Papillary Muscle Hypertrophy in Chronic Rheumatic Mitral Valve Stenosis

A Clinicopathologic Study

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SUMMARY

A quantitative pathologic study was performed on papillary muscles in 15 cases of chronic rheumatic mitral stenosis. The papillary muscles were grossly hypertrophied as compared with papillary muscles in 10 normal specimens. Hypertrophy of papillary muscles was associated with pronounced fibrosis. Angiographically, these hearts were remarkable in small left ventricular cavity with almost complete cavity elimination at end systole. Left ventricular wall dyskinesia was also present. It is probable that derangement of the mitral valve structure due to rheumatic process leads to more stress on the supporting elements, thus resulting in papillary muscle hypertrophy.

Additional Indexing Words:

Left ventricular cavity elimination Chest pain Mitral valve apparatus

CHARACTERISTICS of papillary muscles (PMs) have been the subject of several reports dealing with mitral valve prolapse¹⁾ and nonobstructive cardiomyopathy.²⁾ However, as far as we know, no detailed morphometric study has been reported regarding the PMs in rheumatic mitral stenosis (MS). The purpose of this report is the presentation of the clinical, hemodynamic, angiographic, and pathologic aspects of a selected group of patients with chronic rheumatic MS with particular reference to PMs. Attempt will be made to correlate the anatomic and clinical findings.

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MATERIALS AND METHODS

In a retrospective review of 200 left ventricular angiograms in adult patients with chronic rheumatic MS, 48 cases were found remarkable in relatively small left ventricular cavity with almost complete end-systolic cavity elimination due to what appeared to be PM hypertrophy. Review of these 48 cases showed that in 15 cases the mitral valve as well as the entire length of the PMs were available at the pathology museum. In the remaining 33 cases either the patients had not undergone operation at this center or the surgical specimen was not adequate for morphometric study developed for this project. In all 15 cases the diagnosis of rheumatic etiology was documented by previous history, laboratory data and surgical findings. The age of the patients ranged between 15 and 50 (mean 29.5) years. In these 15 cases left ventricular angiograms obtained in right anterior oblique position were critically reviewed with particular attention to the mitral valve ring, left ventricular cavity and left ventricular wall contraction pattern.

Echocardiograms of 15 cases were also analyzed qualitatively and quantitatively. Pathologic study of the specimens consisted of 3 parts:

A. Gross qualitative examination.

B. Qualitative microscopic examination of cross sections from the 3 levels described under "C" using trichrome and hematoxylin and eosin staining.

C. Morphometric study. The circumferences of the PMs were measured at 3 levels ie at the tip, 5 mm below the tip and 10 mm below the tip. The tip of the PM was defined visually at the line of junction of the mitral valve cusp and the PM. The circumferences at the 3 cross sectional levels were calculated using the formula $C = \pi \times 2R$, where $2R$ represented the arithmetic mean of 4 transverse diameters measured at intervals of 45 degree (Fig. 1), and C represented circumference.

For comparison of the PM measurements, 10 adult heart specimens were chosen which had grossly normal mitral apparatus. Eight hearts were obtained from patients who expired because of coronary artery disease, whereas in 2 cases systemic hypertension was present. The same morphometric method was applied to the PMs of these hearts.

Fig. 1. On the left hand the 3 levels used for measurement are shown. On the right hand the mean diameter of the PM is measured by averaging 4 values obtained at intervals of 45 degree.

RESULTS

The clinical findings in the 15 patients are summarized in Table I. The remarkable aspects were severe physical disability and high frequency of chest pain.

Review of the echocardiographic findings are summarized in Table II. Remarkable findings were a very small left ventricular end-diastolic dimension and a peculiar unexplained picture depicted by a high positioned echo beam (Fig. 2).

Hemodynamic values are summarized in Table III. The results of angiocardiographic evaluation are shown in Table IV. Remarkable findings were reduced mitral valve ring motion in 9 of 15 patients. Anterior and posterior left ventricular wall dyskinesia and early relaxation were present in all (Fig. 3). The results of PM measurements are shown in Table V. Note that at all 3 levels the circumference of the PMs is significantly greater than that of normal PMs.

