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Evaluation of the Effect of Weight Loss After Sleeve Gastrectomy on Ventricular Repolarization Parameters

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ABSTRACT

Objective: Obesity is associated with adverse alterations in ventricular repolarization and increased electrophysiological heterogeneity. Sleeve gastrectomy provides substantial and sustained weight loss; however, its effects on ventricular repolarization parameters assessed by surface electrocardiography (ECG) remain incompletely characterized. The aim of this study was to evaluate the effect of weight loss after sleeve gastrectomy on ventricular repolarization parameters assessed by surface ECG.

Materials and Methods: This retrospective study included 95 morbidly obese patients who underwent laparoscopic sleeve gastrectomy. Standard 12-lead electrocardiograms were evaluated preoperatively and during routine follow-up approximately 6 months after surgery. Changes in ventricular repolarization parameters were analyzed in relation to post-operative weight loss.

Results: Substantial weight loss was observed at follow-up. Among time-based electrocardiographic parameters, modest but statistically significant post-operative reductions were detected in corrected QT interval (QTc), QT interval dispersion (QTd), and corrected JT interval (JTc), while all values remained within generally accepted physiological ranges. In addition, ratio-based indices reflecting relative depolarization-repolarization timing, including Tpeak–Tend dispersion/QT and QRS duration/QT, showed significant post-operative decreases. No significant post-operative changes were observed in other ventricular repolarization parameters.

Conclusion: Weight loss following sleeve gastrectomy was associated with subtle changes in selected ventricular repolarization parameters detectable on surface ECG, suggesting limited electrophysiological adaptation rather than overt alterations in myocardial conduction or repolarization. The clinical implications of these findings remain uncertain, and further prospective studies with longer follow-up are warranted to clarify the electrophysiological effects of sleeve gastrectomy.

Keywords: JTc interval, Obesity, QT dispersion, QTc interval, Sleeve gastrectomy, Ventricular repolarization

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INTRODUCTION

Obesity is a preventable metabolic risk factor that is rapidly increasing globally and is considered a significant public health problem.[1] Excess body weight is associated not only with metabolic disorders but also with an increased risk of coronary heart disease and sudden cardiac death.[2-4] Furthermore, obesity has been shown to adversely affect cardiac electrical stability, with evidence of altered ventricular repolarization and increased electrophysiological heterogeneity. These changes are thought to arise in part from obesity-related myocardial remodeling and ectopic adipose tissue accumulation, ultimately leading to complex alterations in cardiac structure and electrical conduction.[2,5,6] Moreover, a better understanding of obesity-related electrocardiographic changes and the identification of cardiac risk markers reflecting these processes is becoming increasingly important.

In terms of effective and sustainable weight loss, long-term studies have shown that diet and lifestyle interventions generally result in modest weight loss, while pharmacological treatments provide a slightly greater but still limited effect over time.[7] Therefore, it is stated that achieving permanent weight loss is often only possible with surgical methods, especially in individuals resistant to lifestyle and medical approaches.[7-10] Bariatric surgery stands out as a powerful treatment option because it provides significant and long-term weight loss, as well as reducing cardiometabolic morbidity, and long-term mortality.[8,10] According to the international records, sleeve gastrectomy is the most commonly performed bariatric procedure worldwide.[11] With the demonstration of the beneficial effects of weight loss on cardiac functions, studies examining changes in parameters reflecting ventricular repolarization on electrocardiography (ECG) before and after obesity surgery have also increased in recent years.[12-14]

It has been reported that obese individuals show significant impairments in ECG parameters reflecting the duration and homogeneity of ventricular repolarization.[15] Significant and sustained weight loss achieved after bariatric surgery has been shown to reduce repolarization heterogeneity by significantly decreasing corrected QT dispersion (QTcd), corrected JT dispersion (JTcd), and transmural repolarization indicators.[13] In addition, the ameliorative effects of weight loss on corrected QT interval (QTc) and QTcd have been reported, particularly in relation to the regression of left ventricular hypertrophy.[14] Sleeve gastrectomy series also report that, consistent with these findings, significant improvements can be observed in parameters related to ventricular repolarization measured on ECG in the post-operative period, and this may potentially contribute to a reduction in the risk of ventricular arrhythmias and sudden cardiac death; however, it has been emphasized that these results need to be confirmed in larger cohorts.[12]

Therefore, it is clear that further studies are needed to better understand the changes in electrocardiographic parameters associated with ventricular repolarization after sleeve gastrectomy. The aim of this study is to evaluate the effect of post-operative weight loss on ventricular repolarization indices in morbidly obese patients who underwent sleeve gastrectomy.

