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Evaluation of Left Atrial Functions in Patients with Primary Mitral Regurgitations after Mitral Valve Replacement in Comparison to Mitral Valve Repair: Strain Imaging Study

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ABSTRACT

Background: Mitral regurgitation (MR) is the most often valvular heart illness and is closely related with left atrial (LA) dilation, which reflects both the chronicity and severity of MR. The LA plays a vital role as a reservoir during ventricular filling, and its enlargement is a predictor of adverse results, even in asymptomatic cases. Surgical correction, particularly mitral valve repair (MVr), is recommended for severe MR. Recent advances like two-dimensional speckle tracking echocardiography (2D-STE) enable detailed assessment of LA function, providing valuable guidance for management and prognostic evaluation. **Aim:** To assess alterations in left atrial strain in cases with chronic severe primary MR before and following MV surgery, and to compare the effects of MV repair and MV replacement (MVR) on LA mechanics. **Patients and methods:** This research involved 30 cases with chronic primary severe MR and preserved left ventricle (LV) ejection fraction. Cases have been randomized into two equal groups: MVr (n=15) and MVR (n=15). All cases had comprehensive clinical and echocardiographic evaluation, including 2D, Doppler, and 2D-STE imaging, performed preoperatively and six months postoperatively to assess changes in LA strain and cardiac dimensions. **Results:** Both groups demonstrated significant postoperative improvement in LA strain and reduction in LA size. The MVr group showed a slightly greater improvement in LA mechanics compared with MVR. **Conclusion:** In cases with chronic severe 1ry MR, mitral valve surgery—either repair or replacement—outcomes in significant left atrial reverse functional and remodeling recovery, with MVr offering superior outcomes, as shown by 2D speckle tracking.

Keywords: Echocardiography, Mitral valve repair, Mitral regurgitation (MR), Speckle tracking echocardiography (STE), Left atrial strain.

INTRODUCTION

The occurrence of valvular heart illnesses increases with age, and population studies have shown mitral regurgitation is the most frequent valvular disorder[1].

MR causes volume overload of both the LA and LV. The left atrium is the 1st chamber to receive the excess volume and hence, left atrium dilation, as a marker of left atrium remodeling, reflects both the duration and the severity of mitral regurgitation and is related with elevated cardiovascular mortality and morbidity irrespective of left ventricular function[2].

The primary mechanical function of the LA is to regulate LV filling and cardiovascular performance through its key roles: serving as a "reservoir" for pulmonary venous return throughout ventricular systole, acting as a "conduit" for pulmonary venous return throughout early ventricular diastole, and functioning as a "booster pump" to enhance ventricular filling throughout late ventricular diastole[3].

Current guidelines advocate for mitral valve surgery in cases exhibiting severe mitral regurgitation and manifest symptoms, left ventricular dysfunction (LVEF ≤ sixty percent, LVESD ≥ forty millimeters), atrial fibrillation, or pulmonary hypertension (resting systolic pulmonary arterial pressure above fifty millimeters of mercury). Left atrial dilatation (LA volume ≥ sixty milliliters per square meter or diameter ≥ fifty-five millimeters) serves as a negative prognostic indicator and is classified as a class IIa recommendation for surgical intervention, even in asymptomatic cases with severe mitral regurgitation[4].

When surgery is indicated, mitral valve repair is the preferred surgical strategy when durability is anticipated, as assessed by the Heart Team, due to its superior survival outcomes compared to MV replacement. When restoration is impractical, MV replacement with preservation of the subvalvular apparatus is preferred[4].

Transcatheter mitral valve implantation for severe 1ry mitral regurgitation may serve as a safe alternative for cases with surgical contraindications or elevated operative risk. Recently, two-dimensional speckle tracking echocardiography has been recognized as a promising, noninvasive, straightforward, and accessible method for evaluating left atrial function, facilitating the identification of early LA dysfunction prior to morphological alterations[5].

In cases with persistent severe mitral regurgitation who underwent successful mitral valve surgery, baseline left atrial global longitudinal strain (LAGLS) serves as an independent predictor of results following the operation. Cases exhibiting elevated baseline LAGLS demonstrated superior long-term results relative to those with diminished LAGLS[6].

The purpose of this research was to assess alterations in left atrial strain in cases with chronic severe primary mitral regurgitation following and prior to mitral

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valve surgery and to evaluate if there are differences between mitral valve repair and mitral valve replacement regarding the impact on left atrial strain.

PATIENTS AND METHODS

This comparative analytical observational research has been conducted on 30 cases with chronic primary severe mitral regurgitation, defined regarding the European Association of Cardiovascular Imaging (EACVI) guidelines[7], in sinus rhythm with LV ejection fraction (EF) ≥ 40%. All patients met the criteria for surgical intervention as suggested by European Society of Cardiology guidelines[4]. Of the 30 patients, 15 underwent mitral valve replacement, and 15 had MV repair.

Exclusion criteria included patients with EF less than 40%, other valvular diseases greater than mild degree, congenital heart diseases, cardiomyopathies and pericardial diseases, permanent pacemakers, atrial fibrillation (AF), poor echocardiographic windows, end-stage renal illness, and end-stage hepatic illness.

