Original Article

The Morphometric Measurements of the Gross Structural Changes of Mitral Valve in Valvular Stenosis With or Without Regurgitation

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Abstract:

Rheumatic mitral stenosis is still a cardiac problem in developing countries. Reconstructive and replacement surgery of the diseased valves are often needed. Most of the studies on stenotic mitral valves are echocardiographic one. Morphometric measurements of the stenotic mitral valve and comparison with that in the normal mitral valve is done in this study. Thirty seven hearts of normal adult-male unclaimed dead-bodies from the mortuary of Forensic Medicine, Dhaka Medical College, Dhaka; and twelve surgically excised stenotic mitral valves of the adult-male cardiac patients from the National Institute of Cardiovascular Diseases, Dhaka, Bangladesh were studied in fresh condition. The detail morphometric findings were compared between two groups. Though the total annular circumference was similar in both groups, yet the effective orifice area reduced significantly in stenotic valves. The anterior leaflet-area was increased, but the posterior leaflet-area was decreased in the stenotic valves. The thickness of the stenotic leaflets and chordae tendineae were increased compared to that in normal valves. The knowledge of the pathological changes of the valves would help to understand the exact pathophysiological mechanisms involved in the cardiac valve diseases.

Key words: Mitral valve, Mitral stenosis, Rheumatic valve disease.

Introduction:

Cardiac valvular diseases are one of the common causes of disability and mortality due to heart diseases in developing countries. Most of the mitral valvular diseases are rheumatic in origin. Other less common causes are calcification of mitral annulus, infective endocarditis, systemic lupus erythematosus, rheumatoid arthritis, and carcinoid heart disease1. Metabolic disorders, diet medicine (fen-phen), radiation therapy to chest and the congenital disorders may also be the reasons for valvular heart disease². Among all heart valves, the mitral valve is the most common one to be involved in rheumatic heart disease. This study provides the morphometric parameters of normal mitral valve annulus, leaflets, chordae tendineae and the papillary muscles along with the qualitative and quantitative pathological changes in the stenotic mitral valve.

Anatomy of mitral valve: The mitral valve, also known as the left atrioventricular valve, in comparison to other cardiac valves is a complex structure consisting of four major components; annulus, leaflets, chordae tendineae and papillary muscles. Also important for its

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functioning are the left atrial musculature inserting to the leaflets and the ventricular myocardium to which the papillary muscles are attached³. Left ventricular myocardium also supports directly the mural leaflet through the basal cords⁴. Thus the mitral apparatus is a complex of functionally integrated six anatomical elements working in delicate concert. These six elements are: left atrial wall, mitral annulus, mitral valve leaflets, chordae tendineae, papillary muscle and left ventricular wall^{4,5}.

Unlike the three other cardiac valves, each of which consists of three cusps or leaflets, the mitral valve has only two leaflets (bicuspid), which are quite different in shape. Owing to the oblique location of the valve, strictly speaking, its two leaflets do not occupy anterior/posterior positions. The corresponding terms for anterior and posterior are "aortic" and "mural" respectively^{3,4,6}. It is the aortic (anterior) leaflet that is in fibrous continuity with the aortic valve, and it is either a triangular or a semicircular flattened flap. The mural (posterior) leaflet is frequently double notched at its free margin giving it a triscalloped appearance (occasionally biscalloped or quadriscalloped). The scallops are usually quadrangular or triangular in shape. The indentations between leaflets at the commissures do not reach the annulus but end about 5mm short in the adult heart. These parts of the valve tissue, which are about 10mm in breadth, are referred as the commissural tissues. Because of oblique plane of the valve, these two commissural tissues are better positioned as posteromedial and anterolateral commissures or inferoseptal and superolateral commissures respectively.

The annulus of the mitral valve is best divided into two parts. The anterior third is straight and not a true annulus, but the leaflet continues with the wall of the ascending aorta (subaortic curtain), aortic valve and the membranous part of interventricular septum⁷ (Figure 1). The horseshoe-shaped posterior two thirds consist of a true bundle of fibrous tissue, but there is pronounced variations in this part of the annulus. The so-called disjunctions in this part of the annulus are "weaker" in terms of lacking a well formed fibrous cord, and these are the common areas affected in "annular dilation" in mitral regurgitation and also most often involved in calcification of the annulus. With severe dilation, the minor axis of the valve orifice becomes so distended that the leaflets are unable to approximate each other³.

