Electrocardiography and Echocardiography in Patients with Chest Pain and Mitral Leaflet Prolapse

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SUMMARY: 34 patients with catheter verified, haemodynamically trivial mitral leaflet prolapse with chest pain and normal coronary arteries were studied. Nineteen had no auscultatory signs of MLP despite clear catheter or echocardiographic evidence of prolapse. Infero-lateral repolarisation changes on the resting electrocardiogram were present in 29%. 50% had false positive exercise tests. M-mode echocardiography proved diagnostically disappointing but sector scans revealed mitral leaflet prolapse in 62%. The apical 4 chamber acoustic window was markedly superior to the parasternal long axis acoustic window.

Introduction

Idiopathic mitral leaflet prolapse (MLP) is a common cardiac abnormality with a prevalence of between 5% and 18% depending upon the race, age and sex of the population studied and the diagnostic methods and criteria employed. The diagnosis must be considered in patients complaining of chest pain, palpitations, dyspnoea, syncope or transient neurological events particularly if objective findings such as thin body habitus, non-ejection systolic clicks or non-ejection systolic murmurs are also present. The diagnosis is usually based upon auscultatory findings supported by non-invasive investigations particularly echocardiography. Errors in technique and flexibility in data interpretation have however led to considerable diagnostic confusion. This seems to be particularly the case when chest pain dominates the clinical picture as symptomatology and electrocardiography may strongly suggest coronary heart disease. This study attempts to evaluate the role of resting and exercise electrocardiography and M-mode and 2-dimensional echocardiography in patients with mitral leaflet prolapse and angiographically normal coronary arteries presenting with chest pain.

Methods and Patients

Patients who had undergone cardiac catheterisation at St. Thomas’ Hospital between 1980 and 1983 were reviewed retrospectively. Those with mitral leaflet prolapse (MLP) were then studied prospectively.

Exclusion Criteria

1. Abnormal coronary angiograms
2. Abnormal left ventriculogram
3. Valve dysfunction other than MLP
4. Mitral incompetence of more than trivial degree
5. Age over 70 years.

The following non-invasive investigations were carried out:

A. Assessment of cardiac symptoms and auscultatory signs
B. Resting 12 lead electrocardiogram in the standing position recorded at 25mm/seconds.
C. Symptom-limited treadmill exercise test using the Bruce protocol. A standard 12 lead ECG was recorded in the standing position immediately before and immediately after exercise. Heart rate and rhythm were monitored throughout. A computer averaged signal was recorded before, during and for 5 minutes after exercise employing leads V1, V5 and aVf.
D. Echocardiography A Hewlett Packard 3.5mHz transducer was used with the patient in the left lateral position. Multiple sector scans were obtained utilizing the parasternal long axis and apical 4 chamber acoustic windows. Representative scans were recorded during ventricular systole on photographic paper. The diagnosis of MLP was based on mitral valve morphology as recorded on the polaroid films. M-mode scans were derived from sector scans utilizing the parasternal long axis acoustic window with the M-mode cursor perpendicular to and intersecting the free margin of the mitral leaflets. Traces were recorded on photographic paper at a paper speed of 25 mm/second.

Definitions

1. Ischaemic ST Segment Response to Exercise. More than 1 mm of horizontal or downsloping ST segment depression measured at 80 ms from the J point.
2. **M-mode MLP.** Posterior displacement of the mitral valve of more than 3 mm during ventricular systole.

3. **Sector Scan MLP.** Movement of either mitral valve leaflet below the plane of the mitral valve annulus towards the left atrium during ventricular systole.

4. **Mitrval Valve Annulus**
   A) **Parasternal View.** The annulus is represented by a line joining the insertion of the mitral valve leaflet into the left atrium anteriorly and the left ventricular myocardium posteriorly.
   B) **Apical 4 Chamber View.** The annulus is represented by a line joining the point of connection. If the interatrial septum and the ventricular myocardium medially and the atrioventricular groove laterally.

**Results**

34 patients (20 men and 14 women) were studied. Their mean age was 49 years (range 27-63 years).