All cases had a comprehensive evaluation that involved detailed history taking, a clinical investigation, and a 12-lead electrocardiography.

Echocardiography: Two-dimensional and Doppler echocardiography have been performed one week before and six months following surgery. Echocardiographic studies have been conducted utilizing commercially available ultrasound systems, Philips Affiniti 30 (Philips Healthcare, Andover, United States of America), equipped with a 3.5 megahertz transducer. Three cardiac cycles have been documented timed at end-expiration to minimize respiratory variability. All recordings have been stored for subsequent offline analysis. All information has been transferred to a workstation for offline analysis (TOMTEC-ARENA Imaging System, GmbH).

Measurements involved:

- LV end-diastolic diameter, end-systolic diameter, and LV volumes (end-systolic and end-diastolic) along with EF, which have been measured using M-mode echocardiography regarding the EACVI [7].
- LA anteroposterior diameter, assessed with 2D-guided M-mode echocardiography from the parasternal long-axis perspective at end-systole.
- Left atrial volume, ascertained via the biplane area-length technique from the apical four- and two-chamber perspectives at end-systole. The left atrial volume was normalized to body surface area as advised.

Mitral regurgitation has been assessed using color Doppler, and the severity was quantified by the vena contracta width and the Effective Regurgitant Orifice Area (EROA) following EACVI recommendations [7]. Color Doppler information has been acquired at fifteen to seventeen frames per second with a depth of 16 centimeters, and the Nyquist limit was set to fifty to sixty centimeters per second. The color gain has been modified to remove random colors in regions devoid of flow. Mitral inflow velocities have been assessed at the tips of the mitral leaflets with pulsed Doppler at end-expiration, ensuring the Doppler beam has been oriented to reduce the angle among the blood flow vector and the beam.

Systolic pulmonary artery pressure has been determined by summing the estimated right atrial pressure with the systolic right ventricular-right atrial gradient obtained from the peak velocity of systolic trans-tricuspid regurgitant flow.

Speckle Tracking Echocardiography: Most studies utilizing LA strain focus on global longitudinal strain (GLS), which is described as strain in the direction tangential to the endocardial atrial border in an apical view[8]. A subdivision of the LA wall into segments isn't suggested due to the thin nature of the LA myocardium and insufficient resolution for reliable local tracking in echocardiographic images. Additionally, variations in interpolation across pulmonary vein orifices and the LA appendage make segmental definitions challenging [8].

The EACVI/ASE/Industry Task Force [8] recommends interpreting left atrial strain as global strain derived from the length alteration of the entire left atrial contour in the image plane. While LA muscle bundles were recognized in the posterior wall of the LA, analysis in the apical long-axis view can be confounded by difficulty in separating the ascending aorta from the LA wall.

For 2D STE analysis, the onset of the QRS complex (ventricular end-diastole) has been utilized as the zero reference point. The left atrial was at its minimum volume following contraction, and a line was manually drawn along the LA endocardium across the pulmonary veins and/or LA appendage orifices. Cine-loop preview features were applied to confirm that the internal line followed the left atrial endocardium throughout the cardiac cycle. In cases of unsatisfactory tracking, manual adjustments were made. LA strain curve **(Figure 1)** starts with the reservoir phase where LA filling and stretching produce positive strain, peaking in systole just before MV opening. This is defined as LA reservoir strain (LASr) and is determined at the end of the reservoir phase as the average peak systolic strain from twelve atrial segments. Following MV opening, passive LA emptying generates a negative strain deflection that plateaus during diastasis, referred to as LA conduit strain (LAScd). A 2nd negative deflection during atrial systole corresponds to LA contractile strain (LASct).

