Mitral Insufficiency Simulating Aortic Stenosis

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The location and transmission of the murmurs of mitral insufficiency and aortic stenosis are usually sufficiently characteristic to separate these conditions readily. Occasionally the murmur and thrill of aortic stenosis are prominent at the cardiac apex and may superficially resemble the findings in mitral insufficiency. Much less frequently the murmur of mitral insufficiency may be transmitted well to the primary aortic area. When accompanied by a thrill, considerable difficulty in distinguishing this murmur from that of aortic stenosis may be encountered.

Two cases of mitral insufficiency, presumably secondary to rheumatic heart disease, are presented in which the murmur and thrill closely simulated aortic stenosis. In these two patients the location and transmission of the murmur strongly suggested associated aortic stenosis but cardiac catheterization and particularly phonocardiography aided in the differentiation.

Case Reports

Case 1

J.L.P., a 54-year-old white man, was referred to the Duke Medical Center for evaluation of a heart murmur, first detected at age 20. He had scarlet fever as a child but no symptoms of acute rheumatic fever. For 20 years he had felt an occasional skipped heart beat and experienced infrequent episodes of rapid heart action. One month prior to admission, fatigue, exertional dyspnea, paroxysmal nocturnal dyspnea, orthopnea, weight gain, and a cough developed. These symptoms responded to digitalis and diuretics. There was no history of chest pain, trauma, syncope, edema, or illness suggestive of subacute bacterial endocarditis.

Physical examination disclosed a large, moderately obese man. The neck veins were not distended and the lungs were clear. The heart was moderately enlarged with a left ventricular heave. At the apex there was a grade III/VI harsh, moderately high-pitched holosystolic murmur and thrill; the murmur was well transmitted to the axilla and posterior thorax. An accentuated first sound and a ventricular gallop were also present at the apex. In the primary aortic area there was a grade III/VI murmur of similar quality, thought to be holosystolic, and a prominent thrill. The murmur was transmitted well into the neck but was considerably less intense along the left sternal border and below the primary aortic area on the right. The aortic second sound was of normal intensity and the pulmonic second sound was accentuated with moderate splitting. There were no diastolic murmurs. Rhythm was normal sinus with occasional extrasystoles. The heart's rate was 80 per minute and the blood pressure was 150/90 mm. Hg. The liver was not enlarged and there was no edema.

The electrocardiogram disclosed a semivertical axis, diaphragmatic wall ischemia, digitalis effect, and occasional ventricular premature contractions. Fluoroscopy and teleoentgenograms of the heart (fig. 1) demonstrated moderate enlargement of the left ventricle, slight enlargement of the left atrium, and calcification of the mitral valve.

The phonocardiogram confirmed the holosystolic character of the murmur in both the aortic and mitral areas (fig. 2). The murmur was identical in all areas and at times exhibited slight accentuation in late systole. The murmur extended a short distance beyond the aortic closure sound, which was identified by the dicrotic notch on the carotid pulse tracing. There was no accentuation in mid-systole or suggestion of a diamond shape. Moderate splitting of the second sound and accentuation of the mitral first sound were also evident. The murmur diminished in intensity and duration with ventricular premature contractions (fig. 3).

Cardiac catheterization was performed according to methods previously reported, including retrograde left ventricular catheterization and a

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catheter passed over the transseptal needle into the left atrium. Intracardiac and arterial pressures were within normal limits except for minimal elevation of right ventricular and pulmonary artery systolic pressures (pressures were 27/4 and 29/11 [17 mean] mm. Hg respectively). The left atrial mean pressure was 10 mm. Hg with “A” and “V” waves of equal magnitude. Cardiac output by the indocyanine dye method was 3.7 liters per minute. There were no aortic systolic or mitral diastolic pressure gradients when pressures were measured simultaneously proximal and distal to the valves. Also there was no systolic pulmonary valve gradient. Indocyanine dye curves following injection in the left ventricle and sampling in the left atrium and a cinefluorogram demonstrated considerable mitral insufficiency (fig. 4). No abnormality of the aortic valve was seen on selective contrast cinefluorography.

Case 2

E. L., a 40-year-old housewife, developed a heart murmur following an illness characterized by fever and arthralgia in 1956. She continued to have recurrent fever and arthralgia over the ensuing 3 years but there were no symptoms of heart failure. Because of an increase in intensity of the murmur she was referred to the Duke Medical Center. There was no history of chest trauma, syncope, or chest pain.

