Relation of angina to coronary artery disease in mitral and in aortic valve disease

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SUMMARY Of 129 patients with either mitral or aortic valve disease angina was present in 55 (42%). It was more frequent in aortic (60%) than in mitral valve disease (33%). The standard 12-lead electrocardiogram was not helpful in distinguishing underlying occlusive coronary artery disease. Coronary arteriography demonstrated coronary artery disease in 26 patients (20%), only 2 of whom had no angina. The incidence of coronary artery disease was almost identical in both the mitral and aortic groups (22% and 17%, respectively), but the percentage of those with demonstrable coronary artery disease accompanying angina was much higher in the mitral group (67% as against 29%). Angina in mitral valve disorders is thus much more likely to be the result of disease of the coronary arteries. Coronary arteriography is mandatory in all patients in both groups who have angina. Otherwise it seems unnecessary as coronary artery disease was found in only 2 patients who did not have angina.

Angina is a relatively common complaint in patients with aortic valve disease, and recent communications (Basta et al., 1975; Harris et al., 1975; Mandal and Gray, 1976) have indicated the value of selective coronary arteriography in distinguishing angina caused by accompanying coronary artery disease from that caused solely by severe aortic valve disease with diminished coronary blood flow. To date, however, very little has been published on the significance of angina in mitral disease. We therefore studied 129 consecutive patients with either mitral or aortic valve disease being assessed for surgical treatment to ascertain first of all the incidence of both angina and coronary artery disease and secondly whether there was any significant difference in the incidence of either angina or coronary artery disease in the two groups.

Patients and methods

One hundred and twenty-nine consecutive patients with either mitral or aortic valve disease were investigated to assess the presence of coronary artery disease. The age range was 40 to 64 years (mean 52 years), and there were 78 women and 51 men. All were symptomatic, exertional dyspnoea and chest pain being the most prominent complaints.

Eighty-two patients had solitary mitral valve disease and 47 had isolated aortic valve disease. Standard 12-lead electrocardiography, right heart catheterisation for recording resting pulmonary artery and mean wedge pressures, and left heart studies were performed in all patients. Left ventricular function was assessed from a study of the cineangiogram and the end-diastolic pressure recorded before and after angiography. Finally, selective coronary arteriography was carried out in all by the Judkins technique, a film being obtained in both oblique positions after separate injections into each coronary artery. Reduction of the vessel lumen by more than 50 per cent was used as the yardstick for indicating significant disease, and was classified as single, double, or triple vessel disease (right coronary, left anterior descending, and circumflex). Involvement of the origin of the left main coronary artery was not encountered in any of our patients. There were no deaths attributable to coronary arteriography, and complications were few and not serious.

Results

Angina was present in 55 (42%) of the 129 patients, and was classified simply as: grade 1—angina occurring only with severe exertion; grade 2—angina with ordinary activity, stress, or exposure to

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cold; grade 3—angina at rest (Mandal and Gray, 1976). Twenty-seven (33%) of the 82 with mitral valve disease and 28 (60%) of the 47 with aortic valve disease had angina. The angina was grade 1 in 45 of the 55 patients with this symptom, was grade 2 in 9 (5 in the mitral and 4 in the aortic group), and there was only 1 with grade 3.

Electrocardiographic Findings
The electrocardiogram was normal in 23, and in only 10 patients was there unequivocal evidence of ischaemic changes. In the remainder atrial fibrillation controlled by digoxin was present in 52 and left ventricular hypertrophy in 44, making the interpretation of any ischaemic changes difficult.

Left Ventricular Function
Measurement of the left ventricular end-diastolic pressure both before and after angiography and study of the cineangiogram in the right anterior oblique projection were the two indices employed for assessment of left ventricular function. Facilities for estimating end-diastolic volume and ejection fraction were not available. Information was obtained from the angiogram as to the contractility of the left ventricle, its rate of emptying and the presence of any area of hypokinesis, dyskinesis, or aneurysm. Function was then classified according to the NYHA standards. Impaired or poor function was present in 33 patients, 11 of whom had accompanying coronary artery disease.