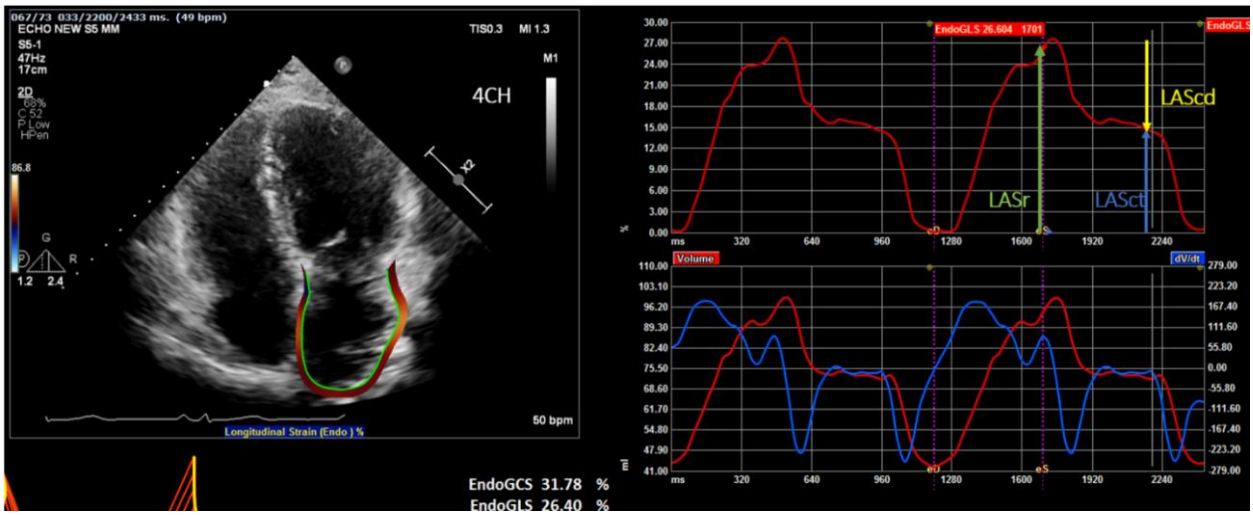

Fig. 1: LA strain curve is composed of a positive peak at the end-systole (reservoir), followed by two descending phases in early diastole (passive emptying) and in late diastole (active emptying).

Ethical Approval:

Informed written agreement has been attained from all participants, and the protocol of the research received approval from the institutional ethics committee. The research has been registered with the local ethics committee of Menoufia University, Faculty of Medicine (IRB approval number: 12/2022 CARDIO 35). The research adhered to the Helsinki Declaration throughout its execution.

Statistical Analysis

Information has been examined utilizing the IBM SPSS software package version 20.0 (Armonk, NY: IBM Corp). Actually, the "Shapiro-Wilk" test was used to verify the normality of distribution and it was proven that all the variables done by "t-test" is normally distributed.. Qualitative information has been presented as percentages and numbers, while quantitative information has been described utilizing range (maximum and minimum), mean, standard deviation (SD), median, and interquartile range (IQR). Statistical significance has been set at the five percent level, and various tests were used based on data types: Chi-square test for categorical parameters to compare among groups; Fisher's exact test for corrections when more than twenty percent of cells had expected counts < 5; Mann-Whitney test for abnormally distributed parameters to compare two groups; independent Student's t-test for normally distributed quantitative parameters to compare two groups; paired t-test for normally distributed information to compare 2 periods; and the Wilcoxon signed ranks test for abnormally distributed parameters to compare 2 periods.

RESULTS

Baseline features were generally comparable between the repair (n = 15) and replacement (n = 15) groups. Cases in the replacement group were older (41.7 ± 16.1 vs. 32.6 ± 10.1 years; *p*-value equal 0.076) and had a greater occurrence of diabetes mellitus (33.3% vs. 0%; *p-value equal* 0.040). Insignificant variances were observed between groups with respect to sex distribution, body surface area, body mass index, or hypertension status **(Table 1)**.

Table (1): Baseline features of the study population

	Repair (Number = 15)		Replacement (Number = 15)		Test of Significance	p
	No.	**%**	**No.**	**%**		
Gender						
Male	5	33.3	7	46.7	χ^2= 0.556	0.456
Female	10	66.7	8	53.3		
Age (Year)						
Min – Max.	19.0 – 51.0		20.0 – 75.0		t= 1.844	0.076
Mean ± SD.	32.60 ± 10.15		41.67 ± 16.11			
Median (IQR)	32.0 (25.50 – 39.0)		36.0 (29.0 – 53.50)			
Weight (Kg)						
Min – Max	40.0 – 115.0		42.0 – 100.0		t= 0.396	0.695
Mean ± SD	68.33 ± 17.89		70.87 ± 17.14			
Median (IQR)	64.0 (59.0 – 72.50)		66.0 (60.50-83.0)			
Height (cm)						
Min – Max	149.0 – 179.0		142.0 – 190.0		t= 0.625	0.537
Mean ± SD	161.4 ± 9.01		163.7 ± 10.77			
Median (IQR)	160.0 (154.0 - 167.0)		163.0 (157.5 – 170.0)			
BMI (kg/m^2)						
Min – Max	16.0 – 38.40		17.50 – 42.20		t= 0.156	0.877
Mean ± SD	27.01 ± 6.69		26.62 ± 7.08			
Median (IQR)	25.0 (22.60 - 31.95)		23.70 (22.40 – 29.25)			
BSA (m^2)						
Min – Max	1.32 – 2.35		1.34 – 2.23		t= 0.237	0.814
Mean ± SD	1.76 ± 0.25		1.78 ± 0.24			
Median (IQR)	1.66 (1.63 – 1.93)		1.76 (1.65 – 1.90)			
HTN						
No	14	93.3	11	73.3	χ^2= 2.160	0.330
Yes	1	6.7	4	26.7		
DM						
No	15	100.0	10	66.7	χ^2= 6.000*	0.042*
Yes	0	0.0	5	33.3		

HTN: Hypertension, BSA: Body Surface Area, BMI: Body Mass Index, DM: Diabetes Mellitus
SD: Standard deviation, IQR: Interquartile range, χ^2: Chi square test, t: Independent t-test, *: Statistically significant at p - value not more than 0.05.

