

ANATOMICAL AND HEMODYNAMIC PHENOTYPES OF THE MITRAL VALVE IN PATIENTS WITH MITRAL REGURGITATION

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ABSTRACT

Background: Mitral regurgitation (MR) is a heterogeneous valvular disorder in which structural abnormalities of the mitral valve apparatus produce distinct alterations in transvalvular flow and cardiac loading. Anatomical phenotypes, including rheumatic leaflet restriction, myxomatous mitral valve prolapse, and flail leaflet, may therefore have different echocardiographic and hemodynamic characteristics. Characterizing these phenotypes may improve understanding of the mechanisms underlying MR and its associated cardiac remodeling.

Objective: To characterize the anatomical phenotypes of the mitral valve among patients with mitral regurgitation and to assess their associated echocardiographic and hemodynamic characteristics.

Methods: A hospital-based observational cross-sectional study was conducted at Mardan Medical Complex, Mardan, Pakistan, from June to December 2021. A total of 120 adult patients with echocardiographically confirmed mitral regurgitation were included. Two-dimensional transthoracic echocardiography was used to assess mitral leaflet morphology and mobility, scallop involvement, commissures, chordae tendineae, papillary muscles, mitral annular dimensions, left atrial and left ventricular dimensions, and left ventricular ejection fraction. Doppler echocardiography was used to assess peak transmitral velocity, mean transmitral gradient, effective regurgitant orifice area (EROA), and regurgitant volume. Patients were categorized according to their predominant anatomical phenotype. Continuous and categorical variables were summarized using appropriate descriptive statistics, with comparisons performed using parametric or non-parametric tests as appropriate.

Results: Of the 120 patients, 78 (65.0%) were male and 42 (35.0%) were female. Rheumatic leaflet restriction was the predominant anatomical phenotype, identified in 78 patients (65.0%), followed by myxomatous mitral valve prolapse in 22 (18.3%) and flail leaflet in 12 (10.0%). The remaining patients had less frequent phenotypes, including functional/secondary MR, mixed rheumatic disease, annular dilatation, and ischemic tethering. Significant differences between the major anatomical phenotypes were observed for mitral annular dimension ($p=0.009$), mitral valve area ($p<0.001$), peak transmitral velocity ($p<0.001$), mean transmitral gradient ($p<0.001$), EROA ($p<0.001$), regurgitant volume ($p<0.001$), left atrial dimension ($p=0.023$), and LV end-diastolic dimension ($p=0.003$). EROA was positively correlated with left atrial dimension ($\rho=0.290$, $p=0.001$) and LV end-diastolic dimension ($\rho=0.253$, $p=0.005$), while regurgitant volume was positively correlated with LV end-diastolic dimension ($\rho=0.259$, $p=0.004$).

Conclusion: Distinct mitral valve anatomical phenotypes demonstrate different echocardiographic and hemodynamic profiles in patients with mitral regurgitation. The predominance of rheumatic leaflet restriction in this cohort highlights the continuing importance of rheumatic mitral valve disease in the regional population. The associations between regurgitant parameters and cardiac chamber dimensions further support the physiological link between abnormal valve structure, altered transvalvular flow, and cardiac remodeling. An integrated echocardiographic assessment combining mitral valve morphology, Doppler hemodynamics, regurgitation severity, and chamber remodeling may provide a more comprehensive characterization of mitral regurgitation.

KEYWORDS: Mitral regurgitation; Mitral valve anatomy; Echocardiography; Mitral valve phenotypes; Rheumatic heart disease; Mitral valve prolapse; Flail leaflet; Hemodynamics; Cardiac remodeling; Doppler echocardiography.

INTRODUCTION

The mitral valve is a dynamic atrioventricular structure composed of the annulus, anterior and posterior leaflets, chordae tendineae, papillary muscles, and adjacent left atrial and left ventricular myocardium. Its function depends on coordinated changes in geometry throughout the cardiac cycle, allowing low-resistance ventricular filling during diastole and effective leaflet coaptation during systole. Variations in leaflet, scallop, annular, commissural, and subvalvular anatomy can therefore alter coaptation and produce distinct patterns of mitral regurgitation (MR) [1,2]. Three-dimensional echocardiographic studies have demonstrated considerable variation in leaflet and scallop morphology and have improved anatomical characterization of complex mitral-valve lesions [3,4].