Presence of Coronary Artery Disease at Angiography
Selective coronary arteriography showed significant disease in 26 patients, an incidence of 20 per cent. In 2 of these patients there was no history of angina. The sex incidence showed a male preponderance, with 14 (27%) of 51 men affected compared with 12 (15-5%) of 78 women. The mean age of those with coronary artery disease was 54 ± 7 years as against a mean of 51 ± 7 years for those without, which is statistically significant (P < 0.05).

Triple vessel disease was observed in 3, double vessel disease in 11, and single vessel disease in 12. In 8 patients the electrocardiogram showed ischaemic changes, in 6 left ventricular hypertrophy, in 10 atrial fibrillation, and in 2 it was normal.

Presence of Angina and Coronary Artery Disease and Aortic Valve Disease
Although angina was a common complaint in atrioventricular disease (60%), there were only 8 patients (17%) with coronary artery disease (Table 1 and Fig. 1). Coronary artery disease was in fact present in only 29 per cent of those with angina (Fig. 2). Of the 47 patients with aortic valve disease, 18 had stenosis, 15 had regurgitation, and 14 had a mixed lesion of the valve. The incidence of angina was not significantly different in any of the three lesions, nor did it bear any relation to the peak systolic gradient across the valve. The angina was classified as grade 1 in 23, grade 2 in 4 patients, and grade 3 in 1. All 15 patients with grades 2 or 3 angina had coronary artery disease.

Presence of Angina and Coronary Artery Disease in Mitral Valve Disease
Angina was present in 27 patients (33%) of the 82 patients in this group, and coronary artery disease

<table>
<thead>
<tr>
<th>Valve involved</th>
<th>Total</th>
<th>Number with angiina</th>
<th>Number with coronary artery disease</th>
<th>% Coronary artery disease related to angina</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mitral</td>
<td>82</td>
<td>27 (33%)</td>
<td>18 (22%)</td>
<td>67</td>
</tr>
<tr>
<td>Aortic</td>
<td>47</td>
<td>28 (60%)</td>
<td>9 (17%)</td>
<td>29</td>
</tr>
</tbody>
</table>

Fig. 1 Total in each group, showing the number who had angina and coronary artery disease.
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![Chart](chart.png)

Fig. 2. Percentage in each group with angina and coronary artery disease, and percentage of those with coronary artery disease related to percentage with angina.

In 18 (22%) of those with angina, 67 per cent had coronary artery disease (Table 1 and Figs 1 and 2). The 5 patients with grade 2 angina had coronary artery disease, and 12 of those with grade 1 had coronary artery disease. Solitary mitral stenosis was found in 30, regurgitation in 15, and a mixed lesion in 37. The distribution of angina did not differ significantly in any of these. There were 10 patients with angina (all grade 1) and no coronary artery disease. The mean systolic pressure in the pulmonary artery in this group was 48 mmHg (range 40-60 mmHg), compared with 42 (range 28-130 mmHg) for the entire group of 82 patients and 42 for those with coronary artery disease (Table 2). In 12 patients there was significant pulmonary vascular disease, and 6 of these had angina, 2 of whom had coronary artery disease. The patient with the highest systolic pressure recorded in the pulmonary artery (130 mmHg) did not have angina, possibly because of the severe incapacity and limitations to physical activities imposed by the critical degree of mitral stenosis.

Discussion

There is uncertainty as to the prevalence of coronary artery disease in the community at large, as to date there have been few publications on the subject. Among 2014 presumably healthy men aged 40 to 59 years, Erikssen et al. (1976) suspected coronary artery disease in 115 (5.5%), the criteria used being based on a typical history of angina and ischaemic changes present on either the resting electrocardiogram or during maximal bicycle exercise test. Coronary arteriography performed in 105 of these 115 patients showed arterial obstructions in more than 68 per cent. This study was carried out in Scandinavia, whereas it is generally accepted that ischaemic heart disease is much more prevalent in south-west Scotland. Lipoprotein abnormalities were found in 15 per cent of an apparently healthy population in this area (Lorimer et al., 1973), which may be related to and explain the high incidence of coronary heart disease. Twenty-six (20%) of our patients had proven coronary artery disease, the distribution being similar in those with either mitral valve or aortic valve disease.