Both surgical groups demonstrated significant postoperative alterations in LV geometry and function. In the total cohort, end-diastolic diameter reduced from 5.57 ± 0.72 cm to 4.89 ± 0.68 centimeter (*p* below 0.001) and end-diastolic volume from 155.5 ± 44.9 ml to 116.7 ± 32.6 ml (*p* below 0.001). EF declined from 61.7 ± 9.7% to 54.7 ± 8.7% (*p*-value under 0.001). Between-group comparisons showed that EF was numerically higher in the repair group both preoperatively and postoperatively, although these differences were not statistically significant. End-systolic diameter reduction reached statistical significance only in the replacement group ($p = 0.046$). Other insignificant between-group differences have been observed. Also, "ESV" was proven to be not normally distributed by the same test, so, as result, we used "Mann-whitney" and "Wilcox on" as showed in table 2.

Table (2): Echocardiographic features of the study population

		Total (Number = 30)	Repair (Number = 15)	Replacement (Number = 15)	Test of Significance	p
Ejection Fraction %	**Pre-Operative**					
	Min – Max.	39.46 – 78.46	39.46 – 76.27	41.63 – 78.46	t= 1.412	0.169
	Mean ± SD.	61.74 ± 9.74	64.21 ± 9.13	59.27 ± 10.01		
	Median (IQR)	64.10 (54.71 – 68.56)	66.60(61.89 – 69.02)	55.56(54.28 – 66.88)		
	Post-Operative					
	Min – Max.	38.10 – 70.0	44.68 – 67.28	38.10 – 70.0	t= 0.464	0.646
	Mean ± SD.	54.66 ± 8.72	55.41 ± 6.92	53.91 ± 10.42		
	Median (IQR)	55.37 (49.74 – 60.59)	54.77(50.95 – 59.88)	55.97(45.62 – 60.71)		
	t_0 (p_0)	4.790*(<0.001*)	4.038* (0.001*)	2.722* (0.017*)		
ESD (cm)	**Pre-Operative**					
	Min – Max.	2.54 – 5.30	2.54 – 5.30	3.0 – 5.0	t= 0.427	0.672
	Mean ± SD.	3.69 ± 0.69	3.64 ± 0.78	3.75 ± 0.60		
	Median (IQR)	3.70 (3.20 – 4.20)	3.55 (3.20 – 4.16)	3.80 (3.25 – 4.10)		
	Post-Operative					
	Min – Max.	1.39 – 4.90	1.39 – 4.73	2.60 – 4.90	t= 0.398	0.694
	Mean ± SD.	3.43 ± 0.73	3.38 ± 0.81	3.49 ± 0.67		
	Median (IQR)	3.30 (3.0 – 4.0)	3.30 (2.90 – 3.95)	3.30 (3.0 – 3.95)		
	t_0 (p_0)	2.886*(0.007*)	1.858 (0.084)	2.194* (0.046*)		
ESV (ml)	**Pre-Operative**					
	Min – Max.	23.20 – 135.0	23.20 – 135.0	33.60 – 122.0	U= 99.500	0.595
	Mean ± SD.	60.65 ± 27.08	58.76 ± 30.01	62.54 ± 24.70		
	Median (IQR)	57.05 (41.90 – 70.20)	47.10(40.05 – 77.30)	60.30 (44.0 – 69.90)		
	Post-Operative					
	Min – Max.	29.60 – 113.0	29.60 – 104.0	30.0 – 113.0	U= 109.000	0.902
	Mean ± SD.	54.50 ± 23.66	53.99 ± 23.07	55.0 ± 25.04		
	Median (IQR)	48.15 (35.0 – 70.0)	48.10(36.35 – 71.05)	48.20(35.85 – 68.35)		
	Z (p_0)	1.882 (0.060)	0.909 (0.363)	1.704 (0.088)		

EDD (cm)	**Pre-Operative**					
	Min – Max.	4.0 – 7.26	4.0 – 7.26	4.60 – 6.50	t= 0.248	0.806
	Mean ± SD.	5.57 ± 0.72	5.61 ± 0.88	5.54 ± 0.54		
	Median (IQR)	5.60 (5.14 – 5.90)	5.70 (5.27 – 5.88)	5.50 (5.20 – 5.80)		
	Post-Operative					
	Min – Max.	3.20 – 6.16	3.20 – 6.16	4.10 – 6.10	t= 0.109	0.914
	Mean ± SD.	4.89 ± 0.68	4.91 ± 0.79	4.88 ± 0.57		
	Median (IQR)	4.93 (4.37 – 5.40)	5.0 (4.29 – 5.50)	4.90 (4.45 – 5.30)		
	t_0 (p_0)	6.876* (<0.001*)	5.021* (<0.001*)	4.551* (<0.001*)		
EDV (ml)	**Pre-Operative**					
	Min – Max.	73.80 – 277.0	73.80 – 277.0	99.80 – 218.0	t= 0.511	0.613
	Mean ± SD.	155.5 ± 44.86	159.7 ± 54.96	151.3 ± 33.29		
	Median (IQR)	152.0 (127.0 – 172.0)	161.0(133.5 – 173.5)	152.0(131.0 – 156.0)		
	Post-Operative					
	Min – Max.	71.0 – 188.0	71.0 – 188.0	81.20 – 188.0	t= 0.214	0.832
	Mean ± SD.	116.7 ± 32.58	118.0 ± 35.55	115.4 ± 30.51		
	Median (IQR)	108.8 (89.70 – 147.0)	109.5(92.26 – 151.0)	108. (93.95 – 128.7)		
	t_0 (p_0)	6.301* (<0.001*)	4.700* (<0.001*)	4.087* (0.001*)		

ESD: End-Systolic Diameter, ESV: End-Systolic Volume, EDD: End-diastolic diameter, EDV: End-diastolic Volume, t: Independent t-test, (t_0): Paired t-test, (U): Mann Whitney test, (Z): Wilcoxon signed ranks test, p_0: p value for comparing pre-operative and post-operative, *: Statistically significant at p -value not more than 0.05.