MATERIALS AND METHODS

The study was approved by the Gaziantep University Clinical Research Ethics Committee (Decision No: 2023/244, Date: 29.08.2023). We retrospectively included a total of 95 patients who underwent laparoscopic sleeve gastrectomy at the Gaziantep University General Surgery Clinic between January 2018 and January 2023. The study was conducted in accordance with the Declaration of Helsinki. Demographic and clinical data – including age, sex, body mass index (BMI), pre-operative and 6-month post-operative weight, comorbidities, smoking status, and American Society of Anesthesiologists (ASA) classification – were routinely recorded during anesthesia preparation. The necessary information was retrieved from the hospital's electronic medical record system and, when required, from archived paper files.

Patients with BMI ≥40 kg/m^2, or ≥35 kg/m^2 accompanied by obesity-related comorbidities who failed to achieve adequate weight loss with conservative methods, were considered eligible for surgery. Because this study was retrospective and pre-operative echocardiography is not routinely performed during the standard pre-operative anesthesia evaluation in our center, echocardiographic data were not available for all patients. However, if a previous echocardiogram in the patient record showed structural abnormalities – such as left-ventricular systolic dysfunction (Ejection fraction <50%), significant valvular disease, or left ventricular hypertrophy – these patients were excluded to avoid confounding effects on ventricular repolarization. Additional exclusion criteria included ischemic heart disease, electrolyte imbalance, Type 1 diabetes, missing ECG data, and the use of medications known to affect cardiac conduction. Stable, well-controlled hypertension was included, whereas patients with chronically uncontrolled hypertension or documented end-organ involvement were excluded from the study.

All operations were performed laparoscopically under general anesthesia. A 38–40 Fr bougie was used for sleeve calibration, and gastric resection was initiated 2–10 cm proximal to the pylorus.

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Pre-operative ECGs were obtained during routine anesthesia preparation, while post-operative ECGs were retrieved from routine follow-up visits performed approximately 6 months after surgery. All ECGs were digitally magnified and analyzed using imaging software (Adobe Photoshop, Version 19.1.6). Two cardiologists, who were unaware of any clinical information, independently assessed each ECG recording. If there were differences in measurements, the final value was calculated as the average of the two measurements to reduce variability between observers.

The study analyzed the following ECG parameters:

QRS duration (QRSd), QT, JT interval (JT), and Tpeak–Tend interval (Tp–e) were measured on standard 12-lead ECGs following established definitions. QRSd was measured from the start to the end of the QRS complex in each lead. The QT was measured from the start of the QRS complex to the end of the T wave, where it returned to the baseline. The JT was derived by subtracting QRSd from the QT (JT = QT - QRSd). The Tp–e was defined as the time from the peak of the T wave to its end.

To evaluate spatial variations in ventricular activation and recovery, dispersion values were calculated across all 12 leads. QRS dispersion, QT dispersion (QTd), JT dispersion (JTd), and Tp–e dispersion (Tp–ed) were defined as the difference between the highest and lowest values recorded across the leads.

To reduce the effect of heart rate on interval readings, corrected indices were calculated. QTc and corrected JT (JTc) values were computed using Bazett's formula.[16]

Ratio-based parameters, including Tp–e/QT, Tp-e/QRSd, and QRSd/QT, were determined from the respective interval readings.

To measure changes after surgery, delta (Δ) values were computed for each parameter using the formula Δ = pre-operative measurement – post-operative measurement. Positive Δ values indicated a decrease after surgery, while negative values showed an increase.

All time intervals were reported in milliseconds (ms). To avoid confusion with QRSd, the term QRS dispersion was always written out fully in the manuscript.

Statistical Analysis

The Shapiro–Wilk test was used to determine whether numerical data had a normal distribution. Two dependent measurements of normally distributed variables were compared using a paired t-test, whereas two dependent measurements of non-normally distributed variables were compared using a Wilcoxon test. The Chi-square test was used to examine relationships between categorical variables. Correlation coefficients were calculated to assess relationships between numerical variables.

These Δ values were compared between groups using the Mann–Whitney U test.

