Original Article

Cukurova Anestezi ve Cerrahi Bilimler Dergisi

The Impact of Bariatric Surgery on Reverse Cardiac Remodeling and Serum Galectin-3 Levels

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Abstract

Aim: We aimed to investigate the effect of laparoscopic sleeve gastrectomy (LSG) on cardiac reverse remodeling and fibrosis in obese patients. **Methods:** This prospective study included 72 consecutive patients who underwent LSG for morbid obesity and 72 age- and sex-matched healthy controls between 2020 and 2022. Clinical and echocardiographic evaluations, including serum Galectin-3 (Gal-3) levels and anthropometric measurements, were performed at baseline and at the 6th month postoperatively. **Results:** There was a significant increase in the mean mitral E peak velocity ($p<0.001$) and E/A ratio ($p<0.001$), as well as a significant decrease in the left atrial (LA) diameter ($p=0.015$), left ventricular end-diastolic diameter (LVEDD) ($p<0.001$), left ventricular (LV) mass ($p<0.001$), left atrial volume (LAV) ($p<0.001$), left atrial volume index (LAVI) ($p<0.001$), and mean mitral A peak velocity ($p<0.001$). In the patient group, a non-significant decrease was observed in the Gal-3 levels postoperatively compared with the preoperative levels ($p=0.146$). **Conclusion:** Our findings suggest that LSG is a highly effective intervention for improving cardiac structure and function in morbidly obese patients, contributing significantly to reverse cardiac remodeling. The lack of statistically significant decrease in galectin-3 levels ($p=0.146$) may be due to the fact that the fibrosis process takes longer to reverse than the structural changes, or possibly due to the sample size.

Keywords: Galectin-3; obesity; laparoscopic sleeve gastrectomy; LV mass index; reverse cardiac remodeling

1. Introduction

Obesity, a prevalent metabolic disorder, is a grave global health concern, impacting individuals across all age demographics. It is one of the leading global public health issues and is associated with many other disorders including cardiovascular diseases (CVD), diabetes mellitus (DM), musculoskeletal diseases, and certain types of malignancy[1-3]. Obesity exerts deleterious effects on cardiac structure and is a well-established risk factor for CVD[3,4].

Short-term outcomes of bariatric surgery are favorable for weight loss and management of comorbidities in patients with morbid obesity. Bariatric surgery provides hormonal/neural changes combined with dietary adjustments, which constitute an important

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cornerstone in the management of obesity-related metabolic disorders[5]. In recent years, LSG has been widely used and is increasingly becoming a popular surgical procedure, as it offers certain advantages over traditional bariatric surgery, such as limited changes in the normal anatomy, shorter operation time, and rapid weight loss[6,7]. It has been evidenced in numerous studies that the LSG procedure is an efficacious treatment for obesity-related diseases[8,9].

Morbid obesity is a serious condition and can cause cardiac structural and hemodynamic alterations which predispose to ventricular dysfunction and heart failure (HF). Cardiac remodeling is defined as molecular, cellular, and interstitial alterations in the diameter, shape, and function of one or more chambers of the heart[10-12]. Reverse remodeling is a dynamic process involving multiple biochemical and molecular alterations that affects the cardiac function and reserve capacity of the heart[10,11]. Conditions that reduce or eliminate neurohormonal and/or hemodynamic factors are involved in the reverse remodeling process[12].

The objective of this study was to evaluate the impact of LSG-induced weight loss on reverse cardiac remodeling and to investigate its effects on myocardial fibrosis markers in morbidly obese patients.

2. Materials and Methods

2.1.Data Collection and Study Setting

This study was planned as a prospective cohort study and was conducted in the general surgery clinic between October 2020 and February 2022. The hospital where the study was conducted is a tertiary health center. Informed consent was obtained from all patients. This study was approved by the Cukurova University, Non-Interventional Research Ethics Committee (No.: 103, Date: 03/09/2020).