Gross examination of the surgically removed mitral valves, disclosed a significant hypertrophy of both papillary muscles with thick, rigid, and immo-

Table I. Clinical and Electrocardiographic Data in 15 Selected Patients with Rheumatic MS

	No	%
Chest pain	10	66.6
NYHA class III	8	53.3
NYHA class IV	7	46.5
Normal sinus rhythm	7	46.5
Atrial fibrillation	8	53.3
Right atrial hypertrophy	3	20
Left atrial hypertrophy	4	26.6
Right ventricular hypertrophy	10	66.6

Table II. The Echocardiographic Findings in 15 Patients with MS (values mean $\pm$ 1SD)

EF slope	12.8 $\pm$ 2.1 mm/s
Mitral valve excursion	12.8 $\pm$ 1.8 mm
Left ventricular dimension: end-diastolic	96.8 $\pm$ 6.2 mm
end-systolic	21.6 $\pm$ 2.3 mm
Concomitant movement of M.V. leaflets	15/15
Dense echo line	13/15
Echoes in early diastole and late systole	15/15

Fig. 2. This echocardiogram is a representative picture encountered in this group of patients with MS. Note concomittant movement of the mitral valve leaflet and reduced EF slope pointing to severe mitral stenosis. Also note the constant presence of a dense echo line posterior to the mitral valve leaflets. Reflected echo beams in early diastole and late systole are also demonstrated.

Table III. Summary of the Hemodynamic Findings in 15 Patients with MS

M.V.G.	mean = 24.6 mmHg $\pm$ 6.9 (SD)
P.A.P. systolic	mean = 72.8 mmHg $\pm$ 22.04 (SD)
M.V.A.	0.95 $\pm$ 0.24 cm ²
P.V.R.	250-1,350 dynes/sec/cm ⁻⁵
C.I.	2.63 $\pm$ 0.27 L/min/M ²

Abbreviations: CI=cardiac index; MVA=mitral valve area; MVG=mitral valve gradient; PAP=pulmonary arterial pressure; PVR=pulmonary vascular resistance.

Table IV. Summary of the Angiographic Findings in 15 Patients with MS

	No	%
Restricted mitral valve ring motion	9	60
Left ventricular Dyskinesia: Anterior wall	7	46.5
Posterior wall	3	30
Anterior and Posterior wall	5	33

Diastole

Systole

Fig. 3. A typical frame of left ventricular angiogram in RAO position. Note small end-systolic cavity due to grossly hypertrophied papillary muscles.

Table V. Circumference (mean $\pm$ 1SD, cm) of the Papillary Muscles Measured at 3 Levels in 15 Patients with MS and 10 Patients with Normal Papillary Muscles

	tip	0.5 cm	1 cm	tip	0.5 cm	1 cm
Stenotic mitral valve	43.8 $\pm$ 6.27	45.40 $\pm$ 6.96	46.47 $\pm$ 8.74	42.41 $\pm$ 8.01	45.66 $\pm$ 7.05	47.36 $\pm$ 7.47
Normal mitral valves	15.67 $\pm$ 2.38	21.90 $\pm$ 3.26	26.90 $\pm$ 4.80	12.88 $\pm$ 4.30	21.04 $\pm$ 4.10	26.10 $\pm$ 4.36

P<0.001 for all values.

Fig. 4. Gross specimen of a mitral valve apparatus. Note grossly hypertrophied papillary muscles. See text for further details.

Fig. 5. Photomicrograph of a cross-section of the papillary muscle in a case of rheumatic mitral stenosis. The section was obtained at 1 cm level.

bile valve cusps. The most striking feature was the absorption and impaction of the chordae tendineae and PMs into the cusps leaving no appreciable chordae tendineae (Fig. 4). Histological sections of the PMs revealed tremendous interstitial fibrosis, especially in areas close to the tip (Fig. 5).