Physical examination revealed a moderately obese white woman. The cervical veins were not distended, and the lungs were clear. The heart was not enlarged, and there was no precordial heave. At the apex there were a thrill and a grade IV/V harsh, high-pitched systolic murmur transmitted to the axilla and posterior thorax. A harsh, grade IV/V murmur and a readily palpable thrill were present in the second interspace to the right of the sternum. The murmur was transmitted well into the neck but not below the right second interspace. The murmur was also loud along the left sternal edge although a thrill was not palpable in this area. The intensity of the aortic second sound was normal, and there were no diastolic sounds or murmurs. The rhythm was normal at a rate of 85 per minute and the blood pressure was 150/85 mm. Hg.

The electrocardiogram was normal. Telerentgenograms of the chest and cardiac fluoroscopy demonstrated minimal left ventricular enlargement (cardiothoracic ratio 15.5/29 cm.) with no enlargement of the left atrium (fig. 5).

The auscultatory findings were initially thought to represent aortic stenosis with transmission of the murmur to the apex. Accordingly, left heart catheterization was performed via combined retro-

Figure 1

Posteroanterior and right anterior oblique roentgenograms of the chest in case 1, demonstrating left ventricular and minimal left atrial enlargement.

Figure 2

Phonocardiogram in case 1. The murmur is holosystolic in the aortic area and at the apex.

Figure 3

Case 1. Phonocardiogram demonstrating decreased intensity of the apical holosystolic murmur with ventricular premature contractions (VPC).
Figure 4
Single frames from the cinefluorogram in case 1, right anterior oblique position following left ventricular injection of contrast media via retrograde catheter. Arrows denote region of the mitral valve. At the beginning of injection (A, top) the left ventricle (LV) is opacified; note calcium in the mitral valve, immediately above inferior arrow. The left atrium (LA) is subsequently opacified by reflux during systole of large amounts of contrast media from the left ventricle (B, bottom). In (A) the ventricle is in systole and (B) in diastole.

grade left ventricular and transseptal left atrial routes, and in addition selective contrast cinefluorography was utilized. Pressures in the pulmonary artery and cardiac chambers were normal. No insufficiency wave was present in the left atrial pressure tracing. No gradient across the aortic valve was demonstrable. Similarly, no pulmonary systolic or mitral diastolic gradient was demonstrable. Cinefluorography following injection of contrast material into the left ventricle demonstrated unequivocal mitral insufficiency. The aortic valve area appeared normal on contrast cinefluorograms. Indocyanine dye curves revealed no shunt and were compatible with mitral insufficiency.

Figure 5
Posteroanterior and right anterior oblique roentgenograms of the chest in case 2. There is minimal left ventricular but no demonstrable left atrial enlargement.

Figure 6
Phonocardiogram in case 2 demonstrating late accentuation of the holosystolic murmur in both mitral and aortic areas.

Because of the findings at cardiac catheterization, the phonocardiogram was reviewed. In both the aortic and mitral areas the murmur was holosystolic with accentuation in late systole (fig. 6). There was no midsystolic accentuation, and the murmur clearly extended to the aortic second sound, thus indicating that the murmur and thrill in the aortic area were not due to aortic stenosis. Proper analysis of the auscultatory findings and the phonocardiogram initially would have permitted a correct diagnosis prior to cardiac catheterization.

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Discussion

Despite the lack of pathologic confirmation, it appears quite certain that the murmur and thrill observed in the primary aortic area and transmitted into the carotid vessels in these two patients are not due to aortic valve abnormality. The absence of a systolic gradient across the aortic valve and the normal appearance of the aortic valve on selective contrast cinefluorography excluded significant aortic stenosis. Nevertheless, aortic valve deformity, insufficient to produce a stenotic pressure gradient or visible abnormality on cinefluorography, might still be the site of origin of a systolic murmur. In these two patients, however, the character of the murmur as recorded on the phonocardiogram indicates the mitral origin of the murmur and thrill in the aortic area.

Leatham \(^2\) \(^3\) \(^5\) \(^6\) was the first to describe clearly the characteristics of regurgitant versus ejection murmurs and has emphasized the usefulness of this classification of murmurs. Briefly stated, regurgitant murmurs are holosystolic, beginning with the first heart sound and ending with the second sound. Ejection murmurs begin shortly after the first sound, peak in midsystole and diminish greatly or end just prior to the second sound. In the case of aortic ejection murmurs, the diamond shape is clearly discernible with a midsystolic peak and a termination prior to aortic closure. The differences between holosystolic and ejection murmurs are illustrated diagrammatically in figures 7 and 8.