All surgical procedures were performed by the same surgical team using a standardized technique (Laparoscopic sleeve gastrectomy). Patients with a body mass index (BMI) of ≥40 kg/m² without any obesity-related comorbidity or those with a BMI of ≥35 kg/m² and at least one comorbidity such as obesity-related type 2 DM, hypertension, chronic heart diseases (CHD), dyslipidemia, or obstructive sleep apnea syndrome (OSAS), who underwent LSG were included. The exclusion criteria were as follows: coronary artery diseases (CAD), peripheral arterial disease (PAD), low echocardiographic image quality, valve diseases including rheumatic mitral valve disease, atrial fibrillation (AF), LV dysfunction, congenital heart disease, cerebrovascular disease, renal and hepatic insufficiency, malignancy, endocrine diseases related to obesity such as Cushing syndrome or other endocrine obesity, and no consent for participation in the study. Finally, 72 consecutive obese patients who underwent LSG were included in the study. The control group was comprised of healthy individuals matched for age and gender with the patient group.

2.2.BMI and Body Surface Area (BSA) Measurements

BMI was estimated by kilogram/meter². The calculation of the BSA was performed in accordance with the formula established by Mosteller et al.[13] by multiplying the individual's height in centimetres by his or her weight in kilograms and dividing the value by 3600.

2.3.Transthoracic Echocardiographic (TTE) Examination

All participants underwent TTE using a Vivid S5 cardiovascular ultrasound device (GE Medical Systems, Buckinghamshire, UK) with a 3S probe at 1.5 to 3.6 MHz. After a 5-minute rest, images were obtained in the left lateral decubitus position using standard apical 4-chamber, apical 2-chamber, parasternal long- and short-axis views. Three measurements were recorded at the end of expiration. All measurements were performed by two independent cardiologists. Left ventricular end-systolic diameter (LVESD), LVEDD, interventricular septum (IVS) thickness, and posterior wall (PW) thickness were measured using the parasternal long-axis view. Two-dimensional (2D) M-mode imaging was used for these measurements. LV ejection fraction (LVEF) was determined using the Simpson calculation method. The anteroposterior diameter of the LA was calculated at end-systole of the LV using parasternal long-axis views. The other diameters of the LA were measured using the apical four-chamber views, and superior-inferior (longitudinal) and medial-lateral (transverse) diameters were calculated at the maximal width of the LV, when the mitral valve was closed. The LA area was calculated based on the largest volume in the end-diastolic phase before mitral valve opening. All measurement procedures were conducted in accordance with the standards and recommendations outlined by the American Society of Echocardiography (ASE)[14]. The ellipsoid method was used to calculate the LAV. The LAVI was obtained by dividing the LAV by BSA. The LV mass was estimated using the following simplified formula: $0.8 \times 1.04 \times [(IVSd + LVIDd + PWd)^3 - LVIDd^3] + 0.6$ g[14,15]. The LV mass index was calculated by dividing the LV mass by the BSA.

Transmitral diastolic filling pattern was assessed using pulse-wave Doppler imaging, whereas mitral annular (medial and lateral) velocities were assessed using tissue Doppler imaging. Pulse-wave Doppler imaging was used to record LV filling patterns. Deceleration times, E/A ratios, and peak E and A velocities were recorded[16,17].

2.4.Measurements of Biochemical Parameters and Galectin-3 (Gal-3) Level

As part of the standard protocol, venous blood sampling was performed after completing a 12-hour overnight fast. Lipid profile, fasting blood glucose (FBG), fasting insulin and glycosylated hemoglobin (HbA1c) analyses were performed.

Blood samples taken 1 hour before LSG were centrifuged at 3000 × g for 15 minutes and Gal-3 values were analyzed. The resultant sera were stored at −80°C. The same procedure was applied to the healthy control group. The specimens were centrifuged at 3,000 × g for 15 min to evaluate Gal-3 levels, and the resultant sera were stored at −80°C. All the specimens were analyzed synchronously. On the day of analysis, the sera were maintained at room temperature and analyzed. Gal-3 measurements were calculated and recorded using the same measurement method in both the preoperative and postoperative periods. All analyses were performed in the laboratory at our center. The results are presented in ng/ml.