All analyses were performed using the Statistical Package for Social Sciences version 22.0 (IBM Corp., Armonk, NY, USA). A $p<0.05$ was considered statistically significant.

RESULTS

A total of 95 patients who underwent laparoscopic sleeve gastrectomy were evaluated in the study. Of these, 24 (25.3%) were male, and 71 (74.7%) were female. Among the study population, hypertension was present in 11 patients (11.6%), diabetes mellitus in 15 (15.8%), thyroid disease in 12 (12.6%), asthma in 9 (9.5%), insulin resistance in 6 (6.3%), and obstructive sleep apnea syndrome in 3 (3.2%). Thirty-two patients (33.7%) were active smokers. According to the ASA classification, 26 patients (27.4%) were classified as ASA II, while 69 (72.6%) were ASA III (Table 1).

The average age of participants was 35.47±11.71 years, with an age range of 18–64 years. The mean pre-operative BMI was 45.82±6.52 kg/m^2, while the mean BMI at 6 months postoperatively was 33.29±4.66 kg/m^2. Similarly, the mean pre-operative body weight was 125.82±19.43 kg, which decreased to 91.43±13.58 kg at 6 months following surgery.

Table 1. Baseline demographic and clinical characteristics of the patients (*n*=95)

Variable	*n*	Percentage
Male	24	25.3
Female	71	74.7
Hypertension	11	11.6
Diabetes mellitus	15	15.8
Obstructive sleep apnea syndrome	3	3.2
Thyroid disease	12	12.6
Asthma	9	9.5
Insulin resistance	6	6.3
Smoker	32	33.7
ASA physical status II	26	27.4
ASA physical status III	69	72.6

n: Number of patients, DM: Diabetes mellitus, OSAS: Obstructive sleep apnea syndrome, ASA: American Society of Anesthesiologists

Table 2. Mean values of demographic and anthropometric measurements (*n*=95)

Variable	Mean±SD	Median (Min-Max)
Age (years)	35.47±11.71	34 (18–64)
BMI (baseline) (kg/m^2)	45.82±6.52	44.3 (36.8–66)
BMI (6 months after surgery) (kg/m^2)	33.29±4.66	32.2 (24.8–47.4)
Weight (baseline) (kg)	125.82±19.43	120 (91–184)
Weight (6 months after surgery) (kg)	91.43±13.58	90 (64–130)
Amount of weight loss (kg)	34.39±7.30	33 (19–60)

n: Number of patients, BMI: Body mass index, SD: Standard deviation, Min-Max: Minimum-maximum, kg: Kilogram, kg/m^2: Kilogram/square meter

The mean total weight loss at the end of the 6-month follow-up period was 34.39±7.30 kg. The median values and ranges for each parameter are presented in Table 2.

Comparisons between baseline and post-operative 6-month electrocardiographic parameters are presented in Table 3. QTc values showed a significant decrease from 418.45±28.25 ms preoperatively to 412.01±28.27 ms postoperatively (p=0.013). QTd was also modest but statistically significantly reduced at the 6th post-operative month (53.93±10.95 ms vs. 52.57±10.20 ms, p=0.041). JTc values were significantly lower at follow-up compared with baseline (330.33±28.38 ms vs. 323.62±28.84 ms, p=0.017).

Table 3. Comparisons of baseline and post-operative 6th-month ECG findings

Parameter	Pre-operative (Mean±SD)	Post-operative (Mean±SD)	*p*
QTc	418.45±28.25	412.01±28.27	**0.013***
QTd	53.93±10.95	52.57±10.20	**0.041***
Tp-e	87.31±8.13	87.15±8.98	0.598
Tp-ed	55.98±15.49	57.43±17.02	0.722
Tp-e/QT	0.25±0.05	0.24±0.03	**0.011***
JTc	330.33±28.38	323.62±28.84	**0.017***
JTd	38.87±6.19	38.55±7.09	0.244
QRSd	88.13±8.81	88.39±7.62	0.836
QRS dispersion	28.12±3.52	28.38±3.95	0.549
QRSd/QT	0.25±0.03	0.24±0.03	**0.008***
Tp-e/QRSd	1.00±0.13	0.99±0.12	0.864

**p*: Statistical significance (*p*<0.05, Wilcoxon signed-rank test). All time-based ECG measurements were recorded in milliseconds. QTc: Corrected QT interval, QTd: QT dispersion, QT: QT interval, JTc: Corrected JT interval, JTd: JT dispersion, Tp–e: Tpeak–Tend interval, Tp–ed: Tpeak–Tend dispersion, QRSd: QRS duration, QRS dispersion: Maximum-minimum QRS duration across 12 leads, SD: Standard deviation

Although the absolute reductions observed in QTc, QTd, and JTc were modest in magnitude and both pre-operative and post-operative values remained within generally accepted normal ranges, these changes may reflect a subtle improvement in global ventricular repolarization timing and should be interpreted as surrogate electrophysiological markers rather than direct indicators of clinical risk reduction.