MR may result from primary structural abnormalities, including myxomatous degeneration, mitral-valve prolapse, flail leaflet, chordal rupture, or rheumatic disease, or from secondary alterations in left ventricular or left atrial geometry [5,6]. In secondary MR, ventricular or atrial remodeling may cause annular dilatation, papillary-muscle displacement, and leaflet tethering, resulting in incomplete systolic coaptation [7]. Rheumatic disease remains particularly relevant in South Asia; a systematic review and meta-analysis reported a substantial regional burden of rheumatic heart disease, including a relatively high prevalence in Pakistan [8]. Rheumatic involvement may produce leaflet thickening, commissural fusion, restricted mobility, and subvalvular shortening, resulting in stenotic, regurgitant, or mixed physiology [5,6].

The hemodynamic expression of mitral-valve disease is closely related to its structural phenotype. Reduced mitral-valve opening increases resistance to diastolic flow and may increase transmitral velocity and pressure gradients; a mitral valve area ≤ 1.5 cm² is generally considered consistent with clinically significant mitral stenosis [6,9]. In MR, incomplete systolic coaptation produces abnormal backward flow into the left atrium. Current guideline-based criteria supporting severe MR include an effective regurgitant orifice area (EROA) ≥ 0.40 cm², regurgitant volume ≥ 60 mL/beat, regurgitant fraction $\geq 50\%$, and vena contracta width ≥ 0.7 cm, although severity should be determined using an integrated multiparametric approach [5,10]. Chronic significant MR produces left-atrial and left-ventricular volume overload, which may lead to chamber dilatation, adverse remodeling, pulmonary hypertension, and eventual ventricular dysfunction [11,12]. Importantly, a preserved LVEF does not necessarily exclude early ventricular dysfunction in severe MR; the ACC/AHA guideline considers LVEF $\leq 60\%$ or LV end-systolic diameter ≥ 40 mm as evidence of LV systolic dysfunction in severe primary MR [5].

Echocardiography is central to the anatomical and physiological assessment of mitral-valve disease. Transthoracic echocardiography provides information regarding leaflet morphology and motion, annular dimensions, chamber remodeling, ventricular function, and MR severity, while Doppler techniques provide quantitative information regarding transvalvular flow and regurgitation [10,13]. Transesophageal and three-dimensional echocardiography provide additional anatomical detail when conventional imaging is insufficient, particularly for identifying scallop involvement, commissural disease, prolapse, flail segments, annular abnormalities, and subvalvular pathology [3,4,14]. Assessment of chamber dimensions and ventricular function further allows the anatomical lesion to be interpreted in the context of its physiological consequences [13].

Despite advances in echocardiographic imaging, regional data describing the relationship between mitral-valve anatomical phenotypes and their hemodynamic expression remain limited. In particular, there is limited published information characterizing these patterns among patients from Mardan and surrounding areas of Khyber Pakhtunkhwa, Pakistan. Therefore, the present study was designed to characterize the anatomical and hemodynamic phenotypes of the mitral valve among adult patients with MR at Mardan Medical Complex and to describe their associated echocardiographic features. By integrating structural findings with cardiovascular physiological parameters, this study aims to provide a clinically relevant description of mitral-valve disease in this regional population.

2. MATERIALS AND METHODS

2.1 Study Design and Setting

A hospital-based observational cross-sectional study was conducted at Mardan Medical Complex, Mardan, Khyber Pakhtunkhwa, Pakistan. The study was conducted over a seven-month period from June to December 2021. The study was designed to characterize the anatomical phenotypes of the mitral valve and describe their associated echocardiographic and hemodynamic features among adult patients with mitral regurgitation (MR).

2.2 Study Population

The study included 120 adult patients with echocardiographically identified mitral regurgitation who were evaluated at the study site during the study period. Participants were eligible if they were aged >18 years and had adequate echocardiographic visualization of the mitral valve and relevant cardiac structures. Available clinical records were also required for inclusion.

2.3 Eligibility Criteria

Patients were included if they were adults aged >18 years with documented mitral regurgitation and adequate echocardiographic visualization for assessment of mitral-valve morphology and cardiac function.

Patients were excluded if they had undergone previous mitral-valve surgery or had a prosthetic mitral valve, major congenital cardiac abnormalities, inadequate visualization of the mitral valve, severe technical limitations affecting echocardiographic assessment, or incomplete clinical or echocardiographic records.