2.5.Statistical Analysis

Statistical analyses were conducted using SPSS software, version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables are presented as mean ± standard deviation (SD) or median (minimum–maximum), whereas categorical variables are expressed as counts and percentages. The appropriate statistical test was used to compare categorical variables, namely the Chi-square or Fisher's exact test. The Shapiro–Wilk test was applied in order to assess the normality of distribution. Non-normally distributed continuous variables were analysed using the Mann–Whitney U test. A p-value of below 0.05 was considered statistically significant.

3. Results

The study comprised a total of 72 obese patients who underwent LSG and 72 healthy controls. Patients with morbid obesity (n=28), low echocardiographic image quality (n=15), LV systolic dysfunction (n=2), rheumatic mitral valve disease (n=2), AF (n=2), or lack of written informed consent (n=7) were excluded. All patients were evaluated before and after surgery.

The mean age was 34.7±10.2 (18–69) years, 105 (72.9%) were female. Hypertension was observed in 36 patients (16.7%), DM in 25 patients (11.6%), and OSAS in 12 patients (5.6%). Forty (18.5%) participants were smokers. The mean body weight was 88.8±25.2 kg, the mean height was 1.67±0.09 cm, the mean BMI was 31.9±9.3 kg/m², and the mean BSA was 2.01±0.3 m² (Table 1).

Table 1. Demographic and clinical characteristics of participants

	Overall (n=144)	(%)
Sex		
Male	39	27.1
Female	105	72.9
HT	36	16.7
DM	12	5.6
OSAS	12	5.6
Smoking	40	18.5
	Mean ± SD	**Median (min-max)**
Age, year	34.7±10.2	32 (18-69)
Body weight, kg	88.8±25.2	80 (46-180)
BMI, kg/m²	31.9±9.3	29.0 (18-55.3)
BSA, m²	2.01±0.3	1.96 (1.4-3.1)

HT: hypertension, DM: diabetes mellitus, OSAS: obstructive sleep apnea syndrome, BMI: body mass index, BSA: body surface area

The mean preoperative BMI, BSA, and body weight of patients who underwent LSG was found to be significantly higher than the control group. Postoperatively, mean BMI, BSA, and body weight also remained significantly higher in the LSG group. The finding that preoperative mean systolic and diastolic blood pressures were higher for patient group (both $p < 0.001$). Postoperative mean systolic blood pressure was significantly higher in the patient group ($p < 0.001$). Furthermore, preoperative mean insulin resistance homeostasis model assessment (HOMA-IR), FBG, HbA1c, TG, and Gal-3 levels were significantly higher for the patient group. In contrast, preoperative mean HDL-C levels were found to be significantly lower for the patient group ($p < 0.001$). Postoperatively, mean FBG ($p = 0.020$) and HOMA-IR ($p = 0.015$) levels remained significantly higher and the mean HDL-C levels ($p < 0.001$) remained significantly lower for patient group.

The mean postoperative systolic and diastolic blood pressures were significantly lower than the preoperative values (both $p < 0.001$). The mean postoperative body weight, BMI, and BSA were significantly lower than the preoperative values. In addition, the mean HDL-C level ($p = 0.009$) significantly increased postoperatively, whereas the mean TG ($p < 0.001$), HbA1c ($p < 0.001$), FBG ($p = 0.002$), and HOMA-IR ($p < 0.001$) significantly decreased after surgery (Table 2).

Table 2. Anthropometric measurements and laboratory test results

	Control (n=72)	Patient group (preop.) (n=72)	Patient group (postop.) (n=72)	p1+	p2+	p3++
	Mean ± SD	**Mean ± SD**	**Mean ± SD**			
Age, year	32.6±8.1	36.8±11.6	-	0.021*	-	-
Body weight, kg	66.2±9.9	119.5±11.9	80.7±11.8	<0.001	<0.001	<0.001
Height, cm	1.69±0.08	1.65±0.09	1.65±0.09	0.004	0.004	-
BMI, kg/m²	22.9±1.8	43.6±4.2	29.4±3.8	<0.001	<0.001	<0.001
BSA, m²	1.76±0.2	2.34±0.2	1.92±0.2	<0.001	<0.001	<0.001
SBP, mmHg	118.11±12.92	146.23±14.21	137.34±11.52	<0.001	<0.001	<0.001
DBP, mmHg	73.32±7.12	86.32±13.12	77.32±8.12	<0.001	0.075	<0.001
HbA1c	5.16±0.4	6.14±1.8	5.53±1.1	<0.001	0.215	0.001
FBG, mg/dL	84.9±7.9	108.1±38.7	93.8±24.2	<0.001	0.020*	0.002
HOMA-IR	1.10±0.4	4.85±6.4	1.95±3.4	<0.001	0.015*	<0.001