Regarding ratio-based parameters, the Tp–e/QT ratio demonstrated a small but statistically significant post-operative reduction (0.25±0.05 vs. 0.24±0.03, p=0.011). Similarly, the QRSd/QT ratio showed a modest yet statistically significant decrease at 6 months after surgery (0.25±0.03 vs. 0.24±0.03, p=0.008).

In contrast, no statistically significant differences were detected in Tp–e, Tp–ed, JTd, QRSd, QRS dispersion, or Tp–e/QRSd values (all p>0.05).

Comparative Δ (delta) values for female and male patients are shown in Table 4. No statistically significant variation was detected between the groups across any of the evaluated parameters (p>0.05).

Post-operative changes expressed as Δ values differed according to smoking status (Table 5). While no significant differences were observed between smokers and non-smokers for ΔBMI, ΔQTc, ΔTp–e, ΔTp–e/QT, ΔJTc, ΔJTd, ΔQRSd, QRSd/QT, or ΔTp–e/QRSd (all p>0.05), dispersion-based parameters demonstrated significant between-group differences. Specifically, ΔTp–ed was significantly different between smokers and non-smokers (−3.59±12.38 vs. 2.75±9.43, p=0.036), indicating a post-operative reduction in smokers and a relative post-operative increase in non-smokers (Fig. 1). Similarly, ΔQRS dispersion was significantly lower in smokers compared with non-smokers (−0.89±3.87 vs. 0.97±3.51, p=0.031), reflecting a post-operative decrease among smokers, whereas non-smokers exhibited a relative post-operative increase (Fig. 2). In addition, ΔQTd showed a borderline difference between smokers and non-smokers (p=0.059), indicating a trend toward a greater post-operative reduction in smokers that did not reach conventional statistical significance.

DISCUSSION

Obesity is known to alter the myocardial electrical environment through several mechanisms, including left ventricular hypertrophy, increased sympathetic activity, inflammation, oxidative stress, and excess pericardial and myocardial fat accumulation.[6] These alterations contribute to

Table 4. Comparison of Δ values (pre-operative – post-operative) according to gender

Parameter	Female (Mean±SD)	Median (Q1–Q3)	Male (Mean±SD)	Median (Q1–Q3)	*p*
ΔBMI	12.5±2.34	12.2 (10.8–13.6)	12.55±2.69	12.73 (10.8–13.3)	0.905
ΔQTc	4.8±40.59	8 (−14–28)	11.29±21.47	6.5 (−7.5–26)	0.451
ΔQTd	1.2±7.44	4 (−4–6)	1.83±7.42	2.5 (−4–6)	0.976
ΔTp-e	0.1±8.83	−2 (−4–4)	0.33±11.12	2 (−4–4)	0.782
ΔTp-ed	−1.58±12.47	2 (−6–6)	−1.08±9.83	2 (−7–5)	0.993
ΔTp-e/QT	0.02±0.06	0.01 (−0.02–0.03)	0.01±0.04	0.01 (−0.01–0.03)	0.784
ΔJTc	5.30±39.14	4.0 (−10.5–24.0)	10.88±22.84	9.5 (−9.25–26.75)	0.443
ΔJTd	0.14±5.79	2 (−4–4)	0.88±5.27	2 (−3.5–4)	0.983
ΔQRSd	−0.49±8.08	0 (−4–4)	0.42±7.64	1 (−4–4)	0.717
ΔQRS dispersion	−0.58±3.90	−2 (−4–2)	0.67±3.56	2 (−2–2)	0.203
ΔQRSd/QT	0.01±0.03	0.01 (−0.01–0.03)	0.01±0.03	0.01 (−0.01–0.03)	0.962
ΔTp-e/QRSd	0.01±0.11	0 (−0.06–0.05)	0.01±0.11	0.01 (−0.03–0.05)	0.613