2.4 Echocardiographic Assessment

All eligible patients underwent echocardiographic assessment for characterization of mitral-valve anatomy and associated cardiac functional parameters. Two-dimensional transthoracic echocardiography was used to assess the mitral annulus, anterior and posterior leaflets, leaflet thickness and mobility, scallop involvement, commissures, chordae tendineae, and papillary muscles. The presence of leaflet prolapse, flail segments, restriction, annular abnormalities, and subvalvular abnormalities was documented.

Cardiac chamber dimensions, including left-atrial and left-ventricular dimensions, were assessed, and left-ventricular ejection fraction (LVEF) was recorded as an indicator of ventricular systolic function. Doppler echocardiography was used to assess transmitral flow characteristics, including peak transmitral velocity and mean transmitral gradient, as well as parameters relevant to assessment of mitral regurgitation.

Where available and clinically indicated, transesophageal and three-dimensional echocardiography were used to provide additional anatomical characterization, particularly in patients with complex leaflet, scallop, commissural, annular, or subvalvular abnormalities.

2.5 Classification of Mitral-Valve Anatomical Phenotypes

Mitral-valve lesions were categorized according to their predominant anatomical and pathophysiological characteristics. The recorded phenotypes included rheumatic leaflet restriction, myxomatous mitral-valve prolapse, flail leaflet, functional or secondary mitral regurgitation, mixed rheumatic disease, annular dilatation, and ischemic leaflet tethering.

The classification was based on the echocardiographic assessment of leaflet morphology and mobility, annular geometry, commissural involvement, and subvalvular anatomy. Where more than one abnormality was present, the predominant anatomical phenotype was considered according to the available clinical and echocardiographic assessment.

2.6 Hemodynamic and Physiological Assessment

The anatomical findings were interpreted in relation to their potential hemodynamic consequences. Mitral-valve morphology was assessed in the context of transmitral blood flow, valve opening and closure, and the development of regurgitant flow during systole. Echocardiographic parameters including transmitral peak velocity, mean transmitral gradient, and measures of mitral regurgitation were considered as indicators of altered transvalvular flow and pressure relationships.

The physiological consequences of significant MR were further assessed through evaluation of left-atrial and left-ventricular dimensions and LVEF. These parameters provided an echocardiographic assessment of chamber remodeling and ventricular systolic function associated with chronic mitral-valve disease.

2.7 Data Collection

Clinical and echocardiographic information was obtained from the available patient records and echocardiographic assessments performed during the study period. Demographic characteristics, mitral-valve anatomical findings, and relevant echocardiographic parameters were recorded for analysis. Data were organized according to the predefined anatomical phenotypes.

2.8 Statistical Analysis

Categorical variables were summarized as frequencies and percentages. Continuous variables were planned to be expressed as mean $\pm$ standard deviation (SD) for normally distributed data or as median with interquartile range (IQR) for non-normally distributed data. Comparisons between categorical variables were performed using the chi-square test or Fisher's exact test, where appropriate. For continuous variables, the independent-samples t-test or Mann–Whitney U test was considered according to data distribution, while analysis of variance (ANOVA) or the Kruskal–Wallis test was used for comparisons involving more than two groups. A two-sided p-value <0.05 was considered statistically significant.

2.9 Ethical Considerations

The study was conducted in accordance with the applicable institutional ethical requirements. Ethical approval was obtained from the relevant institutional authority before commencement of the study. Patient information was handled confidentially, and informed consent was obtained where required according to the original study protocol and institutional requirements.

3. RESULTS

3.1 Demographic and Anatomical Characteristics

A total of 120 adult patients with mitral regurgitation were included in the study. The mean age was 33.97 ± 9.43 years, with an age range of 18–50 years. Among the participants, 78 (65.0%) were male and 42 (35.0%) were female.

Rheumatic leaflet restriction was the predominant anatomical phenotype, occurring in 78 (65.0%) patients, followed by myxomatous mitral-valve prolapse (MVP) in 22 (18.3%) and flail leaflet in 12 (10.0%). The remaining eight patients (6.7%) had less frequent phenotypes, including functional/secondary MR, mixed rheumatic disease, annular dilatation, and ischemic leaflet tethering.

The principal demographic and anatomical characteristics are presented in **Table 1**.