**Clinical Findings**

All patients continued to complain of chest pain. In 8 the pain closely resembled exertional angina although atypical features (particularly a slow response to rest) were present. Fifteen had both rest and effort pain while 11 had rest pain only. In them the prolonged length of pain was striking. In addition 17 complained of palpitation and 8 of exertional dyspnoea. Drug therapy included digoxin in 3, beta-adrenergic blocking drugs in 11, other drugs in 17 and no drugs in only 3. Auscultation revealed no abnormalities in 19 (56%). Of the remainder 3 had isolated non-ejection clicks, 2 isolated late systolic murmurs, 9 non-ejection click followed by a late systolic murmur and 1 a non-ejection click and a pansystolic murmur. Of the 10 patients with both click and murmur 8 (80%) were female.

**Electrocardiography**

Two patients were unable to stand (arthritis, lower limb amputation) and were excluded.

A) **Resting ECG.** Traces were normal in 17 (53%). The electrocardiographic abnormalities in the remaining 15 patients are shown in Table I. There was a significant excess of women (77% versus 26%, SE difference 15.4). The commonest abnormalities were inferior or inferolateral repolarisation changes particularly in women. In two patients it is possible that digoxin was responsible for the cardiographic abnormality.

<table>
<thead>
<tr>
<th>Abnormality</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right bundle branch block</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Ventricular ectopics</td>
<td>2†</td>
<td>0</td>
</tr>
<tr>
<td>T wave inversion or ST sagging in leads II, III, aVF</td>
<td>1</td>
<td>10†</td>
</tr>
<tr>
<td>T wave inversion or ST sagging in leads V4, V5, V6</td>
<td>0</td>
<td>3*</td>
</tr>
</tbody>
</table>

† = 1 patient taking digoxin
* = all 3 had changes in leads II, III, aVF.

B) **Exercise ECG.** The mean duration of exercise was 6.6 minutes (7.6 for men and 4.9 minutes for women). Sixteen patients had false positive exercise tests (42% men; 62% women; SE diff 16.1). One male and 1 female were taking digoxin which could have been responsible for the false positive tests. There were no complications.

**Arrhythmias.** Two had infrequent ventricular premature beats at rest (1 was taking digoxin). On exercise the ectopics disappeared in 1 while they increased in the patient taking digoxin. On exercise 1 patient developed infrequent ventricular ectopics while another another had infrequent supraventricular ectopics.

**Echocardiography**

All 34 patients had both M-mode and 2-dimensional echocardiograms. Definite mitral leaflet prolapse was demonstrated in 22 (65%).

A) **M-mode.** Four patients had definite late systolic prolapse (confirmed by sector scan in 3). No holosystolic prolapse was seen. In 4 patients multiple systolic echoes involving the anterior mitral leaflet were seen (all had other evidence of MLP).

B) **Sector Scans.** Definite MLP was seen in 21 (62%) of patients in either the parasternal long axis view or the apical 4 chamber view or both. The 4 chamber view appeared to be considerably superior to the long axis parasternal view. Table II shows the relative sensitivities of the respective acoustic windows. In 11 patients in whom the mitral leaflets were not well seen parasternally the 4 chamber view was diagnostic. In 4 patients in whom the apical acoustic window was poor the parasternal view allowed adequate visualisation of the leaflets and showed MLP in one. Well visualised but normal leaflets seen parasternally were shown to be prolapsing using the 4 chamber view in 4 patients while normal valve motion in the 4 chamber views was always associated with normal motion in the parasternal view. Diagnostic echocardiograms were more common in patients with suggestive physical signs although the difference was not significant (79% versus 55%; SE diff 15.6).

**Discussion**

Chest pain in patients with mitral leaflet prolapse may closely resemble that due to coronary heart disease. In patients with atypical features the detection of the auscultatory signs of mitral valve prolapse is of great importance regarding the probability of underlying coronary heart disease. The finding in this study of normal auscultation in 56% of patients with clear angiographic or echocardiographic evidence of MLP is therefore of great importance as in such patients the diagnosis of MLP may not be considered. Although variation of signs in patients with MLP is well described and may in part explain the disparity between signs and objective findings, a large study in healthy subjects has reveal-
ed that only 50% of those with suggestive auscultatory signs had echocardiographic evidence of MLP and that less than 15% of those with echocardiographic MLP had clicks or murmurs. Clearly multiple physical examinations are required to document accurately abnormal heart sounds and echocardiographic examination of the heart must be carried out even if signs suggestive of MLP are consistently absent.