Baseline mitral regurgitation severity was comparable between repair and replacement groups. Vena contracta width (VCW) did not differ significantly (7.34 ± 0.72 mm vs. 7.03 ± 1.44 mm), with similar interquartile ranges (7.20 [7.0–7.70] mm vs. 7.10 [6.0–8.0] mm). Effective regurgitant orifice area values were also nearly identical between groups (0.53 ± 0.17 cm² versus 0.54 ± 0.08 cm²), with overlapping interquartile ranges (0.50 [0.44–0.58] cm² vs. 0.52 [0.49–0.61] cm²) **(Table 3)**.

Table (3): Comparative analysis between the two study groups regarding vena contracta and EROA

	Repair (Number = 15)	Replacement (Number = 15)	Test of Significance	p
Vena contracta width (mm)				
Min – Max.	6.20 – 9.0	4.0 – 9.30	t= 0.749	0.462
Mean ± SD.	7.34 ± 0.72	7.03 ± 1.44		
Median (IQR)	7.20 (7.0 – 7.70)	7.10 (6.0 – 8.0)		
EROA (cm^2)				
Min – Max.	0.40 – 1.09	0.42 – 0.70	t= 0.136	0.893
Mean ± SD.	0.53 ± 0.17	0.54 ± 0.08		
Median (IQR)	0.50 (0.44 – 0.58)	0.52 (0.49 – 0.61)		

EROA: Effective regurgitant orifice area, t: Independent t-test

LA parameters demonstrated significant reverse remodelling in both surgical groups, with notable differences between repair and replacement. Preoperatively, LA diameter was larger in the replacement group (5.30 ± 0.67 cm versus 4.63 ± 0.67 cm; $p = 0.011$), and this difference persisted postoperatively (4.25 ± 0.68 cm versus 3.62 ± 0.64 cm; $p = 0.014$), despite significant within-group reductions ($p < 0.001$ for both). Preoperative LAVi was comparable between groups (119.0 ± 33.79 vs. 113.6 ± 39.42 ml/m²; $p = 0.686$). Postoperatively, LAVi decreased in both groups, with greater reduction observed in the repair group (69.30 ± 18.42 vs. 98.05 ± 27.37 ml/m²; $p = 0.002$) **(Table 4)**.

Table (4): Comparative analysis between the two groups regarding left atrium diameter, LAVi

		Total (Number = 30)	Repair (Number = 15)	Replacement (Number = 15)	Test of Significance	P
Diameter (cm)	**Pre-Operative**					
	Min – Max.	3.60 – 6.40	3.60 – 6.0	4.10 – 6.40	t= 2.738*	0.011*
	Mean ± SD.	4.97 ± 0.74	4.63 ± 0.67	5.30 ± 0.67		
	Median (IQR)	4.90 (4.45 – 5.50)	4.50 (4.30 – 4.90)	5.30 (4.90 – 5.75)		
	Post-Operative					
	Min – Max.	2.60 – 5.80	2.60 – 4.90	3.30 – 5.80	t= 2.607*	0.014*
	Mean ± SD.	3.93 ± 0.72	3.62 ± 0.64	4.25 ± 0.68		
	Median (IQR)	3.90 (3.40 – 4.30)	3.50 (3.20 – 3.95)	4.10 (3.80 – 4.70)		
	t_0 (p_0)	9.534* (<0.001*)	7.559* (<0.001*)	6.016* (<0.001*)		
LAVi (ml/m2)	**Pre-Operative**					
	Min – Max.	52.40 – 212.3	52.40 – 212.3	62.60 – 168.4	t= 0.408	0.686
	Mean ± SD.	116.3 ± 36.18	113.6 ± 39.42	119.0 ± 33.79		
	Median (IQR)	123.6 (84.06 – 132.05)	107.0(87.28 – 131.3)	125.1(90.60 – 142.6)		
	Post-Operative					
	Min – Max.	37.05 – 130.0	37.05 – 98.10	48.0 – 130.0	t= 3.375*	0.002*
	Mean ± SD.	83.68 ± 27.19	69.30 ± 18.42	98.05 ± 27.37		
	Median (IQR)	74.06 (64.70 – 111.70)	68.20(57.87 – 81.39)	111.7(72.64 – 119.0)		
	t_0 (p_0)	3.9485* (<0.001*)	4.824* (<0.001*)	3.487* (0.004*)		

LAVi: left atrium volume index, t: Independent t-test, (t_0): Paired t-test, *: Statistically significant at p -value not more than 0.05.