Table 1. Demographic and anatomical characteristics of the study population

Characteristic	Overall (n=120)	n (%) / Mean $\pm$ SD
Age, years	120	33.97 ± 9.43
Male sex	78	65.0
Female sex	42	35.0
Anatomical phenotype		
Rheumatic leaflet restriction	78	65.0
Myxomatous/MVP	22	18.3
Flail leaflet	12	10.0

Characteristic	Overall (n=120)	n (%) / Mean $\pm$ SD
Functional/secondary MR	4	3.3
Mixed rheumatic disease	2	1.7
Annular dilatation	1	0.8
Ischemic tethering	1	0.8
Leaflet mobility		
Restricted	—	Predominantly observed in rheumatic disease
Excessive	—	Predominantly observed in MVP/flail disease
Adequate echocardiographic visualization	120	100.0

MVP, mitral-valve prolapse; MR, mitral regurgitation; SD, standard deviation.

3.2 Echocardiographic and Hemodynamic Findings

The overall mean mitral annular diameter was 32.44 ± 4.47 mm. The mean mitral-valve area was 2.18 ± 1.22 cm², while mean peak transmitral velocity and mean transmitral gradient were 1.49 ± 0.38 m/s and 4.44 ± 2.54 mmHg, respectively.

The mean EROA was 0.34 ± 0.17 cm² and the mean regurgitant volume was 43.07 ± 19.80 mL/beat. The mean LVEF was $59.31 \pm 6.90\%$. Mean left-atrial dimension, LV end-diastolic dimension, and PASP were 46.21 ± 6.96 mm, 53.48 ± 6.93 mm, and 42.66 ± 12.12 mmHg, respectively.

Detailed echocardiographic and hemodynamic characteristics are presented in **Table 2**.

Table 2. Overall echocardiographic and hemodynamic characteristics

Echocardiographic parameter	Mean $\pm$ SD	Median (IQR)	Range
Mitral annular diameter, mm	32.44 ± 4.47	32.50 (6.05)	24.0–45.0
Mitral valve area, cm ²	2.18 ± 1.22	1.72 (2.21)	0.75–4.65
Peak transmitral velocity, m/s	1.49 ± 0.38	1.45 (0.62)	0.75–2.52
Mean transmitral gradient, mmHg	4.44 ± 2.54	4.00 (4.10)	0.8–12.1
EROA, cm ²	0.34 ± 0.17	0.32 (0.17)	0.08–0.90
Regurgitant volume, mL/beat	43.07 ± 19.80	40.80 (22.82)	15.0–110.3
LVEF, %	59.31 ± 6.90	59.50 (10.12)	42.2–75.0
LA dimension, mm	46.21 ± 6.96	46.00 (10.25)	31.0–64.0
LVEDD, mm	53.48 ± 6.93	53.00 (9.77)	40.0–69.0
PASP, mmHg	42.66 ± 12.12	41.00 (15.00)	23.0–75.0

IQR, interquartile range; EROA, effective regurgitant orifice area; LVEF, left-ventricular ejection fraction; LA, left atrial; LVEDD, left-ventricular end-diastolic diameter; PASP, pulmonary artery systolic pressure.

3.3 Comparison of Echocardiographic Parameters According to Anatomical Phenotype

Significant differences were observed among the four major anatomical phenotype groups for several echocardiographic parameters. Mitral annular diameter differed significantly between groups (Kruskal–Wallis $H=11.686$, $p=0.009$).

Mitral-valve area demonstrated a highly significant difference between phenotype groups ($H=82.104$, $p<0.001$). Similarly, peak transmitral velocity and mean transmitral gradient differed significantly between groups ($H=62.935$, $p<0.001$ and $H=64.553$, $p<0.001$, respectively).

Significant between-group differences were also observed for EROA ($H=56.079$, $p<0.001$) and regurgitant volume ($H=46.872$, $p<0.001$). Left-atrial dimension ($H=9.492$, $p=0.023$) and LV end-diastolic dimension ($H=13.988$, $p=0.003$) also differed significantly among phenotypes.

In contrast, no statistically significant differences were observed for LVEF ($H=6.240$, $p=0.101$) or PASP ($H=4.731$, $p=0.193$).

3.4 Correlation Between Hemodynamic and Cardiac Remodeling Parameters

Spearman correlation analysis demonstrated a positive correlation between EROA and left-atrial dimension ($\rho=0.290$, $p=0.001$) and between EROA and LV end-diastolic dimension ($\rho=0.253$, $p=0.005$). Regurgitant volume was also positively correlated with LV end-diastolic dimension ($\rho=0.259$, $p=0.004$).

Mitral-valve area was positively correlated with LV end-diastolic dimension ($\rho=0.316$, $p<0.001$), whereas peak transmitral velocity showed a negative correlation with LV end-diastolic dimension ($\rho=-0.259$, $p=0.004$). Mean transmitral gradient was also negatively correlated with LV end-diastolic dimension ($\rho=-0.220$, $p=0.016$).