The prevalence of abnormal resting electrocardiograms in this study was unexpectedly high but may in part be explained by patient selection (i.e. only those with chest pain severe enough to require coronary angiography). The dominant abnormalities were inferior or inferolateral repolarisation changes. Excluding the two patients (1 male, 1 female) taking digoxin there was a significant excess of women with these changes (75% versus 6%. SE 13.6). Although inferolateral repolarisation abnormalities were well described in patients with MLP, they are not sufficiently specific to have much diagnostic value. Their main importance is that ischaemic heart disease may be mimicked although an unconfirmed report suggested they identified a subset of patients at higher risk of sudden death.

The high prevalence of false positive exercise tests may again reflect an artifact of patient selection. It does however underline the dangers of misinterpretation when using exercise electrocardiography in patients with MLP who even with normal coronary arteries may produce ST segment changes which are highly suggestive of coronary heart disease. The mechanism of this pseudoischaemic response remains speculative. In some patients with MLP similar electrocardiographic changes can be induced by hyperventilation while in others they can be abolished by beta-adrenergic blocking. This finding may be of aetiological importance in that similar ST segment abnormalities have been described in patients with high circulating levels of catecholamines and there is a suggestion that some MLP sufferers have a hyperadrenergic state. Three patients were taking digoxin, two of which had positive exercise tests. As digoxin may cause ST segment changes on exercise even when the resting cardiogram is normal, interpretation becomes impossible. Although abolition of abnormal ST responses on exercise by beta-blocking drugs makes coronary artery disease less likely it unfortunately does not always identify false positive reactors as four of the patients in this study who had positive exercise tests were taking atenolol and had resting heart rates of less than 65 beats per minute. Alternative explanations for positive exercise tests in MLP patients are Syndrome X, exercise induced coronary artery spasm and early cardiomyopathy.

In most patients with suspected MLP, symptoms do not justify catheterisation, so echocardiography assumes a crucial diagnostic role. Sector scans correlate well with angiographic evidence of MLP at least in patients with considerable mitral incompetence. In this study of patients with symptomatic but haemodynamically mild MLP sector scans were diagnostic in 62%. The commonest cause of failure was inadequate visualisation of both leaflets due to poor acoustic window and this will remain a limitation of the technique. When the leaflets were well seen but normal, failures to correlate with the angiogram may have been due to intermittent prolapse or as there is some observer variation in angiographic interpretation, overdiagnosing at catheter was possible. As reported by others, patients in this study with clinical evidence of MLP were more likely to have diagnostic electrocardiograms yet importantly echocardiography revealed definite MLP in half of those patients in whom cardiac auscultation was normal.

Most sector scan studies of MLP have employed the long axis parasternal acoustic window but it has been recently suggested that the apical 4 chamber view might be superior, particularly in identifying mild MLP. In this study the 4 chamber view was more sensitive than the parasternal view and allowed clearer visualisation of the leaflets. To be confident about the diagnosis multiple scans from the parasternal long axis and 4 chamber acoustic windows should be made. Representative scans should be recorded photographically so that mitral leaflet position can be accurately related to the plane of the mitral valve annulus. Careful technique is essential as foreshortening of the apical 4 chamber view may artifically produce MLP particularly involving the anterior leaflet. Although the apical short axis view cannot be used to diagnose MLP it may be possible to see redundancy of the anterior leaflet which should stimulate a careful search for MLP in other views.

M-mode scans are less diagnostically sensitive or specific. We found the method disappointing in that only 4 diagnostic scans were made. However in 1 M-mode was positive in the presence of negative sector scans so undoubtedly M-mode scans should be recorded in every

**Table II**

<table>
<thead>
<tr>
<th>Parasternal long axis view</th>
<th>Poor Echo window</th>
<th>Normal</th>
<th>Anterior MLP</th>
<th>Posterior MLP</th>
<th>Anterior and Posterior MLP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apical 4 chamber view</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>5</td>
<td>16</td>
<td>13</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>19+</td>
<td>7</td>
<td>8</td>
<td>11</td>
<td>2</td>
</tr>
</tbody>
</table>

+ Includes 3 patients with MLP seen in the parasternal view.