In our two patients the murmurs recorded in the aortic area were clearly holosystolic or regurgitant in character beginning with the first heart sound and extending with full intensity to the aortic closure sound. In both patients the murmur appeared to go through the aortic second sound. This phenomenon has been noted occasionally in mitral insufficiency and has been attributed to a left ventricular-left atrial pressure gradient existing for a brief interval after aortic closure. \(^2\) \(^7\) No midsystolic peak or suggestion of a diamond shape to the murmur was present. In fact, there was accentuation of the murmur in late systole, a quality that Leatham \(^6\) has considered characteristic of the murmur of mitral insufficiency.

If the murmur and thrill in the aortic area were arising from the aortic valve, some of the characteristics of an ejection murmur should be evident. Thus the phonocardiogram
ventricular septal defect, although unlikely on the basis of location of the murmurs, was excluded by cinefluorography and indicator-dilution curves. It should also be remembered that the murmur in muscular subaortic stenosis may be holosystolic and maximal at the apex or over the precordium, presumably due to distortion of the mitral valve by the hypertrophied ventricular septum.8

Isolated instances of mitral insufficiency simulating aortic stenosis have been reported in which rupture of the chordae tendineae has been the lesion responsible for insufficiency of the mitral valve.9-11 Osmundson, Callahan, and Edwards9 suggested in their patient that the murmur in the aortic area was due to a regurgitant jet striking the left atrial wall adjacent to the aortic valve and found a jet lesion in this location at autopsy. Jet lesions have been described in other case reports.10, 11

The same mechanism, that is, a regurgitant jet striking the left atrial wall in close proximity to the aortic valve inducing vibration in these structures, probably explains the aortic murmur and thrill in our patients. It is difficult to explain the diamond-shaped aortic murmur reported by Shapiro and Weiss11 in their patient with ruptured chordae tendineae and a normal aortic valve at autopsy.

Henke, March, and Hultgren12 believe that the variation in intensity of a murmur with the length of the preceding diastole is useful in differentiating the murmurs of aortic stenosis and mitral insufficiency in the presence of an irregular rhythm. With short preceding diastolic intervals the murmur of aortic stenosis is less intense in contrast to a lack of variation in intensity of the murmur in the three patients with mitral insufficiency and atrial fibrillation reported by these authors. In our case 1 the mitral insufficiency murmur decreased in length and intensity following short diastoles due to ventricular premature beats (fig. 3). Thus variation in intensity of the murmur is not a satisfactory sign in differentiating these two murmurs when the irregular rhythm is due to ventricular premature beats.

In both patients in the present report the

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Figure 8

Diagram, similar to figure 7, illustrating the holosystolic murmur in mitral insufficiency. A. Immediately after closure of the mitral valve. B. Just prior to opening of the aortic valve, showing mitral regurgitation and the murmur beginning with the first heart sound. C. Late systole. D. Immediately after closure of the aortic valve. The mitral regurgitation and murmur extend to aortic closure.
mitral insufficiency is presumed to be due to rheumatic heart disease, despite lack of laboratory or pathologic confirmation. The acquired nature of the lesion and the long history with relatively slow progression are consistent with rheumatic mitral insufficiency. Calcification of the mitral valve in case 1 would favor a rheumatic basis for the mitral insufficiency. A more rapidly progressive course would be expected if rupture of the chordae tendineae was the responsible lesion. There was no history suggestive of bacterial endocarditis or chest trauma in either patient.

Murmurs and thrills over the precordium and to the right of the sternum are common in massive mitral insufficiency with a giant left atrium. However, this situation was clearly not present in our patients, since only minimal enlargement of the left atrium could be demonstrated in one and none in the other.

We suspect that isolated mitral insufficiency masquerading as combined mitral insufficiency and aortic stenosis is far more common than the few case reports indicate. Location and transmission of the murmur in this particular circumstance are not reliable. However, careful auscultation and phonocardiography to detect the holosystolic or regurgitant nature of the murmur should uncover more instances of mitral insufficiency simulating aortic stenosis. When physiologically significant aortic stenosis is suspected, an aortic closure sound of normal intensity may be an alerting clue to consider other causes for a murmur in the aortic area.

Summary

Two patients with mitral insufficiency presumably secondary to rheumatic heart disease are reported in whom loud systolic murmurs and thrills in the aortic area simulated aortic stenosis. Left heart catheterization in both patients disclosed significant mitral insufficiency but no evidence for aortic stenosis or intracardiac shunt such as ventricular septal defect. Phonocardiography demonstrated the holosystolic characteristics of the aortic area murmurs and was indispensable in documenting their mitral origin. A regurgitant jet striking the left atrial wall in close proximity to the aortic valve, previously reported in rupture of the chordae tendineae, appears the probable mechanism of the aortic murmur and thrill.

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