LDL-C, mg/dL	112.9±36.4	121.7±38.1	121.3±37.2	0.150	0.102	0.745
HDL-C, mg/dL	57.5±13.3	45.1±9.9	49.7±10.8	<0.001	<0.001	0.009
TG, mg/dL	131.6±147.7	145.0±80.6	109.9±46.2	0.001	0.304	0.001
TC, mg/dL	193.3±44.2	195.9±45.2	193.3±46.1	0.727	0.876	0.751
Gal-3, ng/dL	6.9±4.4	9.0±4.6	7.8±3.8	0.002	0.070	0.146

**p<0.05, p<0.001, +: Mann-Whitney U test, ++: Wilcoxon rank test, p1: control vs. preoperative patient group, p2: control vs. postoperative patient group, p3: preoperative patient group vs. postoperative patient group. Preop.: preoperative, postop.: postoperative, BMI: body mass index, BSA: body surface area, SBP: systolic blood pressure, DBP: diastolic blood pressure, HbA1c: glycated hemoglobin, FBG: fasting blood glucose, HOMA-IR: homeostasis model assessment of insulin resistance, HDL-C: high-density lipoprotein cholesterol, LDL-C: low-density lipoprotein cholesterol, TC: total cholesterol, TG: triglyceride, Gal-3: galectin-3.*

The mean preoperative LA diameter, LA area, LVEDD, LVESD, LV mass, LV mass index, LAV, LAVI (mL/m²), IVS, PW, and E/e′ ratios were found to be significantly higher for patient group. Furthermore, a comparative analysis revealed that the mean preoperative mitral E peak velocity, EF, E/A ratio, mitral A peak velocity, mitral medial e′ peak velocity, and mitral lateral e′ peak velocity were significantly lower for patient group ($p < 0.05$). The mean postoperative LV mass, LAV, LV mass index, LAVI, LA diameter, LA area, PW, IVS, E/e′ ratio, and E/A ratio were significantly higher for the patient group ($p < 0.05$). The mean postoperative mitral E peak velocity, mitral A peak velocity, mitral lateral e′ peak velocity, and mitral medial e′ peak velocity were significantly lower than those in the control group ($p < 0.05$).

When mean EF, mitral E peak velocity and E/A ratios were evaluated, preoperative values were found to be lower than postoperative values ($p < 0.05$). In addition, when the preoperative values of mean LA diameter, LVEDD, LV mass, LAV, LAVI, IVS, PW, mitral A peak velocity and E/e′ ratio were compared with the postoperative values, it was seen that these measurements were higher preoperatively and this was statistically significant ($p < 0.05$) (Table 3).

There was no mortality or morbidity in any of the patients.

Table 3. Comparison of echocardiographic parameters

	Control (n=72)	Patient group (preoperative) (n=72)	Patient group (postoperative) (n=72)	p1+	p2+	p3++
	Mean ± SD	Mean ± SD	Mean ± SD			
EF (%)	66.6±3.6	64.5±5.5	66.2±5.6	0.028*	0.971	0.039*
LA size, mm	33.2±1.7	37.4±4.1	35.9±3.2	<0.001	<0.001	0.015*
LA area, cm²	15.2±1.3	20.5±2.9	19.7±3.0	<0.001	<0.001	0.068
LVEDD, mm	46.3±1.9	47.8±3.6	45.6±2.8	<0.001	0.136	<0.001
LVESD, mm	26.7±1.4	27.7±2.6	27.1±2.2	0.007	0.246	0.112
LV mass, g	148.5±20.0	217.8±42.3	190.3±48.3	<0.001	<0.001	<0.001
LV mass index, g/m²	84.4±10.2	92.9±17.1	99.4±26.4	<0.001	<0.001	0.389
LA volume, mL	30.4±1.9	50.5±5.2	38.7±4.7	<0.001	<0.001	<0.001
LA volume index, mL/m²	17.4±2.3	21.5±1.7	20.1±3.1	<0.001	<0.001	<0.001
IVS, mm	8.1±0.8	10.4±1.0	10.0±2.4	<0.001	<0.001	0.001
PW, mm	7.9±0.9	10.0±1.4	9.4±1.0	<0.001	<0.001	0.015*
Mitral E peak velocity, cm/sec	87.4±6.8	72.1±7.5	78.0±6.9	<0.001	<0.001	<0.001
Mitral A peak velocity, cm/sec	61.8±5.5	56.8±9.5	49.9±6.5	<0.001	<0.001	<0.001
E/A	1.41±0.1	1.29±0.2	1.57±0.1	<0.001	<0.001	<0.001