**p*: statistical significance ($p<0.05$, Mann–Whitney U test); Δ was calculated as pre-operative value – post-operative value. Positive Δ indicates a decrease in post-operative values, whereas negative Δ indicates an increase. All time-based ECG measurements were recorded in milliseconds. BMI: Body mass index, QTc: corrected QT interval, QTd: QT dispersion, QT: QT interval, JTc: Corrected JT interval, JTd: JT dispersion, Tp–e: Tpeak–Tend interval, Tp–ed: Tpeak–Tend dispersion, QRSd: QRS duration, QRS dispersion: Maximum-minimum QRS duration across 12 leads, SD: Standard deviation, Q1-Q3: Interquartile range.

Table 5. Comparison of Δ values (Pre-operative – Post-operative) according to smoking status

Parameter	Non-smoker (Mean±SD)	Median (Q1–Q3)	Smoker (Mean±SD)	Median (Q1–Q3)	*p*
ΔBMI	12.54±2.56	12.2 (10.8–13.6)	12.46±2.16	12.5 (10.65–13.55)	0.887
ΔQTc	5.79±42.21	9 (−14–30)	7.72±22.81	3 (−10–24.5)	0.922
ΔQTd	0.21±7.33	2 (-4–6)	3.63±7.11	4 (−2–8)	0.059
ΔTp-e	0.03±9.77	−2 (−4–4)	0.41±8.77	2 (−4–4)	0.994
ΔTp-ed	−3.59±12.38	0 (−8–4)	2.75±9.43	3 (−4–7)	**0.036***
ΔTp-e/QT	0.02±0.07	0 (−0.02–0.03)	0.02±0.03	0.01 (−0.01–0.03)	0.254
ΔJTc	6.29±41.5	4.0 (−11.5–26.5)	7.53±20.5	5.5 (−9.3–22.0)	0.375
ΔJTd	0±6.2	2 (−4–4)	0.97±4.37	2 (−2–4)	0.398
ΔQRSd	−0.49±7.88	−1 (−4–2)	0.19±8.17	2 (−4–4)	0.246
ΔQRS dispersion	−0.89±3.87	−2 (−3–2)	0.97±3.51	2 (−2.5–3.5)	**0.031***
QRSd/QT	0.01±0.03	0.01 (−0.01–0.02)	0.01±0.02	0.01 (−0.01–0.03)	0.274
ΔTp-e/QRSd	0.01±0.11	0.01 (−0.06–0.05)	0.01±0.11	0 (−0.04–0.04)	0.856

**p*: Statistical significance ($p<0.05$, Mann–Whitney U test); Δ was calculated as pre-operative value – post-operative value. Positive Δ indicates a decrease in post-operative values, whereas negative Δ indicates an increase. All time-based ECG measurements were recorded in milliseconds. BMI: Body mass index, QTc: Corrected QT interval, QTd: QT dispersion, QT: QT interval, JTc: Corrected JT interval, JTd: JT dispersion, Tp–e: Tpeak–Tend interval, Tp–ed: Tpeak–Tend dispersion, QRSd: QRS duration, QRS dispersion: Maximum-minimum QRS duration across 12 leads, SD: Standard deviation, Q1-Q3: Interquartile range.

adverse structural and electrophysiological remodeling and to the development of obesity-associated cardiomyopathy.[6] Prolonged and heterogeneous ventricular repolarization is a characteristic feature of obesity-related electrical abnormalities.[14] Accordingly, obesity-related myocardial changes have been clinically associated with delayed ventricular repolarization and increased repolarization dispersion on surface ECG.[15] Within this context, the present study examined post-operative changes in ventricular repolarization parameters following sleeve gastrectomy.

QTc is widely used as a heart rate-corrected measure of overall ventricular repolarization duration, whereas