DISCUSSION

Hypertrophy of the PMs has been described in mitral valve prolapse¹⁾ as well as nonobstructive cardiomyopathy.²⁾ Jersaty reported prominence of the papillary muscles associated with excessive emptying of the left ventricular cavity, particularly the apex in most patients with prolapse of mitral valve. Previous angiographic studies have documented severe distortion of the mitral valve apparatus, including fusion of the PMs and the leaflets, and enlarged papillary muscles.³⁾ Boucek et al reported hypertrophy of the papillary muscles in patients with MS,⁴⁾ and Akins et al described the angiographic features of distorted mitral apparatus in this condition.⁵⁾ However besides these and a cursory remark on PM hypertrophy in rheumatic MS⁶⁾ we have not come across any quantitative study of the PMs in this disorder. In this study we documented that PM hypertrophy was present and was probably responsible for angiographic finding of end-systolic left ventricular cavity elimination. Thus in all 15 specimens the PMs were grossly hypertrophied as opposed to normal, and led to small left ventricular end-systolic dimension. This is in contrast to previous reports which showed end-systolic volumes significantly larger in MS patients than in normal controls.⁷⁾ Abnormal contraction pattern noted in our patients has been reported before.⁷⁾⁻¹⁰⁾ The reason for occurrence of PM hypertrophy is not established. Cobb¹¹⁾ and Barlow¹²⁾ have reported that the mitral apparatus is so designed that during normal cardiac function, there is minimum stress on both chordae tendineae and papillary muscles. This is due to: 1-keystone effect during systole and 2-special geometry of the mitral valve. Moreover studies of chordae tendineae and PM tension have indicated that tension is diminished in these structures after mitral valve opening. Thus severing chordae tendineae and papillary muscles increased tension in the remaining chordae tendineae and PMs during systole. Thus one can speculate that because of severe distortion of the architecture of the mitral valve and its supporting elements, these stress reducing mechanisms are eliminated. Increased stress on the PMs leads to hypertrophy. Another finding in this study is the remarkably high frequency of chest pain in patients with severe MS. Papillary muscles, being the last portions of the heart to be perfused by the coronary arterial blood, are very sensitive to myocardial ischemia.¹³⁾ Severe hypertrophy of the papillary muscle with anatomically

demonstrable fibrosis may be due to ischemia of PMs and may explain the frequency of chest pain in these patients.

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REFERENCES

1. Jeresaty RM: *In* Valvular Heart Disease, eds by Sonnenblick EH, Lesch M, Grune and Stratton, Inc, NY, p 203, 1974
2. Hess OM, Goebel NH, Luescher H, Schneider J, Angehrn W, Krayenbuehl HP: Left ventricular function in patients with hypertrophic nonobstructive cardiomyopathy. *Europ J Cardiol* **9**: 129, 1979
3. Szamosi A: Cineangiographic diagnosis of papillary muscle involvement in rheumatic mitral stenosis. *Acta Radiol Diag* **18**: 561, 1977
4. Boucek RJ, Sowton E, Eommer LS: Assessment of ventricular elements of mitral valve by left ventriculography. *Brit Heart J* **39**: 1088, 1977
5. Akins CW, Kirklin JK, Block PC, Buckley MJ, Austen WG: Preoperative evaluation of subvalvular fibrosis in mitral stenosis. *Circulation* **60** (Suppl 1) 1-71, 1979
6. Aryanpur I, Shakibi J, Yazdanyar A, Mehranpur M, Paydar, M, Azar H, Motlagh FA, Tarbiat S, Siassi B: Closed versus open mitral commissurotomy in children with rheumatic mitral stenosis. *J Thor Cardiovasc Surg* **76**: 223, 1978
7. Heller SJ, Carleton RA: Abnormal left ventricular contraction in patients with mitral stenosis. *Circulation* **42**: 1099, 1970
8. Bolen JL, Lopes MG, Harrison DC, Alderman ED: Analysis of left ventricular function in response to afterload changes in patients with mitral stenosis. *Circulation* **52**: 894, 1975
9. Curry GC, Elliott LP, Ramsey HW: Quantitative left ventricular angiocardiographic findings in mitral stenosis. *Am J Cardiol* **29**: 621, 1972
10. Hildner FJ, Javier RP, Cohen LS, Samet P, Nathan MJ, Yahr WZ, Greenberg JJ: Myocardial dysfunction associated with valvular heart disease. *Am J Cardiol* **30**: 319, 1972
11. Cobbs WBJ: Valvular heart disease. *In* The Heart, ed. by Hurst JW, Logue RB, 2nd ed, McGraw Hill Book Co, New York, 1970
12. Barlow JB, Pocock WA: The problem of nonejection systolic click and associated systolic murmur. Emphasis on billowing mitral leaflet syndrome. *Am Heart J* **90**: 636, 1975
13. Roberts WC, Cohen LS: Left ventricular papillary muscles. Description of the normal and a survey of conditions causing them to be abnormal. *Circulation* **46**: 138, 1972