Mitral lateral e′ peak velocity, cm/sec	13.9±1.1	11.9±1.7	12.3±1.1	<0.001	<0.001	0.409
Mitral medial e′ peak velocity, cm/sec	11.6±0.9	9.0±0.5	9.4±0.9	<0.001	<0.001	0.072
Mean E/e′	3.5±0.4	6.9±1.0	3.6±0.4	<0.001	0.014*	<0.001

**p<0.05, p<0.001, +: Mann-Whitney U test, ++: Wilcoxon rank test, p1: control vs. preoperative patient group, p2: control vs. postoperative patient group, p3: preoperative patient group vs. postoperative patient group. Preop.: preoperative, postop.: postoperative, EF: ejection fraction, LV: left ventricle, LA: left atrium, LVEDD: left ventricular end-diastolic diameter, LVESD: left ventricular end-systolic diameter, IVS: interventricular septum, PW: posterior wall.*

4. Discussion

The present study investigates the effect of LSG on cardiac reverse remodelling and fibrosis in obese patients. The primary conclusion of our study was that LSG enhances LV systolic and diastolic functions, as demonstrated by TTE. The present study demonstrated a substantial enhancement in LVEF, LAV, LAVI, LV mass, mitral E peak velocity, A peak velocity, E/A ratios, and LVEDD. Furthermore, a substantial enhancement in the cardiac risk profile of this patient cohort was observed. In the study, there was no mortality or morbidity in any of the patients, which can be attributed to the effective use of laparoscopic techniques in recent years, the more frequent use of LSG than other bariatric procedures, and increased surgical experience.

Cardiac remodeling is a term used to describe alterations in the diameter, shape, and function of one or more chambers of the heart. These alterations can be classified as functional, structural, cellular, and interstitial. Remodeling has been most frequently described for LV and LA[10,11]. The reverse remodelling process, which is characterised by effective weight loss following bariatric surgery, has been demonstrated to be associated with enhanced ventricular functions, cardiac morphology, and functional capacity in patients diagnosed with heart failure (HF)[12]. Our study showed that a significant improvement in diastolic function was observed with increased E peak velocity, reduced A peak velocity, and increased mitral E/A ratio. Furthermore, during the six-month follow-up period, a significant decrease in the LA mass, LAV, and LA mass index was observed, as confirmed by TTE. These findings are consistent with the extant literature[18-22].

Although the exact pathophysiological mechanism of bariatric surgery implicated in reverse remodeling is unclear, decreased systolic and diastolic blood pressures can improve LV hypertrophy, reduce LAV, and reduce ventricular mass. One possible explanation for the improved diastolic functions is altered ventricular mass and LAV. Additionally, improved blood lipid profiles and metabolic parameters related to weight loss, and hormonal alterations can improve diastolic function.

Several studies have demonstrated that bariatric surgery improves LV systolic functions[23-27]. Our patient population was relatively young with fewer comorbid factors than those in previous studies, which possibly revealed a significant improvement in systolic function. Although three-dimensional (3D) measurement of LV and EF is more sensitive, feasible, and highly reproducible than 2D measurement, we used M-mode 2D TTE in our study. In numerous studies, the evaluation of systolic function has been conducted employing 2D speckle tracking echocardiography or 3D echocardiography. As the aforementioned methods were not employed in the present study, a comparison of the EF values with those reported in previous studies was not possible.