Left atrial global longitudinal strain (LAGLS) improved significantly following surgery in both groups ($p < 0.001$). Preoperative values were comparable between repair and replacement patients (20.15 ± 3.72% vs. 21.21 ± 4.88%). Postoperatively, LAGLS was significantly higher in the repair group (34.26 ± 3.73% vs. 30.50 ± 5.27%; $p = 0.049$). The magnitude of improvement was also greater following repair (Δ14.11% vs. Δ9.29%) **(Figure 2, Table 5)**.

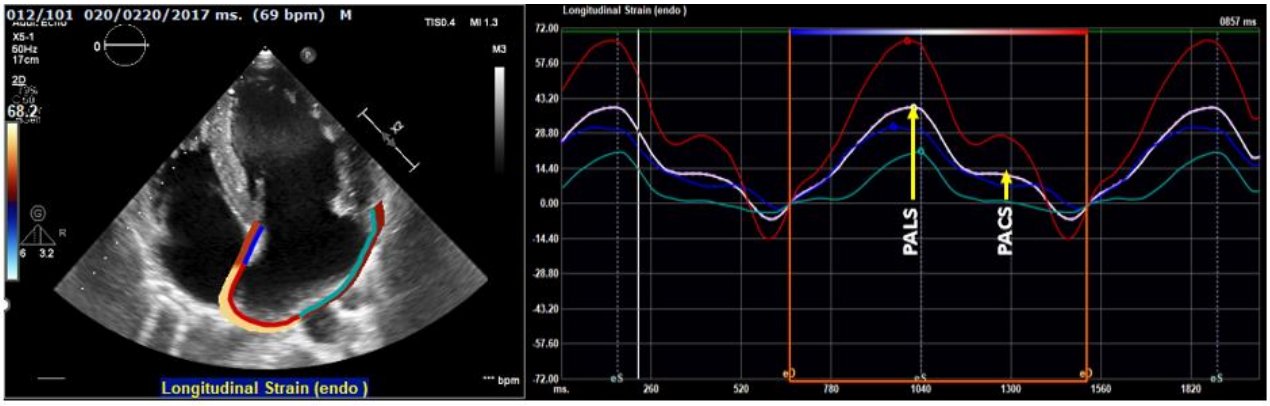

Fig. 2: Parameters of left atrial longitudinal strain. A composite graphic illustrating the measurement of peak atrial longitudinal strain and peak atrial contraction strain (PACS) via STE from an apical four-chamber perspective. The white curve illustrates the mean atrial longitudinal strain during the cardiac cycle.

Estimated pulmonary artery systolic pressure (ePASP) was comparable between groups at baseline. At six months postoperatively, ePASP decreased significantly in both groups (repair: 32.0 ± 8.5 to 18.0 ± 5.0 mmHg, $p < 0.001$; replacement: 35.0 ± 9.2 to 25.0 ± 7.0 millimeters of mercury, $p < 0.001$). Similar improvement has been observed in the overall cohort (33.5 ± 8.9 to 21.5 ± 6.5 mmHg, $p < 0.001$). Postoperative intergroup comparison showed lower ePASP in the repair group compared with the replacement group ($p = 0.004$) **(Table 5)**.

Table (5): Comparative analysis between the two groups studied regarding LAGLS and ePASP

		Total (Number = 30)	Repair (Number = 15)	Replacement (Number = 15)	t	p
LAGLS (%)	**Pre-Operative**					
	Min – Max.	11.95– 28.00	13.67 – 25.81	11.95 – 28.00	0.669	0.509
	Mean ± SD.	20.68 ± 4.3	20.15 ± 3.72	21.21 ± 4.88		
	Median (IQR)	20.82 (23.29 -18.13)	19.45 (23.29- 18.13)	21.33(26.32- 18.17)		
	Post-Operative					
	Min – Max.	22.56 – 40.23	28.45– 40.12	22.56 – 40.23	2.26*	0.032*
	Mean ± SD.	32.38± 4.87	34.26± 3.73	30.50 ± 5.27		
	Median (IQR)	32.00(29.53– 35.53)	34.56(31.56– 37.11)	30.23(27.12– 33.89)		
	t_0 (p_0)	9.65* (< 0.0001*)	15.64* (<0.0001*)	6.75* (<0.001*)		
e-PASP (mmHg)	**Pre-Operative**					
	Min – Max.	20.0 – 55.0	20.0 – 50.0	22.0 – 55.0	1.2	0.24
	Mean ± SD.	33.5 ± 8.9	32.0 ± 8.5	35.0 ± 9.2		
	Median (IQR)	32.0 (26.0–40.0)	30.0 (25.0–38.0)	34.0 (28.0–42.0)		
	Post-Operative					
	Min – Max.	10.0 – 40.0	10.0 – 30.0	15.0 – 40.0	3.100*	0.004*
	Mean ± SD.	21.5 ± 6.5	18.0 ± 5.0	25.0 ± 7.0		
	Median (IQR)	20.0 (16.0–26.0)	17.0 (14.0–22.0)	24.0 (20.0–30.0)		
	t_0 (p_0)	6.200* (<0.001*)	4.800* (<0.001*)	5.500* (<0.001*)		

LAGLS: Left atrium global longitudinal strain, e-PASP: estimated pulmonary artery systolic pressure, **t:** t: Independent t-test, (t_0): Paired t-test, *: Statistically significant at p -value not more than 0.05.

