Original Article

Echocardiographic Assessment of Left Ventricular Mass Regression and Functional Changes after Bariatric Surgery

Faten Abdul-Lateef Abdul-Zahra[1], Asaad Hasan Noaman[2] and Safauldeen Salim Albaaj[3]

ABSTRACT

Objective: To determine the echocardiographic outcomes of left ventricular mass and diastolic functions in obese patients undergoing bariatric surgery.

Study Design: Prospective study

Place and Duration of Study: This study was conducted at the Al-Batool Hospital (Bariatric Surgery Clinic) between the period of 1st January 2025 and 31st July 2025.

Methods: Enrolment of 51 adult participants was done at the age of 18-65 years. The mass of the left ventricles and the index of diastolic functions were determined preoperative and postoperative through conventional two-dimensional transthoracic echocardiography.

Results: Bariatric surgery showed a substantial decrease on mean left ventricular mass (139.274141.3 g) relative to preoperation levels (175.9648.4 g; $p<0.05$). It was revealed that postoperative assessment showed A-wave velocity and E / e + ratio declined significantly ($p<0.05$), whereas e 2 velocity and E / A ratio increased significantly after surgery ($p<0.05$). The velocity of the E-wave did not differ significantly between the pre and postoperative measurements ($p > 0.05$).

Conclusion: Bariatric surgery enhances diastolic improvement and regression of left ventricular mass in obese patients, indicating substantial cardiac remodelling.

Keywords: Left ventricular mass, Diastolic function, Obesity, Cardiac remodeling, Echocardiography

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INTRODUCTION

Left ventricular hypertrophy (LVH) that is often more likely to develop in obese people is characterized by an increase in the ventricular mass. Obese normotensive subjects exhibited a prevalence rate of left ventricular (LV)-hypertrophy about 14 percent as compared to their slim counterparts (5%). Seventy-eight percent of morbidly obese individuals may develop LV hypertrophy.[1]

Stressors such as obesity, metabolic syndrome, and hypertension noted over a long period of time can produce modifications in the length, shape, and characteristics of the heart, a phenomenon referred to as cardiac remodeling.

[1.] Department of Health, Najaf, Iraq.
[2.] Department of Medical Physiology / General Surgery[3], College of Medicine, University of Kufa, Iraq.

Correspondence: Faten Abdul-lateef Abdul-Zahra,
Department of Health, Najaf, Iraq.
Contact No: 07865147164
Email: fatena.alhchamy@student.uokufa.edu.iq

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Though such changes are also potentially good in the initial stages, they often have maladaptive results such as heart failure. Enhanced LV mass, chamber distension and LV hypertrophy are structural remodeling features in obese individuals.[2]

Left ventricular hypertrophy is classified as eccentric. Eccentric remodeling is not to be confused with concentric remodeling that is usually observed in conditions of pathologically high pressure. Concentric remodeling is featured by an augmentation in the wall-thickness without equivalent augmentation in the ventricles dimensions and it is signalled by augmented relative wall-thickness.[3]

The changes in the hemodynamics of obese people include the increase in left ventricular wall stress and myocardial tension, which are connected to obesity. Increased central blood volume, stroke volume and CO are the major causes of the stress on the LV wall in normotensive obesity.[4] This increases the possibility of LV expansion and eccentric hypertrophy. High-level LV wall stress is likely to be corrected by eccentric LV hypertrophy that leads to diastolic dysfunction in obesity. Obesity is also linked with hemodynamic stress, which can then compete with the dilatation of the chamber but an increasing wall thickening might result in systolic dysfunction later, particularly in combination with neurohormonal and metabolic factors such as sympathetic hyperactivity, activation of renin-

angiotensin-aldosterone system, and adipokine dysregulation.[3]

There are numerous pathophysiological mechanisms that cause left ventricular increase of mass in obesity where the left ventricular mass has a distinct positive correlation with excess body weight severity. Left ventricle hypertrophy is closely associated with cardiac remodeling, diastolic dysfunction, as well as death and cardiovascular events are the other ways to measure cardiac remodeling in obese patients.[5] Diastolic dysfunction and left ventricle hypertrophy is strongly linked with bariatric surgery and achieved significant and sustained weight loss.[6] New evidence suggests that weight loss following bariatric surgery can regress left ventricular hypertrophy, and improve the functional parameters of the heart, notably by improving the diastolic function.[7] There is, however, limited and disparate evidence regarding the degree to which left ventricular mass is being regressed, and how such cardiac functional outcomes are altered after bariatric surgery. Hence, the present study involved echocardiography to determine the regression of left ventricular mass and functional alterations in relation to bariatric surgery.