Basta et al. (1975) encountered angina in 51 out of 88 patients (58%) with aortic valve disease and demonstrable coronary artery disease in 14 per cent. Harris et al. (1975) had a similar incidence of angina in their patients with aortic valve disease (54.9%) but the number with coronary artery disease was considerably higher (23.2%). Why coronary artery disease should be more prevalent in patients with disease of either the mitral or aortic valve has never been satisfactorily explained.

Earlier pathological studies (Horan and Barnes, 1948; Nakib et al., 1965) reported an inverse relation between the severity of aortic valve obstruction and degree of coronary artery disease, implying some kind of nebulous protection afforded by aortic stenosis on the coronary arteries. This theory was refuted in more recent studies (Linhart and Wheat, 1967; Coleman and Soloff, 1970) in which coronary artery disease was found quite commonly accompanying aortic stenosis. This certainly was so in our patients both with aortic and mitral valve disease. No convincing evidence has been forthcoming to disprove the concept that the rheumatic process itself might in fact accelerate atherosclerosis (Zeek, 1932).

Angina was a frequent complaint in our group of

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**Table 2 Associated factors accompanying angina in mitral valve disease with or without coronary artery disease**

<table>
<thead>
<tr>
<th></th>
<th>No.</th>
<th>Grade of angina</th>
<th>Pulmonary arterial pressure (mmHg)</th>
<th>Pulmonary vascular disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronary artery disease</td>
<td>18</td>
<td>2 or 3</td>
<td>42 (35-60)</td>
<td>2</td>
</tr>
<tr>
<td>No coronary artery disease</td>
<td>10</td>
<td>1</td>
<td>48 (40-60)</td>
<td>4</td>
</tr>
</tbody>
</table>
patients with aortic valve disease, the incidence being about identical in those with either aortic stenosis or regurgitation. It was certainly more common than in those with mitral valve disease, though the incidence of coronary artery disease was strikingly similar in both groups. It is now established that patients with prolapse of the mitral valve, occurring either as a congenital abnormality or resulting from recent myocardial infarction with involvement of the papillary muscles, may develop cardiac pain (Gahl et al., 1977). None of our patients belonged to either category, as in all the mitral valve itself was diseased because of previous rheumatic infection. Again, severe pulmonary hypertension with or without accompanying pulmonary vascular disease is acknowledged to produce angina-like pain without any underlying coronary artery disease (Resnekov and Falicov, 1977). Of our 12 patients with mitral valve disease who had pulmonary vascular disease, 6 had angina.

In both the aortic and the mitral valve groups there was no patient with severe angina and normal coronary arteries. The 10 patients with grade 2 or grade 3 angina all had coronary artery disease, whereas of the 45 with grade 1 (mild) coronary artery disease was present in 14 only. We conclude, therefore, that when angina occurs along with disease of the mitral valve it is much more likely to be the result of pathological changes in the coronary arteries than in aortic valve disease. Coronary artery disease was found in only 2 of our patients who had no angina, a finding similar to Proudfit and his colleagues (1966). We contend that coronary arteriography is thus unnecessary in patients without angina.

The limitations of standard 12-lead electrocardiography in the detection of ischaemic heart disease are well known. It is useful in early diagnosis of acute myocardial infarction in only 50 per cent (McGuiness et al., 1976) though the pathological Q wave itself is only 79 per cent accurate for infarction (Horan et al., 1971) and may be absent in 50 per cent of patients with left ventricular asynergy (Bodenheimer et al., 1976). Overall sensitivity for ischaemia varies from 50 to 70 per cent (Wood et al., 1950; Friesinger and Smith, 1972; Murray et al., 1976), and the resting electrocardiogram may be normal in the presence of major coronary disease (Benchimol et al., 1973). It remains to be seen whether the increased sensitivity reported with vectorcardiographic (McConahay et al., 1970; Gray et al., 1972) or surface mapping techniques based solely on the QRS complex (Flowers et al., 1976) becomes clinically useful in detecting myocardial lesions where digoxin or metabolic upset confuses interpretation of the ST segment.

References


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