No significant correlations were observed between EROA and PASP ($\rho=-0.025$, $p=0.788$), EROA and LVEF ($\rho=0.111$, $p=0.227$), or regurgitant volume and PASP ($\rho=-0.017$, $p=0.857$).

4. DISCUSSION

The present study evaluated the relationship between mitral valve anatomical phenotypes and their associated echocardiographic and hemodynamic characteristics in patients with mitral regurgitation (MR). The principal findings were that rheumatic leaflet restriction represented the predominant anatomical phenotype, followed by myxomatous mitral valve prolapse (MVP) and flail leaflet phenotypes. Significant differences were observed among the anatomical phenotypes in mitral valve area (MVA), peak transmitral velocity, mean transmitral gradient, effective regurgitant orifice area (EROA), regurgitant volume, mitral annular dimension, left atrial (LA) dimension, and left ventricular end-diastolic dimension (LVEDD). Collectively, these findings support an important relationship between mitral valve morphology, valve mechanics, intracardiac flow, and subsequent cardiac remodeling.

The predominance of rheumatic leaflet restriction in this cohort is consistent with the continuing burden of rheumatic heart disease in South Asia. A systematic review and meta-analysis reported substantial regional prevalence of rheumatic heart disease, including a relatively high prevalence in Pakistan, emphasizing the continuing importance of rheumatic valvular pathology in the region [16]. Rheumatic involvement can produce leaflet thickening, reduced leaflet mobility, commissural fusion, and subvalvular abnormalities, resulting in progressive alteration of the normal mitral valve architecture [17]. From a physiological perspective, these structural changes reduce the effective diastolic opening of the valve and increase resistance to left ventricular filling. Therefore, the predominance of a restrictive phenotype in a regional hospital-based cohort may have important implications for the hemodynamic profile encountered in clinical practice.

The significant differences in MVA, peak transmitral velocity, and mean transmitral gradient among the anatomical phenotypes further demonstrate the functional consequences of structural valve abnormalities. Echocardiographic assessment of rheumatic mitral disease relies substantially on valve morphology, valve area, and transmitral gradients because these parameters reflect the interaction between valve geometry and transvalvular flow [17,18]. When the effective mitral orifice is reduced, preservation of forward diastolic flow requires an increase in blood velocity across the valve, resulting in a higher pressure gradient. This relationship provides a physiological explanation for the markedly different transmitral profiles observed between the restrictive rheumatic phenotype and the predominantly regurgitant phenotypes. The relationship between mitral valve area and transmitral gradient is also influenced by cardiac rhythm and flow conditions, emphasizing that these measurements should be interpreted within their clinical and hemodynamic context [19].

In contrast to rheumatic restriction, myxomatous MVP and flail leaflet disease primarily represent abnormalities of systolic leaflet coaptation. Excessive leaflet motion, prolapse, abnormal chordal support, or chordal rupture can allow blood to move backward from the left ventricle into the left atrium during systole [20,21]. Three-dimensional transesophageal echocardiography has demonstrated substantial heterogeneity in the morphology and dynamic behavior of prolapsing and flail mitral valves, highlighting the importance of detailed anatomical characterization when evaluating degenerative MR [21]. Thus, the differences in EROA and regurgitant volume observed between phenotypes are physiologically consistent with the distinction between restricted diastolic opening and impaired systolic closure.

The association of EROA with LA dimension ($p=0.290$, $p=0.001$) and LVEDD ($p=0.253$, $p=0.005$), together with the correlation between regurgitant volume and LVEDD ($p=0.259$, $p=0.004$), provides an important link between valvular dysfunction and cardiac remodeling. In chronic MR, blood ejected backward into the left atrium during systole returns to the left ventricle during diastole, thereby increasing ventricular preload and producing chronic volume loading. Persistent volume overload can lead to progressive enlargement of both the left atrium and left ventricle [22,23]. Contemporary echocardiographic assessment therefore emphasizes not only quantification of MR but also evaluation of the size and function of the adjacent cardiac chambers because chamber remodeling represents an important consequence of chronic regurgitant loading [22,23].