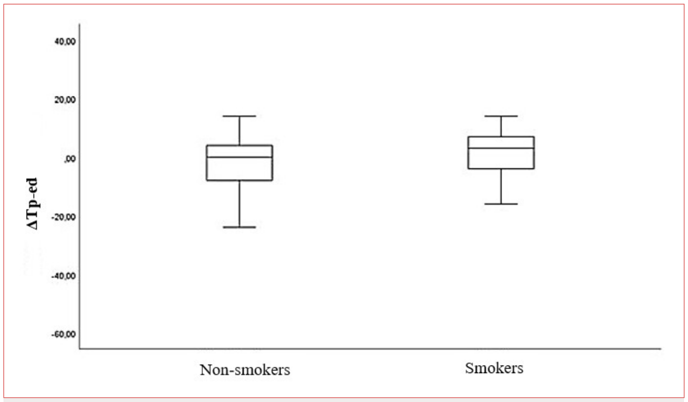

Figure 1. Comparison of ΔTp–ed values between smokers and non-smokers. Box-and-whisker plots illustrate the distribution of ΔTp–ed values by smoking status. Δ values were defined as the difference between pre-operative and post-operative measurements. Negative Δ values reflect an increase in post-operative Tp–ed, whereas positive Δ values indicate a post-operative reduction. A significant between-group difference was observed, with smokers showing a post-operative decrease in Tp–ed and non-smokers demonstrating a relative post-operative increase (p=0.036, Mann-Whitney U test). Tp–ed: Tpeak–Tend dispersion, Δ: Pre-operative minus post-operative.

Figure 2. Comparison of ΔQRS dispersion values between smokers and non-smokers. Box-and-whisker plots illustrate the distribution of ΔQRS dispersion values by smoking status. Δ values were defined as the difference between pre-operative and post-operative measurements. Negative Δ values reflect an increase in post-operative QRS dispersion, whereas positive Δ values indicate a post-operative reduction. A significant between-group difference was observed, with smokers showing a post-operative decrease in QRS dispersion and non-smokers demonstrating a relative post-operative increase (p=0.031, Mann-Whitney U test). QRS dispersion: Interlead dispersion of QRS duration, Δ: Pre-operative minus post-operative.

QTd was proposed as an electrocardiographic index reflecting interlead variability and spatial heterogeneity of ventricular repolarization.[17] However, QTc and QTd have been controversially affected across studies. According to the results of a study conducted by Alam et al.,[18] in 11 patients who underwent gastric banding or biliopancreatic diversion, QTc values showed no significant change despite weight loss during the 12-month post-operative follow-up. Similarly, Doherty et al.[19] compared the diet group with the control group in their study of 20 women who lost weight through dieting, but no significant change in QTc values was observed. Gupta et al.[20] demonstrated that weight loss achieved with a low-calorie liquid protein diet was associated with changes in QTd in obese patients. Al-Salameh et al.[21] found that 28 patients who had sleeve gastrectomy had significantly lower mean QTc values. However, there was no link discovered between QTc alteration and weight loss. Russo et al.[13] performed jejunoileal bypass on 100 obese patients, and QTc and QTcd values of the patients decreased significantly 1 year postoperatively. In a recent study by Gul et al.,[12] a non-significant increase in the QTc interval was observed in 48 patients who underwent sleeve gastrectomy, while a statistically significant decrease in QTcd was noted. In this study, patients were evaluated at 1 and 6 months after sleeve gastrectomy. In the present study, QTc values showed a modest but statistically significant reduction at 6 months after sleeve gastrectomy; however, mean QTc values remained within the generally accepted normal range both preoperatively and postoperatively (418.45±28.25 ms vs. 412.01±28.27 ms). This change was accompanied by a significant decrease in QTd, which also remained within physiological limits (53.93±10.95 ms vs. 52.57±10.20 ms). These findings indicate that weight loss achieved after sleeve gastrectomy shifts ventricular repolarization toward a shorter and more homogeneous timing pattern while remaining within accepted normal limits.

Both QTcd and JTcd serve as indicators of localized differences in ventricular recovery time and myocardial action potential duration.[13] Compared with QT-based indices, JT-related parameters have been proposed as more specific markers of ventricular repolarization, particularly when depolarization duration may influence QT measurements.[22] In the study by Russo et al.,[13] which included severely obese patients undergoing bariatric surgery, JTc values were reported to decrease significantly at 12 months postoperatively. Similarly, in the study by Gul et al.,[12] both JTc and JTcd showed significant reductions following bariatric surgery, suggesting a more homogeneous ventricular repolarization profile after sustained weight loss. In our cohort, JTc demonstrated a modest but statistically significant reduction at 6 months after

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sleeve gastrectomy, whereas JTd remained unchanged. This dissociation between changes in JTc and JTd may reflect earlier functional adaptation of global repolarization timing compared with indices reflecting spatial heterogeneity of ventricular repolarization, which may require longer follow-up to show measurable change.