DISCUSSION

MV surgery is the therapeutic intervention for patients with symptomatic severe mitral regurgitation. A significant LA dilatation (≥ sixty milliliters per square meter) in the presence of sinus rhythm has been identified as a prognostic indicator of adverse clinical results in cases with asymptomatic severe mitral regurgitation and is considered one of the surgical indications [4].

While the predictive significance of preoperative left atrial size in cases having mitral valve repair is well-established, the data regarding left atrial reverse remodeling following mitral valve surgery remain completely unexamined. In this research, we present a comprehensive analysis of 30 cases with chronic primary severe mitral regurgitation who met the criteria for surgical intervention regarding the European Society of Cardiology [4], to examine the impact of mitral valve surgery on the left atrium global longitudinal strain (LA-GLS)—as a surrogate for left atrial function as a reservoir—assessed by 2D strain analysis, and to investigate whether there is any difference between mitral valve repair and replacement regarding this variant.

Our main findings include the following: (1)- Patients underwent MVr demonstrated superior preservation of LA strain parameters when compared to those underwent MVR. (2)- there is a statistically significant reduction in both left atrial volume index (LAVi) and LA diameter after surgical intervention in both groups with slight preference in favour of MVr. (3)- There is statistically significant reduction in LVEF, LVESD, LVEDD and LVEDV in the total population after surgery. There was statistically insignificant variance between both groups in all these variables. (4)- There is statistically significant reduction in e-PASP in the total population and significantly lower ePASP in the repair group than the replacement group. Regarding the left atrial global longitudinal strain, we found that the repair group demonstrated a substantial postoperative improvement, with mean LA-GLS increasing from 20.15 ± 3.72 to 34.26 ± 3.73 (70%). In the replacement group, LA-GLS also increased, albeit to a lesser extent, from 21.21 ± 4.88 to 30.50 ± 5.27 (43.8%).

Recent evidence suggests that left atrial (LA) function, rather than LA volume, provides a more robust marker of atrial remodelling and clinical outcomes[9]. Functional indices, particularly peak atrial longitudinal strain (PALS), enable detection of subclinical atrial dysfunction prior to overt chamber enlargement and comprehensively reflect reservoir, conduit, and contractile function across the cardiac cycle. Impaired LA strain was consistently related to adverse cardiovascular results, underscoring its prognostic superiority over LA volume, which may be influenced by non-pathological factors and does not always mirror current atrial dysfunction.

These observations are supported by clinical studies. **Kim *et al*.**[10] demonstrated in 169 patients undergoing MVr for primary MR that higher postoperative LA strain predicted improved outcomes. Similarly, **Oh *et al*.**[11] evaluated 338 patients with severe MR and found that preoperative LA-GLS was an independent predictor of long-term outcomes; although strain declined immediately post-surgery, recovery was observed at one year. These results agree with accumulating data highlighting the prognostic role of LA function in MR and other cardiovascular diseases[12],[13].

Importantly, LA strain offers an objective measure of left atrial performance[14]. **Cameli *et al*.**[15], reported that reduced LA deformation, assessed by global PALS, correlated with fibrosis and remodelling in patients referred for surgery. In a complementary study, **Stassen *et al*.**[16] followed 226 patients after MVr and observed that LA strain decreased acutely postoperatively, but improved over time, reflecting dynamic changes in atrial loading and adaptation.

Early postoperative reductions in LA-GLS likely reflect transient myocardial stunning and altered hemodynamics following MR correction. Recovery appears more favorable after MV repair compared with replacement, as repair preserves the native MV apparatus and annular dynamics, thereby supporting more physiological LA remodelling. By contrast, valve replacement may limit atrioventricular plane motion and attenuate postoperative atrial functional recovery.

In our study, the LAVi decreased significantly post-operation, with the repair group showing a 39% reduction (from 113.6 ± 39.42 to 69.30 ± 18.42) and the replacement group an 18% reduction (from 119.0 ± 33.79 to 98.05 ± 27.37). Similarly, left atrial diameter decreased by 22% in the repair group (from 4.63 ± 0.67 to 3.62 ± 0.64) and 20% in the replacement group (from 5.30 ± 0.67 to 4.25 ± 0.68). Several studies have consistently demonstrated significant reductions in LA size following surgical correction of MR. **Antonini-Canterin *et al*.**[17] observed in 79 patients with severe degenerative MR a marked decrease in LA diameter, LA area, and indexed LA volume (LAVi) within 1–6 months postoperatively. Similarly, **Marsan *et al*.**[18] using real-time 3D echocardiography in 65 patients undergoing MV surgery, showed progressive reductions in LA volumes at 6 months and further at 1-year follow-up.

In a larger cohort of 720 cases, **Balachandran *et al*.**[19] confirmed substantial postoperative LAVi reduction after MVr (preoperative 55 [45–66] mL/m² vs postoperative 42 [36–50] mL/m²). This decline reflects both a passive process (removal of the regurgitant volume immediately after surgery) and an active process (reduced wall stress facilitating reverse remodelling). However, LA enlargement does not necessarily imply dysfunction, as preserved atrial mechanics may coexist with increased

LA volume in the absence of diastolic dysfunction[20]. These data underscore the need to integrate structural indices with functional assessment for a comprehensive evaluation of atrial performance.

