16</td>
<td></td>
<td></td>
</tr>
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<td>6</td>
<td>46</td>
<td>M</td>
<td>23</td>
<td></td>
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<tr>
<td>7</td>
<td>37</td>
<td>M</td>
<td>15</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*AI=aortic incompetence; MVP=mitral valve prolapse; IRBBB= incomplete right bundle branch block; LAD= left bundle branch block; LVH= left ventricular hypertrophy.

Table 3  Cardiac abnormalities in control group

<table>
<thead>
<tr>
<th>Patient No</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Echocardiographic findings*</th>
<th>Electrocardiographic findings*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>41</td>
<td>M</td>
<td>MVP</td>
<td>RBBB</td>
</tr>
<tr>
<td>2</td>
<td>27</td>
<td>M</td>
<td>MVP</td>
<td>LAD 35°</td>
</tr>
<tr>
<td>3</td>
<td>59</td>
<td>M</td>
<td></td>
<td>LAD 35°</td>
</tr>
<tr>
<td>4</td>
<td>55</td>
<td>M</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>61</td>
<td>F</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Abbreviations: MVP=mitral valve prolapse; RBBB=right bundle branch block; LAD=left axis deviation.

Clinically inaudible in one patient (patient 1 in table 2, and was detected only with Doppler echocardiography. The patient had, in addition to mild aortic incompetence, thickening of the posterior aortic wall (fig 1). In the other patient (patient 2 in table 2) a diastolic murmur was clearly audible. A small pericardial effusion was present in one of the patients with ankylosing spondylitis.

Table 4  Comparison of 24 patients with ankylosing spondylitis with and without cardiac abnormalities

<table>
<thead>
<tr>
<th>Clinical characteristic</th>
<th>Cardiac abnormality</th>
<th>Absent (n=17)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Present (n=7)</td>
<td>40-1</td>
</tr>
<tr>
<td>Mean age (years)</td>
<td>42-3</td>
<td></td>
</tr>
<tr>
<td>Mean disease duration (years)</td>
<td>18-4</td>
<td>17-5</td>
</tr>
<tr>
<td>No (%) with uveitis (%)</td>
<td>3 (43)</td>
<td>9 (53)</td>
</tr>
<tr>
<td>No (%) with peripheral arthritis</td>
<td>4 (37)</td>
<td>5 (29)</td>
</tr>
<tr>
<td>Mean lumbar flexion (cm)</td>
<td>3.36</td>
<td>2.93</td>
</tr>
<tr>
<td>Mean chest expansion (cm)</td>
<td>2.64</td>
<td>2.40</td>
</tr>
</tbody>
</table>

Table 4 compares the clinical features of patients with ankylosing spondylitis who had a cardiac abnormality with those who did not. Twenty-nine per cent of the patients with ankylosing spondylitis had a cardiac abnormality. Analysis of the results showed that those with a cardiac abnormality were older, had longer disease duration, and more peripheral disease; however, the results were not statistically significant. There was no significant difference between those with a cardiac abnormality and the presence of uveitis or restriction of chest or lumbar movements compared with those without.

Discussion

The reported occurrence of clinical aortic incompetence in ankylosing spondylitis varies in reported series of patients with ankylosing spondylitis from 1 to 10%. In early aortic valve disease a murmur is not always audible and it therefore seems likely that the true prevalence is higher than this.

In our study two of 24 patients had evidence of aortic incompetence detected by Doppler echocardiography. This was clinically inaudible in one patient. This patient had, in addition to mild aortic incompetence, evidence of posterior aortic wall thickening, a feature which has not been previously reported in ankylosing spondylitis. Bulkey and Roberts, in a pathological study of eight patients with ankylosing spondylitis and aortic incompetence, found that the aortic valve cusps and the aorta immediately behind and above the sinuses of valsalva were thickened, the latter by dense adventitial scar tissue and fibrous proliferation. In each patient the scar tissue in the root of the aorta extended below the base of the valve to produce a subaortic fibrous ridge. This feature has been described echocardiographically and is known as a subaortic bump. We found no evidence of a ridge in our study; however, it seems likely that the thickening of the posterior aortic wall in our patient with aortic incompetence represents...
fibrous thickening, a result of aortitis, and is therefore part of the disease spectrum of ankylosing spondylitis. Aortic regurgitation itself does not produce aortic root thickening.

Previous studies using two dimensional and M mode echocardiography have looked for evidence of early aortic valve disease. Thomas et al found mild dilatation of the aortic root in six of 23 patients with ankylosing spondylitis. This was thought to be related to aortic root disease secondary to ankylosing spondylitis. Tucker et al found a subaortic bump in six of 35 patients, and Labresh reported this in 10 of 36 patients with clinically normal hearts.

Two dimensional and M mode echocardiography are less sensitive in the detection of aortic regurgitation than Doppler echocardiography. This is because Doppler echocardiography measures regurgitant flow directly; the other methods use less direct methods including fluctuation on the aortic valve leaflet or contiguous cardiac structures.

Grayburn et al compared the accuracy of clinical, two dimensional, M mode, and pulsed Doppler echocardiography in the assessment of aortic valve disease in 106 patients using the 'gold standard' of supravalvular aortography. They found the sensitivity and specificity of pulsed Doppler echocardiography in the detection of aortic regurgitation to be 96 and 96% respectively, 43 and 91% for two dimensional echocardiography and 46 and 81% for the M mode finding of anterior mitral valve flutter. In 19 patients, Doppler echocardiography detected aortic incompetence in the absence of a murmur. To our knowledge no previous studies have assessed a group of patients with ankylosing spondylitis using Doppler echocardiography to look for early aortic valve disease.

Other findings in our study include one patient in the ankylosing spondylitis group who had a pericardial effusion and three who had a conduction abnormality. We found no increased occurrence of mitral valve prolapse in our patients with ankylosing spondylitis compared with the control subjects. A previous study has suggested an increased occurrence of mitral valve prolapse in patients with ankylosing spondylitis, although this study did not have a comparison group. As in other studies, cardiac disease was greater in patients with ankylosing spondylitis who were older and had longer disease duration. Peripheral arthritis occurred in 57% of the patients with ankylosing spondylitis with cardiac abnormalities compared with 29% without. These results were not statistically significant.

Uveitis was present in 12 (50%) of our patients with ankylosing spondylitis. This is higher than the reported prevalence of 25%. Edmunds et al, in a review of 1335 patients with ankylosing spondylitis, reported an occurrence of 40% and suggested that this higher percentage may reflect the long mean disease duration in these patients. This may have been a factor in our series where the average disease duration was 17-75 years.

Doppler echocardiography is useful in the assessment of cardiovascular disease in patients with ankylosing spondylitis and may detect the presence of asymptomatic aortic incompetence. Further studies using this technique are required to define more clearly the occurrence, natural history, and prognosis of aortic valve disease in ankylosing spondylitis.

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