Galectin-3 has been shown to induce cardiac inflammation and fibrosis and is associated with diastolic dysfunction, and inhibition of Gal-3 reduces cardiac inflammation and fibrosis[28]. As posited by Florido et al.[29], elevated Gal-3 levels exhibit a marked correlation with obesity. It is imperative to elucidate the role of obesity in the processes of cardiac inflammation and fibrosis. In the present study, the mean Gal-3 levels were found to be significantly elevated in patients with a high BMI prior to surgery. A tendency towards a decrease in Gal-3 levels post-surgery was observed; however, this difference did not attain statistical significance. The absence of a substantial discrepancy in the Gal-3 levels prior to and following surgery can be ascribed to the comparatively brief duration of the postoperative follow-up period. Further long-term studies are required to evaluate Gal-3 levels and to investigate the association between cardiac alterations and fibrosis.

In the present study, a significant decrease in BMI was achieved, which is consistent with the findings of previous studies[28-31]. The augmented decrease in BMI after LSG in our study, as compared to the findings of previous studies, can be elucidated by the fact that the patients were meticulously selected for surgery by a multidisciplinary team, and all patients underwent non-pharmacological treatment in addition to LSG, utilising a stringent postoperative programme with enhanced patient compliance.

Surgical treatment of obesity is an effective method for weight loss, which is implicated in the management of obesity-related conditions[32]. In the present study, a significant decrease in systolic and diastolic blood pressure was observed, along with a notable improvement in HT, DM, and dyslipidaemia. In a study conducted by Buchwald et al.[33] reported that patients undergoing bariatric surgery demonstrated a return to normal blood pressure or improvement in blood pressure, with a significant improvement in HbA1c and FBG levels. Another study, Eid et al.[34] documented a 77% clinical improvement in patients with DM who underwent sleeve gastrectomy, achieving complete remission. The enhancements in HbA1c, FBG, and HOMA-IR can be ascribed to the modified ghrelin and glucagon-like peptide-1 (GLP-1) levels following LSG. Long-term follow-up can provide a more accurate evaluation of the effects of LSG on comorbidities in these patients.

In the present study, the mean postoperative HDL-C levels were found to be significantly higher, while the mean postoperative TG levels were found to be significantly lower than the preoperative values. However, no statistically significant differences were observed in the mean LDL-C and TC levels before and after surgery. Despite improved lipid profiles in operated patients, hormonal factors such as ghrelin and GLP-1, genetic factors, consumption patterns, and lifestyle changes may also be implicated.

4.1 Limitations

The present study was subject to certain limitations. Initially, the study design featured a single-centre approach and a comparatively limited sample size. Secondly, the performance of 3D tissue echocardiography was not possible, which may have prevented the acquisition of more accurate data. Clearer and stronger findings could have been obtained with an echocardiography device that provides more powerful images. Thirdly, although BMI is a valuable variable in the diagnosis of obesity, measurement of body fat tissue using more specific imaging tools may have yielded more reliable conclusions. Finally, the follow-up period for patients was limited to six months. The lack of longer-term follow-up can be considered a limitation of this study. It is evident that further large-scale, multicentre, prospective studies are required in order to provide robust confirmation of these findings.

5. Conclusions

Our study findings suggest that LSG is a viable method for treating obesity through effective weight loss, and that it provides positive improvements in cardiovascular risk profile and LV systolic and diastolic function. Furthermore, our findings indicate that it significantly contributes to the reversal of cardiac remodeling.

genAI: This article was written without the use of artificial intelligence.

Statement of Ethics: This study was approved by the Cukurova University, Non-Interventional Research Ethics Committee (No.: 103, Date: 03/09/2020).

Animal and Human Rights Statement: All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

Informed Consent: Informed consent was obtained from all patients who participated in this study.

Scientific Responsibility Statement: The authors declare that they are responsible for the article's scientific content, including study design, data collection, analysis and interpretation, writing, and some of the main line, or all of the preparation and scientific review of the contents, and approval of the final version of the article.

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AA: Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Software, Supervision, Validation, Visualization, Roles/Writing – original draft, Writing – review & editing.

OY: Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Software, Supervision, Validation, Visualization, Roles/Writing – original draft, Writing – review & editing.

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