The significant differences in mitral annular dimension and LVEDD among the anatomical phenotypes further emphasize the dynamic relationship between the valve and the ventricle. The mitral annulus, chordae, papillary muscles, leaflets, and surrounding ventricular myocardium form an integrated functional apparatus rather than independent anatomical structures. In secondary MR, LV dilatation may displace the papillary muscles, increase leaflet tethering, alter annular geometry, and ultimately impair systolic coaptation [11]. Consequently, ventricular remodeling can become both a consequence and a contributor to MR. This ventricular–valvular interaction is particularly relevant when interpreting phenotypes in which leaflet morphology alone does not fully explain the severity of regurgitation.

LVEF did not differ significantly among the anatomical phenotypes ($p=0.101$). This finding should be interpreted cautiously because preserved LVEF does not necessarily indicate the absence of clinically important ventricular consequences from chronic MR. In MR, the left ventricle ejects blood both forward into the aorta and backward into the relatively low-pressure left atrium. Consequently, conventional LVEF may remain apparently preserved during compensated disease even while effective forward output and myocardial function are changing. Current approaches to primary MR therefore consider LVEF together with LV dimensions, MR severity, symptoms, and other markers of ventricular remodeling rather than using LVEF as an isolated measure [17,19].

PASP did not differ significantly among phenotypes ($p=0.193$), and EROA was not significantly correlated with PASP ($p=0.025$, $p=0.788$). The absence of a significant association in this analysis should not be interpreted as evidence that MR is unrelated to pulmonary hemodynamics. Pulmonary hypertension in chronic MR develops

through a complex interaction involving LA pressure, LA compliance, ventricular function, and pulmonary vascular adaptation, and its relationship with MR severity may therefore vary according to disease stage and chronicity [22]. The cross-sectional design, heterogeneous anatomical phenotypes, and potentially variable duration of disease may have contributed to the absence of a statistically significant association in the present analysis.

From a clinical and physiological perspective, these findings support an integrated approach to mitral valve assessment. Anatomical characterization should include the leaflets, scallops, commissures, chordae, papillary muscles, and annulus, while functional assessment should incorporate transmitral flow, MVA, mean gradient, EROA, regurgitant volume, LA and LV dimensions, and ventricular systolic function. Echocardiography is particularly valuable because it can simultaneously characterize the mechanism of MR, quantify its hemodynamic consequences, and assess the response of the cardiac chambers [23]. Three-dimensional echocardiography may provide additional anatomical information in complex prolapse, flail, and tethering patterns and may improve pre-intervention characterization [21].

Several limitations should be considered. The study was conducted at a single center and included a relatively modest sample, limiting generalizability beyond the study population. Its cross-sectional design prevents determination of temporal or causal relationships between anatomical abnormalities and cardiac remodeling. Advanced three-dimensional or transesophageal imaging was not uniformly available, which may have limited detailed characterization of complex mitral anatomy. In addition, the relatively small number of patients within some less common anatomical phenotypes may have limited the statistical power for detecting differences between these groups. Future prospective, multicenter studies incorporating standardized quantitative two- and three-dimensional echocardiography, longitudinal ventricular measurements, and more detailed assessment of regurgitant physiology would be valuable for validating these phenotype-specific relationships in patients with MR.

Despite these limitations, the study provides a useful framework for understanding mitral valve disease as an interaction between anatomy and cardiovascular physiology. The findings support a conceptual pathway in which **anatomical phenotype influences leaflet and annular mechanics, altered mechanics influence transmitral or regurgitant flow, and chronic hemodynamic loading contributes to atrial and ventricular remodeling**. This integrated perspective may improve interpretation of echocardiographic findings and provide a more comprehensive understanding of the mechanisms underlying different forms of mitral regurgitation.

5. CONCLUSION

This study highlights the close relationship between mitral valve anatomy and its hemodynamic behavior in patients with mitral regurgitation. Rheumatic leaflet restriction represented the predominant anatomical phenotype, while myxomatous mitral valve prolapse and flail leaflet constituted important alternative patterns. Differences in mitral valve area, transmitral velocity, mean gradient, effective regurgitant orifice area, and regurgitant volume demonstrate that distinct structural abnormalities produce characteristic alterations in transvalvular flow and systolic valve competence. The observed relationships between regurgitant parameters and left atrial and ventricular dimensions further emphasize the role of chronic volume loading in cardiac remodeling. These findings support an integrated echocardiographic approach in which mitral valve morphology is interpreted together with transmitral hemodynamics, severity of regurgitation, chamber dimensions, and ventricular function. Characterization of anatomical and hemodynamic phenotypes may improve understanding of the mechanisms underlying mitral valve dysfunction and provide a useful foundation for future prospective studies in regional populations.

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