QRSd reflects the temporal characteristics of ventricular depolarization on surface ECG.[23] In the overall study population, no significant change was observed in QRSd or QRS dispersion at 6 months after sleeve gastrectomy, whereas a modest decrease, but statistically significant in the QRSd/QT ratio was noted. This finding should be interpreted in conjunction with other electrocardiographic parameters, as ratio-based indices may reflect combined changes in depolarization and repolarization rather than isolated conduction alterations.

The Tp–e has been defined as an electrocardiographic parameter reflecting the terminal phase of ventricular repolarization and has been proposed as an index of transmural dispersion of myocardial repolarization.[24] In contrast, clinical surface electrocardiographic measurements suggest that the Tp–e may represent a more global feature of repolarization dispersion rather than a direct measure of transmural heterogeneity, and therefore should be interpreted as a surrogate electrophysiological marker.[25]

In this context, Inanır et al.[26] demonstrated that Tp–e and Tp–e/QTc were significantly prolonged in individuals with extreme obesity compared with healthy controls, suggesting an association between obesity and altered ventricular repolarization characteristics. Similarly, Gul et al.[12] reported significant reductions in Tp–e, Tp–e/QT, and Tp–e/QTc following marked weight loss after bariatric surgery, indicating favorable modulation of repolarization-related indices.

In our study, although no significant change was observed in absolute Tp–e or Tp–ed at 6 months after sleeve gastrectomy, a statistically significant reduction was detected in the Tp–e/QT ratio. This finding suggests that early post-operative electrophysiological adaptations may be more readily captured by ratio-based indices than by absolute time-based or dispersion measures. Consistent with this interpretation, Smetana et al.[27] emphasized that Tp–e measurements derived from surface ECG are influenced by interindividual anatomical and electrical variability and may therefore exhibit considerable person-to-person variation in clinical populations. By normalizing repolarization dispersion to overall repolarization time, the Tp–e/QT ratio may allow a more consistent assessment of repolarization dynamics on surface ECG.[28]

In our study, no significant differences were observed between genders in terms of weight loss or ventricular repolarization parameters at baseline or at 6 months postoperatively. Although the study population was predominantly female, this distribution is consistent with previous reports in bariatric surgery and weight-loss studies.[15,22]

Accumulating evidence indicates that smoking adversely affects myocardial electrical properties through mechanisms such as oxidative stress, inflammation, endothelial dysfunction, and fibrotic remodeling,[29,30] thereby contributing to increased regional heterogeneity of ventricular depolarization and repolarization. Despite these well-recognized adverse effects, the present study demonstrated a relatively greater reduction in Tp–ed (Fig. 1) and QRS dispersion (Fig. 2) among smokers than non-smokers after sleeve gastrectomy. In addition, ΔQTd showed a borderline between-group difference ($p=0.059$), suggesting that smoking status may exert a more pronounced influence on dispersion-based repolarization parameters, although this finding did not reach conventional statistical significance. This finding may suggest that smoking-related electrophysiological disturbances of the myocardium could regress more rapidly in the context of substantial weight loss. In addition, within our routine clinical practice, patients are strongly advised to quit smoking – particularly during the early post-operative period in conjunction with lifestyle and nutritional optimization – which may have resulted in reduced post-operative exposure to cigarette smoke. Such a reduction could theoretically contribute to autonomic and electrophysiological adaptation processes, thereby influencing post-operative ventricular repolarization parameters. Accordingly, smoking status should be considered not as a constant variable during the post-operative period but rather as a behavioral factor that may change over time. Importantly, evaluating changes in ventricular repolarization parameters according to smoking status was not among the primary aims of this study. Moreover, the number of patients within the smoking subgroups was relatively limited, and the study was not specifically powered to draw definitive conclusions based on smoking status alone, which further warrants a cautious interpretation of these findings. Nevertheless, when the overall pattern emerging from the analysis is considered, smoking status should be regarded as a potential confounding factor that may influence post-operative ventricular repolarization dynamics. Therefore, the changes observed in dispersion-based indices among smokers after weight loss should not be interpreted as definitive indicators of restored electrical homogeneity.