Papillary muscles belong to the system of columnae carneae of the heart. They originate from the ventricular wall directly inferior to the commissural tissue of the valve. At each location the number of papillary muscles is either single or multiple (Figure 2). Chordae tendineae are the fibrous collagenous cords, and usually arise from the papillary muscles as the stem chordae. These stem chordae divide and give rise to secondary chordae, which divide further into tertiary chordae. On average, 24 stem chordae arise from both groups of papillary muscles and about 120 tertiary chordae are attached to the corresponding halves of both leaflets including the respective commissural tissue. Most of the chordae are attached to the free margin of the leaflets (marginal chordae), some to the ventricular aspect of the leaflets away from the free margin (intermediate chordae), and few chordae are attached to the basal zone of the posterior leaflet (basal chordae).

Annular orifice is the potential orifice, which lies at the level of annulus. This orifice encompasses the maximum possible area of communication, which could exist between the atrium and ventricle if the valve elements were removed. It is the effective orifice, which determines the passage of blood in atrioventricular direction. This orifice is bounded anteriorly and posteriorly by two leaflets and laterally and medially by the papillary muscles⁸.

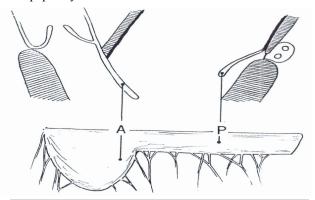


Figure 1: Schematic drawing showing the mitral leaflets. The anterior leaflet (A) is continuous with the aortic valve cusps and aortic wall. Posterior leaflet (P) is primarily continuous with the mural endocardium of the left atrium⁷.

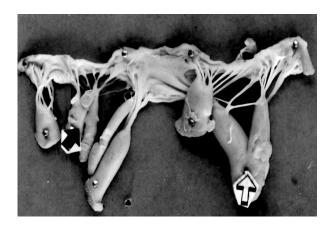


Figure 2: Multiple papillary muscles in both anterolateral (white arrow) and posteromedial (black arrow) groups.

Pathological changes of mitral valve in stenosis with or without regurgitation:

Mitral valve is the most common target for acquired heart valve disease. Rheumatic heart disease is essentially the most common cause of acquired mitral stenosis. The incidence of rheumatic fever has declined remarkably in many parts of Western world over the past several decades. Nevertheless, in developing countries and economically depressed urban areas in United States, rheumatic fever and rheumatic heart disease still remain important public health problems. Other than the rheumatic disease, degenerative valvular disease and infective endocarditis can also cause the pathological changes in the mitral valve.

In rheumatic disease, the valvular change principally takes the form of deforming fibrotic mitral stenosis. In acute rheumatic fever the valves are red and swollen, and are characterized by discrete inflammatory lesionscalled Aschoff bodies found in any of the three layers of the heart (including the valves). There are fibrinoid inflammation and necrosis within leaflets and chordae tendineae. Thrombotic vegetation appears along the line of closure of the leaflets. These vegetation are irregular (1 to 2mm), warty projections probably due to precipitation of fibrin.

In chronic cases the repeated injury and healing leads to excessive fibrosis of the leaflet and chordae tendineae. The Aschoff bodies are replaced by fibrous scar. The leaflets become leathery, and permanently thickened and retracted with marked commissural fusion. Once damage has developed on a valve, the altered hemodynamic stresses on the valve perpetuate and extend the damage even in the absence of continuing rheumatic lesion. Microscopic examination shows neovascularization of the thickened leaflets. Gross deposition of calcium in the leaflets occurs in more than 60% of cases of mitral stenosis. Fibrous bridging across the valvular commissures and calcification create

"fish-mouth" or "button-hole" stenosis (Figure 3). The chordae are also thickened, shortened and frequently fused each other. Thus the sequels of the chronic valvular involvement are either mitral stenosis or mitral regurgitation or both.

In degenerative valve diseases the changes are in extracellular matrix that affects the integrity of the valve. Degenerative changes in the cardiac valves probably are an inevitable part of the aging process, because of the repetitive mechanical stresses to which valves are subjected-40 million beats per year. These changes include calcification of the mitral annulus, decreased numbers of valve fibroblasts and myofibroblasts, increased proteoglycan, and diminished collagen and elastin (myxomatous degeneration), or excess fibrosis and scarring of the leaflets. In some cases there may be degradation of extracellular matrix due to some endopeptidase (metalloproteinases)⁹.

In infective endocarditis, the microbial invasion of heart valves or mural endocardium characteristically results in bulky friable vegetations composed of necrotic debris and thrombus. Though the infective endocarditis can develop on previously normal valves, but cardiac abnormalities predispose to such infections.