METHODS

This prospective study will be done in Al-Batool Hospital (Bariatric Surgery Clinic) between 1 st January 2025 and 31 st July 2025 through a letter No. MEC-99 dated 22-12-2024. They were enrolled in the total number of 51 adults aged 18-65 years, whose weight had been planned to undergo bariatric surgery and the eligibility criteria included body mass index of 40kg/m 2 or 30kg/m 2. The transthoracic echocardiography (Mindrayconsona N9) was done pre surgical and following 5 months of the follow up. The echocardiographic measurements of left ventricular mass were made based on the conventional established echocardiographic guidelines provided by American Society of echocardiography. Left ventricular diastolic performance was measured using the indexes of the trans mitral Doppler echocardiographic, early (E) and late (A) diastolic filling velocity, ratio of E/A and E/e, the average between the peak early diastolic velocities at the septal and lateral mitral annulus (é). To determine the structural and diastolic functional change in the heart, Echocardiographic parameters were compared pre- and post-operative. We have gained all our results via SPSS version 26.

RESULTS

The average age was 31.9±7.30f 13 male and 38 female (Table 1). The resulting data of the study which is presented in Table 2 indicated that the mean weight, the body mass index, the systolic blood pressure and the diastolic blood pressure were significantly lower ($p<0.05$) post operation than pre-operation. Table 3 indicates mean of LV mass in preoperative was (175.96±48.4), and the mean of LV mass in postoperative was (139.27±41.3). Thus, the average LV mass decreased considerably postoperative to that pre-operative ($p<0.05$). Also, the postoperative results showed that there was a statistically significant decrease in the A-wave velocity and E/e prime ratio compared to the preoperative values (p less than 0.05) and a significant increase (p less than 0.05) of the mean of e prime wave velocity, and E/A ratio in postoperative as compared to preoperative. E-wave velocity failed to show statistically significant difference between preoperative and postoperative tests (p.05).

Table No. 1: Patients' age and gender distribution

Variable	No.	%
Age (years	31.9±7.3	
Gender		
Male	13	25.5
Female	38	74.5

Table No. 2: Comparison of preoperative and postoperative anthropometric and hemodynamic parameters

Variable	Before surgery	After surgery	P value
Weight	116.2±20.7	86.8±16.9	<0.001
BMI	44.6±6.8	33.4±6.1	<0.001
BSA	2.3±0.2	1.9±0.2	<0.001
SBP	130.39±12.4	119.02±13	<0.001
DBP	78.2±9.5	68.4±12.6	<0.001

Table No. 3: Comparison of Preoperative and Postoperative LV mass and diastolic indices

Variable	Before surgery	After surgery	P value
E wave velocity	85.2±14.6	85.6±16.2	0.8
A wave velocity	62.6±13.4	57.5±10.9	0.01
e` wave velocity	13.06±3.3	15.3±3.3	<0.001
E/A ratio	1.4±0.3	1.5±0.4	0.03
E/e` ratio	6.9±2.4	5.8±1.1	<0.001
LV mass	175.96±48.4	139.27±41.3	<0.001

DISCUSSION

The decrease in LV mass, with a definite indication of reverse hypertrophy, has been proven to take place following bariatric surgery. This remodelling is reversed, probably due to reduced hemodynamic load, reduced blood pressure on weight loss, and reduced neurohormonal activity.[7] It is thought to be an effect of mitigation of obesity-related inflammatory and metabolic derangements that cause ventricular hypertrophy, impaired diastolic filling, reduced myocardial performance, diminished coronary reserve,

continued volume overload, and electrical instability, thus resulting in better hemodynamic status and inducing reverse cardiac remodeling. Reverse remodeling is supported by a large study (n=398) which found improved global longitudinal strain with improvement in LV mass (205 g to 190 g) in the post-surgery period.[8,9]

Diastolic dysfunction is characterized by impaired ventricular relaxation, high filling pressures and a relative normal systolic functioning.[9] Obesity is linked to poorer myocardial relaxation, fibrosis and high LV filling pressures. Weight loss increases weight by increasing the stress on the hemodynamic and metabolic systems, which results in a higher diastolic performance. The idea that a reduction in velocity of A-wave further implies a reduction in the dependence on atrial contraction to fill up the diastolic function which is an indication of normalization of the A-wave filling pattern is justified by the study that weight loss in 12-month diet reduced the velocity of A-wave and augmented E/A ratio without alteration in left atrial diameter. The e-wave velocity and LA size did not differ significantly during the follow up period, and it is possible that, even though relaxation process was improved, there were no significant changes in global early filling flow velocity and structural atrial remodelling. Their data showed a positive decreasing tendency of the mean E/e 7 with weight loss and only slightly greater (between 8 and 10) values at baseline; however, no statistically significant differences were detected.[10]

Hughes et al[11] reported that the E/e ratios dropped dramatically after bariatric surgery or weight loss methods that do not involve surgery.