In summary, this study demonstrated that weight loss achieved after sleeve gastrectomy was associated with modest but statistically significant changes in certain ventricular repolarization parameters assessed by surface ECG. These changes occurred while all measured parameters remained within accepted physiological ranges and therefore appear to reflect subtle electrophysiological adaptations observed on surface ECG rather than overt abnormalities in ventricular conduction or repolarization. In particular, changes observed in ratio- and dispersion-based indices that jointly evaluate depolarization-repolarization timing may indicate a limited but measurable reorganization of myocardial electrical properties following substantial weight loss. Nevertheless, because these findings are derived from surface ECG measurements, interpretation of their clinical implications should be made with caution. Further prospective studies incorporating long-term follow-up and comprehensive assessments are needed to better clarify the electrophysiological changes observed after bariatric surgery.

Limitations

This study was conducted at a single tertiary center with a retrospective design, which may limit external generalizability. In addition, the 6-month follow-up period was relatively short to capture long-term electrophysiological remodeling or clinically relevant arrhythmic outcomes, and therefore, the findings should be interpreted as early post-operative adaptations rather than definitive long-term effects. Moreover, post-operative ECGs were obtained during routine follow-up visits, and not all patients were evaluated at an identical time point; minor variability around the 6-month assessment may have occurred, which could have influenced the magnitude of observed electrophysiological changes.

In addition, no data on clinical arrhythmic events or sudden cardiac death were available; therefore, all interpretations are restricted to electrocardiographic surrogate markers rather than clinical arrhythmic outcomes.

Autonomic nervous system activity was not directly assessed, and biochemical variables such as electrolyte balance, hormonal status, and inflammatory markers were not incorporated into the analysis, all of which may influence ventricular repolarization indices. Moreover, routine pre-operative echocardiography was not available for all patients due to the retrospective nature of the study. Although patients with documented structural heart disease or left ventricular hypertrophy were excluded when prior echocardiographic data were available, subclinical structural alterations cannot be completely ruled out and may have influenced surface ECG findings.

Electrocardiographic parameters were measured manually, which may introduce observer-related variability despite careful standardization. Although all measurements were independently performed by two blinded cardiologists and final values were obtained by averaging the measurements, formal interobserver reliability statistics, such as intraclass correlation coefficients, were not calculated.

The absence of a non-surgical or normal-weight control group precludes causal inference regarding the extent to which the observed electrophysiological changes can be attributed solely to weight loss, independent of other perioperative or lifestyle-related factors. In addition, post-operative behavioral variables, including changes in smoking habits, physical activity, and dietary adherence, were not prospectively quantified and may have acted as confounding factors influencing ventricular repolarization dynamics.

Future multicenter prospective studies with larger sample sizes, longer follow-up durations, standardized electrophysiological assessments, and multivariate models incorporating lifestyle, metabolic, autonomic, and imaging-based parameters are warranted to validate and extend these findings.

CONCLUSION

In this study, substantial weight loss achieved after sleeve gastrectomy was associated with modest but statistically significant changes in selected ventricular repolarization parameters assessed by surface ECG. Although all measured indices remained within generally accepted physiological ranges, post-operative reductions observed in QTc, QTd, JTc, Tp-e/QT, and QRSd/QT suggest subtle electrophysiological adaptations following weight loss rather than overt alterations in myocardial conduction or repolarization.

The present results support the concept that weight loss following sleeve gastrectomy may be accompanied by limited but measurable reorganization of myocardial electrical properties detectable on surface ECG. Exploratory observations suggest that behavioral factors may modulate post-operative electrophysiological changes, warranting further investigation. However, given the retrospective design, short follow-up duration, and reliance on surface electrocardiographic markers, the long-term clinical and arrhythmic implications of these electrophysiological changes remain uncertain. Larger, prospective studies with longer follow-up and comprehensive physiological assessments are required to further clarify the relationship between sleeve gastrectomy, weight loss, and ventricular repolarization dynamics.

DECLARATIONS

Ethics Committee Approval: The study was approved by the Gaziantep University Clinical Research Ethics Committee (Decision No: 2023/244, Date: 29.08.2023).

Informed Consent: Due to the retrospective nature of the study and the use of anonymized data, the requirement for written informed consent was waived by the institutional ethics committee.

Conflict of Interest: None declared.

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Authorship Contributions:

Author Contributions: Concept – LY, AA, MS; Design – LY, AA, MS; Supervision – LY, MS; Resources – LY; Materials – LY, AA; Data Collection and/or Processing – LY, AA, MS; Analysis and/or Interpretation – BKU, MS, VD, OB; Literature Review – BKU, LY; Writing – BKU, LY, MS, VD, AA, OB; Critical Review – BKU, MS, VD.

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