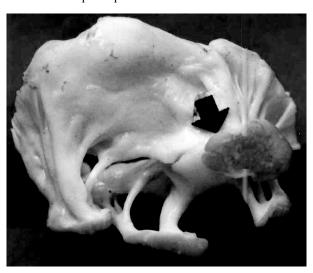


Figure 3: Ventricular view of a stenotic mitral valve showing the 'button-hole' opening of the effective orifice, the chordae are thickened and shortened so much that the papillary muscle is fused with the leaflet (black arrow).

Materials and Method:

Thirty seven hearts from apparently-normal adult-male unclaimed dead bodies were collected during autopsy in the mortuary of Forensic Medicine, Dhaka Medical College, Bangladesh in the year 1999. After general

inspection of the unfixed hearts, the mitral valves were excised for study in fresh condition. Twelve surgically excised stenotic mitral valves from the adult-male cardiac patients were collected for gross examination in fresh condition. All these patients went valve replacement operation in the National Institute of Cardiovascular Diseases (NICVD), Dhaka, Bangladesh in the years of 1998 and 1999. These patients were suffering from mitral stenosis with or without mitral regurgitation. The echocardiographic measurements of the annular diameter and the effective orifice area of the mitral valve were noted from the preoperative echocardiographic reports of these patients. All the study specimens were collected with proper permission from the head of the respective departments.

In the normal unfixed hearts, the mitral orifice was exposed after excision of the left atrial wall from the mitral annular ring. The diameter of the effective orifice was measured by passing a 'sizer' (Figure 4) through the orifice, freely but not too much loosely. Effective orifice area was calculated from the measured diameter. The annular ring was then excised from both ventricular wall and root of the aorta. Left ventricle was then opened dissecting it at the left border to expose the valve complex. The papillary muscles were excised at their bases and the valve complex was freed from the rest of the ventricle. The posterior leaflet was divided at its mid-region for further study.

All the stenotic mitral valves were observed for any calcification. The shape of the effective orifice was noted. When the valve was pliable, the diameter of the effective orifice area was measured with the 'sizer' and the area was calculated from the measured diameter. When the valve was hard enough to impede the passage of 'sizer' properly, the orifice-outline was traced on a transparent sheet and the area was measured by fixing the tracing over a graph paper (mm2). The posterior leaflet was then divided at the mid region and was spread for further examination. As during operative excision approximately 0.2 cm of tissue from the basal part of the valve leaflets was left in situ, so during study 0.2 cm was added to the height at the cut margins of the leaflets. Thus the values obtained by this measurement approximated to that of total valve tissue.

For further study of both normal and stenotic valve, the numbers of scallops in the posterior leaflet were counted. Anterior and posterior annular lengths were noted, and the annular circumference, diameter and area were calculated. The maximum thickness of leaflets was also measured. The leaflet-areas were measured by tracing the outline of the leaflets on transparent sheet and fixing it on a graph paper (mm²). The numbers of chordae attached to the leaflets and commissural tissue were examined and counted. The length of the marginal

chordae of the anterior leaflet was measured. The thickness of the stem-chordae branches was measured. It is worthy to note here that in normal valve these thicknesses were noted if they are more-or-less than 0.1 cm

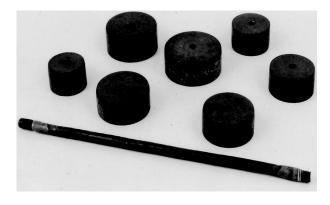


Figure 4: Sizers of different diameters, used in measuring the diameter of the effective orifice of the mitral valves.

Results:

The effective orifice area in the stenotic valves was significantly low than that in the normal valves (Table I). In one stenotic valve the orifice was deformed to "button-hole" slit (Figure 3). Though the total annular circumference of the stenotic valves was similar to that in normal valves, yet there were significant variations in the anterior and posterior annular lengths in the stenotic valves, when measured separately. The anterior annulus was elongated significantly in the stenotic valves than that in the normal valves. On the contrary, the posterior annulus was shortened in the stenotic valves than that in the normal (Table I).

The anterior leaflet-area (mean \pm SD) increased significantly in stenotic valve (6.25±1.11 cm²) than that in normal valve (5.18±0.77 cm²). But the changes in posterior leaflet-area were reverse, reduced in stenotic valve $(4.73\pm1.25 \text{ cm}^2)$ than that in normal $(5.27\pm0.59 \text{ m}^2)$ cm²). Posterior leaflets in most of the normal valve were triscalloped (70.3%), followed by biscalloped (24.3%) and quadriscalloped (5.4%) leaflets. The scallopappearance in stenotic valves was lost due to excess fibrosis. Both anterior and posterior leaflets of the stenotic valves were thickened more than 0.25 cm (up to 1.1 cm) and were non-pliable due to excess fibrosis and calcification (Figure 5), but the most of the normal valve-leaflets were less than 0.25 cm in thickness. In most of the stenotic valves the leaflets were fused in such a way that the commissural tissues were obliterated, and the heights of the fused commissures were increased and as a result, the intercommissural diameter of the effective orifice was diminished.