Furthermore, **Pande *et al*.**[21] analyzed 116 patients undergoing isolated MVR and stratified them by preoperative LA size (<60 mm vs >60 mm). Both groups demonstrated significant postoperative LA size reduction, highlighting the potential for reverse remodelling even after valve replacement.

And when it comes to the left ventricular ejection fraction, volumes and dimensions; our findings demonstrated a significant postoperative reduction in LVEF, from 61.74 ± 9.74% to 54.66 ± 8.72%. Additionally, significant reductions have been detected in LV end-systolic diameter (from 3.69 ± 0.69 cm to 3.43 ± 0.73 cm), end-diastolic diameter (from 5.57 ± 0.72 cm to 4.89 ± 0.68 centimetres), and end-diastolic volume (from 155.5 ± 44.86 mL to 116.7 ± 32.58 mL) in the total population following surgery. However, the change in end-systolic volume didn't reach statistical significance (P-value equal 0.06). A statistically insignificant variances have been observed between the repair and replacement groups in any of these parameters.

The rapid fall in LVEDV reflects immediate preload reduction after MR correction, whereas the slower decline in LVESV reflects impaired contractility from chronic volume overload and its dependence on myocardial recovery and afterload adaptation. Thus, while LVEDV responds rapidly to hemodynamic changes, LVESV normalizes more gradually, paralleling the progressive restoration of myocardial function and performance following surgical intervention.

These findings are consistent with previous reports. **Craven *et al*.**[22] using CMR in 72 patients undergoing MVr or chordal-preserving MVR, demonstrated significant reductions in indexed LVEDV, LVEF, and LAVi at six months, irrespective of surgical strategy. Similarly, in a large cohort of 2,778 patients undergoing MVr, **Shafii *et al*.**[23] reported early reductions in LVEDD within six months that persisted at five years, with more gradual LVESD decline stabilizing by year five. LVEF exhibited an initial non-significant fall, followed by modest improvement over the first postoperative year that was sustained long term. **Le Tourneau *et al*.**[24] further highlighted the expected postoperative decline in LVEF as a consequence of abrupt elimination of regurgitant volume and reduction in LVEDV—an "afterload mismatch" particularly evident in patients with preoperative LV dysfunction. Over time, however, progressive LV reverse remodelling with reduction in LVESV leads to recovery of stroke volume and eventual improvement in ejection fraction during follow-up.

In our cohort, estimated pulmonary artery systolic pressure (ePASP) declined significantly after surgery (33.5 ± 8.9 to 21.5 ± 6.5 mmHg, $p < 0.001$), with a greater reduction observed following MVr (32.0 ± 8.5 to 18.0 ± 5.0 mmHg) compared with MVR (35.0 ± 9.2 to 25.0 ± 7.0 mmHg; both $p < 0.001$). Intergroup comparison confirmed significantly lower postoperative ePASP in the repair group (p equal to 0.004). These results are in line with evidence that MVr more effectively preserves LV geometry, improves pulmonary vascular hemodynamics, and facilitates superior early postoperative recovery compared with valve replacement[25].

Previous studies consistently report substantial postoperative reductions in pulmonary pressures. One study by **Ali K. *et al*.**[26] noted a 22% decline in systolic pulmonary artery pressure (SPAP) after MVR, from 70.3 ± 6.8 to 39.4 ± 5.8 mmHg within three months. Similarly**, Walls *et al*.**[27] demonstrated that although pulmonary pressures decrease following all surgical modalities (repair, bioprosthetic, mechanical), MVr is related with a lower prevalence of persistent postoperative pulmonary hypertension, likely reflecting its more physiological restoration of mitral and ventricular function.

Taken together, these results reinforce the concept that MVr confers greater benefit in reverse remodelling compared with MVR. MVr was associated with improved LA strain (GLS), more pronounced reductions in LAVi and LA diameter, and superior unloading of pulmonary pressures, whereas LV reverse remodelling occurred to a similar extent with both surgical approaches. The expected transient postoperative decline in LVEF was also confirmed. These findings are consistent with accumulating data highlighting LA strain as a more sensitive marker of atrial function than volume alone, and underline the prognostic significance of LA functional recovery. Furthermore, the more marked reduction in ePASP following MVr underscores the physiological advantages of valve preservation in maintaining annular–ventricular coupling and mitigating postoperative pulmonary hypertension. Collectively, these observations support MVr as the preferred strategy in cases with chronic 1^{ry} MR.

CONCLUSION

In this study of 30 cases with chronic severe primary MR, MV surgery—either repair or replacement—was related with significant left atrial reverse remodelling and functional enhancement as evaluated by 2D speck tracking echocardiography. Postoperative increases in LAGLS and reductions in LAVi and LA diameter were observed in both groups, with changes being statistically significant in the favor of mitral valve repair. These findings suggest that MV repair provides superior restoration of LA mechanics compared with replacement and should be preferred when feasible.

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