The proportion of peak velocity blood flow during left ventricular relaxation at early diastole (E wave) compared to peak velocity flow during atrial contraction at late diastole (A wave) is referred to as the E/A ratio. It is thus a pointer of the proper functioning of the left ventricle of the heart.[12] A meta-analysis of 36 studies involving 680 participants showed that there was an average value of the E/A ratio of 0.155 (95% CI: 0.106-0.205; $p < 0.001$) increase in the surgical intervention. The correction of obesity-induced changes in heart shape and performance, regulated by neurohormonal and metabolic status, a major increase in ē velocity, which is a sign of improved myocardial relaxation, can be achieved by bariatric surgery.[13]

The primary purpose of the study included echocardiographic structural and functional changes, despite the fact that blood pressure and anthropometric changes were improved after surgery, which is consistent with other studies in the field.[14,15]

The weakness of this research is that it has a small population and does not examine systolic performance and LV mass index. Also, the duration of follow-up might restrict the analysis of the long-term cardiac remodeling. Nevertheless, the current research has demonstrated evidence of positive structural and diastolic cardiac remodelling after bariatric surgery in spite of these limitations.

CONCLUSION

Left ventricular mass regression and improvement of diastolic performance are found in relation to bariatric surgery in obese patients. These findings demonstrate the positive impact of weight loss on cardiac remodeling of obesity and justify that echocardiographic determination of the LV mass and diastolic value can be used to assess the cardiovascular recovery after bariatric surgery.

Author's Contribution:

Concept & Design or acquisition of analysis or interpretation of data:	Faten Abdul-lateef Abdul-Zahra, Asaad Hasan Noaman
Drafting or Revising Critically:	Faten Abdul-lateef Abdul-Zahra, Safauldeen Salim Albaaj
Final Approval of version:	All the above authors
Agreement to accountable for all aspects of work:	All the above authors

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REFERENCES

1. Avelar E, Cloward TV, Walker JM, Farney RJ, Strong M, Pendleton RC, et al. Left ventricular hypertrophy in severe obesity: interactions among blood pressure, nocturnal hypoxemia, and body mass. Hypertension (Dallas, Tex.: 1979), 2007; 49(1):34-9.
2. Noaman AH, Dananah FM, Khuthur AAB, Jasim SA. Left ventricle dilatation: early marker of structural remodelling of the heart in obese people. Medical Forum Monthly 2025;36(3): 48-50.
3. Yahya M, Al-Muthanna I, Al-Dujaili AAB. Correlation between the posterior left ventricular wall thickness and relative wall thickness with global longitudinal strain (GLS) in women with preeclampsia. J Rare Cardiovasc Dis 2025;5(1): 1-7.
4. Lastra G, Sowers JR. Obesity and cardiovascular disease: role of adipose tissue, inflammation, and the renin-angiotensin-aldosterone system. Hormone Molecular Biology and Clinical Investigation 2013; 15(2), 49-57.

5. Barzizza F. Obesity and the heart. Minerva gastroenterologica e dietologica 2001;47(4): 229-34.
6. Al-Dejeli AAB, Mohammed HH, Abbas BS, Jbaraah AH, Al-Aboodi AHN. Flow propagation velocity as a marker of impaired early diastolic flow in pre-hypertensive individuals. J Pharm Res 2020;12(3).
7. Alpert MA, Karthikeyan K, Abdullah O, Ghadban R. Obesity and cardiac remodeling in adults: mechanisms and clinical implications. Progress Cardiovascular Diseases 2018;61(2):114-23.
8. Ammar W, Basset HA, al-Faramawy A, Hegazy T, Sharaf Y. Bariatric surgery and cardiovascular outcome. Egyptian Heart J 2020; 72(1): 67.
9. Sargsyan N, Chen JY, Aggarwal R, Fadel MG, Fehervari M, Ashrafian H. The effects of bariatric surgery on cardiac function: a systematic review and meta-analysis. International Journal of Obesity 2024;48(2):166–76.
10. Noaman AH, Jasim SAH, Dananah FM, Hameed HG. Valsalva maneuver is a simple and effective clinical tool for pseudo-normal diastolic dysfunction. Vasc Endovasc Rev 2025;8(1): 37–40.
11. Hughes D, Aminian A, Tu C, Okushi Y, Saijo Y, Wilson, R, et al. Impact of Bariatric Surgery on Left Ventricular Structure and Function. J Am Heart Assoc 2024;13(1):e031505. https://doi.org/10.1161/JAHA.123.031505
12. Karimian S, Stein J, Bauer B, Teupe C. Improvement of impaired diastolic left ventricular function after diet-induced weight reduction in severe obesity. Diabetes, Metabolic Syndrome and Obesity : Targets and Therapy 2017;10:19–25.
13. Yuksel IO, Akar Bayram N, Koklu E, Ureyen CM, Kucukseymen S, Arslan S, et al. Assessment of impact of weight loss on left and right ventricular functions and value of tissue doppler echocardiography in obese patients. Echocardiography (Mount Kisco, NY) 2016; 33(6): 854-61.
14. Humiecka M, Sawicka A, Tarnowski W, Jankowski P. The effect of bariatric surgery on blood pressure in short- and long-term follow-up. J Hypertension 2025;43(Suppl 1): e190.
15. Jamialahmadi T, Abdalla MA, Mirhadi E, Almahmeed W, Sukhorukov VN, Virani S, et al. Impact of metabolic and bariatric surgery on heart rate variability: a systematic review and meta-analysis. Updates Surg 2025;15-9.