In majority of the stenotic valves the number of stem chordae tendineae (excluding the basal chordae) were only 5 to 15 in contrast to 20 to 30 in majority of the normal valves (Table I). The lengths and thickness of the marginal chordae to the anterior leaflet were measured; it was noted that these chordae arising from both anterior and posterior papillary muscles were shortened significantly in the stenotic valves (0.98 and 1.44 cm respectively on average) in comparison to that in normal valves (1.5 and 1.73 cm respectively on average). The average thickness of branches of the stem chordae in the stenotic valve was 0.41 cm (the highest was up to 1.05 cm) in contrast to that in normal valve (<0.1 cm). In stenotic valve the chordae were fibrosed and fused one with other resulting in its shortening and thickening (Figure 6).

Table 1: Comparison of the annulus, effective orifice area and stem chordae in normal mitral valve with that in the stenotic mitral valve

Parameters	Normal mitral valve		Stenotic mitral valve	
	Range	Mean±SD	Range	Mean±SD
Annular circum	8.4-12.0	10.2±0.78	6.9-12.6	10.04±1.62
ference (cm)				
Anterior annular	3.5-5.6	4.26 ± 0.44	3.45-6.38	4.87 ± 0.85
length (cm)				
Posterior annular	4.4-7.5	5.94 ± 0.64	3.1-7.0	5.13±1.15
length (cm)				
Effective orifice	4.16-8.56	6.89 ± 1.24	0.52-3.87	1.22 ± 1.04
area (cm²)				
Number of stem	17-37	23.89±4.56	2-24	10.33±6.15
chordae				
(excluding basal				
chordae) [†]				

[†] Basal chordae were not available with the stenotic valve leaflets.

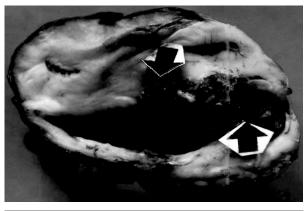


Figure 5: Atrial view of a stenotic mitral valve leaflets showing the ulcerated calcified areas (arrows) in the anterior leaflet and posteromedial commissural tissue.



Figure 6: Ventricular view of a stenotic mitral valve showing the shortened and thickened chordae (arrows).

Discussion:

Though the annular total circumference in stenotic valve remains similar to that in normal valve, yet the effective orifice area reduces markedly, and this is primarily due to obliteration of the commissural tissue as a result of fusion of anterior and posterior leaflets, and the increase in height of fused commissure. Besides these, the leaflets are also non-pliable due to excess fibrosis and calcification. The effective orifice areas in the stenotic valves measured in echocardiography (0.6-2.5 cm²) were similar to that measured manually (0.52-3.87 cm²) in this study. At the same time, the annular diameters measured in echocardiography (2.90-4.10 cm) were also similar to that measured manually (2.61-4.01 cm).

It is interesting to note in this study that, the annular circumference of the stenotic valves was found to be more variable (± 1.62 SD) compared to that in normal valves (± 0.78 SD). The posterior annulus tends to contract, on the other way, the anterior annulus tends to elongate. The later might be one of the causes for regurgitation in the stenotic mitral valve, other than the shortening of chordae and hardening of the leaflets.

The leaflet-areas of both the leaflets, in the normal valves, are almost similar to each other and the ratio of this total leaflet-area to the annular-area is $1.4\,\mathrm{cm}^2$ during ventricular diastole and increases up to $2.5\,\mathrm{cm}^2$ during systole. In mitral stenosis, the anterior leaflet-size increases and its basal part is deformed to convex toward the left atrium. In early ventricular diastole this deformity might buckle in the opposite direction producing the "opening snap". Sometimes this deformity might be such that the prominence of this leaflet is pressed against the base of the posterior leaflet during ventricular systole¹⁰.

Shortening and thickening of the chordae are the common features in the stenotic mitral valves. Their number also reduces due to fusion with one another. In some stenotic valves, due to excessive fibrosis and shortening of the chordae, the papillary muscles appear to be fused with the leaflets (Figure 3).

Conclusion:

The knowledge of the pathological changes of the valves would help to understand the exact pathophysiological mechanisms involved in the cardiac valve diseases. The moderate to severe pathological changes of the cardiac valves, now a days, are mostly detected in either transthoracic or transesophageal echocardiography. The detail morphometric disfiguration of the diseased valve and its comparison with that of the echocardiographic findings would be helpful for both the reconstructive and the replacement surgery of the diseased cardiac valves.

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