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patient. Apart from poor echo windows the major cause of inaccurate M-mode scans is inadequate visualisation of mitral leaflet excursion due to lack of spatial orientation which is an intrinsic limitation of the method. In this study technically satisfactory M-mode scans were arrived at by first visualising the mitral valve in the long axis parasternal view and then deriving an M-mode scan by moving the cursor so that it intersected the free edge of the mitral leaflet perpendicularly. This method may not allow adequate visualisation of prolapsing cusps and might in part explain the poor results. Those using M-mode scans derived from sector scans might anticipate similar problems. No examples of holosystolic prolapse were identified. This echocardiographic finding however arrived at by first visualising the mitral valve in the long axis parasternal view and the echobeam directed inferiorly, false holosystolic prolapse can be produced. Multiple mitral echoes seen during systole have been associated with MLP and probably represent multiple superimposed views of redundant leaflets rather than actual prolapse. They therefore cannot be used to diagnose MLP in the absence of other features although their presence increases the possibility of MLP. Furthermore they are not specific.

We have shown in this study of patients with MLP and chest pain with normal coronary arteries a high prevalence of abnormal cardiograms and false positive exercise tests. Furthermore patients with objective evidence of MLP may have no suggestive ausculatory signs. Photographically recorded sector scans employing all acoustic windows should be taken and mitral leaflet movement related to the mitral annulus. The apical 4 chamber view seemed superior to the long axis parasternal view.

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**Letters to the Editor**

**BRUCELLOSIS**

*From Col E E Vella, L/RAMC*

SIR—The following extract from a modern well known dictionary will indubitably send icy shivers down the spinal cord of many of our rank and file and into the core of the very souls of our Caledonian confreres.

"Brucellosis, named after Sir David Bruce (1855-1931). Australian bacteriologist and physician."

Someone at present wearing or who has worn our ‘In Arduis Fidelis’ cap badge, or perhaps even you, Sir, should demand a public written apology from the Editors and the Publishers of Collins Dictionary of the English Language, 1979, p 192, or else an early dawn appointment in Hyde Park.

I am etc.,

E E VELLA

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16 April 1985

**POSTINGS OF RMOs/GPs**

*From Major D W Smith, RAMC*

SIR—I should like to comment on the letter from Colonel (now Brigadier) Beale stating the AMD 3 policy on the postings of RMOs/GPs. In his letter he states that PGMOs ‘‘after five or more years of medical training, house-jobs and PGMO, want to get ‘stuck in’’. Get ‘stuck in’ they do want, but under supervision.

We, in the Army Medical Services, have always prided ourselves in the fact that we provide a peace-time medical service as good as or better than the National Health Service. However, it is now illegal in the NHS for any doctor who does not have a certificate of GP Vocational Training to have full responsibility for the primary care of any patient. The regulations of the National Health Service Act 1977 do not of course apply to the RAMC but we must not ignore them or our standard of medical service will manifestly deteriorate.

Already there has been some minor criticism of vocational training of Army general practice by an eminent member of the Royal College of General Practitioners. This prompted a reply from Brigadier Beale. How long will it be before there is criticism of the actual service we provide?

Ideally to provide a GP service equivalent to the NHS every GP/RMO who is not working in a group practice under the supervision of a trainer, ought to be vocationally trained. Clearly this would be impossible to achieve, but I think we could reach a better compromise than the one we have at the moment.

All GPs/RMOs who are not yet vocationally trained should not work in single-handed practices looking after troops and families. I feel that they could fill true RMO posts where their clinical responsibility is only to soldiers but when it comes to looking after wives and children they ought to work in a group practice with other doctors, ideally with a trainer and at least with a more experienced doctor. It is possible in BAOR for medical officers working in group practices to fulfil the roles of GP and RMO. This has been highlighted by Major Needham.

The single-handed family practices of which there are several in BAOR should be filled by vocationally trained GPs who are either ineligible or do not wish to become trainers. This is the least we must achieve to be able to compare ourselves favourably with NHS general practice.

There are many examples of these doctors who are working in large group practices, neither trainees nor trainers.

If we can offer GPVT in combination with their RMO posts to potential GPs immediately on completing the PGMO course, we shall have two or three years of useful general practice from them before the end of their Short Service Commissions. More of our general practitioners will be ‘‘as good as’’ their equivalents in the NHS and I also firmly believe that, if they are treated in this manner, more will convert to Regular commissions. I know of many young doctors who have been totally disillusioned with the