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ORIGINAL ARTICLE

Sleeve gastrectomy improves HDL function examined by Apo-A1 and atherogenic indices in non-diabetic obese patients

La gastrectomía en manga mejora la función HDL examinada por Apo-A1 e índices aterogénicos en pacientes obesos no diabéticos

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Abstract

Objective: *Dysregulation of lipid metabolism can be one of the pathophysiological mechanisms linking high-density lipoprotein cholesterol (HDL-C) dysfunction to obesity. The aim of the study is to show possible changes in lipid metabolism with atherogenic indices in obese patients after sleeve gastrectomy (SG) surgery.* **Method:** *Thirty patients who had SG surgery for obesity were included in the prospective study. The atherogenic risk indices were calculated pre-operatively, at 3 and 6 months post-operatively. Furthermore, serum paraoxonase-1 (PON-1), apolipoprotein-A1 (Apo-A1), and platelet-activating factor acetylhydrolase (PAF-AH) levels, amount of oxidized low-density lipoprotein (Ox-LDL) was measured.* **Results:** *We observed improvement in atherogenic risk indices and improved HDL-C functionality after SG. Increases were observed in HDL-C and HDL-C-related Apo-A1 levels 6 months after obesity surgery. Besides, the amount of serum triglycerides (TGs), PON-1 activity, and atherogenic risk indices decreased significantly within 6 months.* **Conclusion:** *As far as we know, there is no study in the literature examining the dynamic changes in SG and PON-1, PAF-AH, Apo-A1, and Ox-LDL parameters. This preliminary study dynamically detected improvement in HDL-C function and reduction in atherogenic risk indices after SG.*

Keywords: *Obesity. High-density lipoprotein cholesterol. Apo-A1. Atherogenic risk indices.*

Resumen

Objetivo: *La desregulación del metabolismo de los lípidos puede ser uno de los mecanismos fisiopatológicos que relacionan la disfunción del colesterol vinculado a lipoproteínas de alta densidad (c-HDL) con la obesidad. El objetivo del estudio es mostrar posibles cambios en el metabolismo de los lípidos con índices aterogénicos en pacientes obesos después de la cirugía de gastrectomía en manga (SG).* **Método:** *Los índices de riesgo aterogénico se calcularon en el pre-operatorio, y a los tres y seis meses del post-operatorio. Además, se midieron los niveles séricos de PON-1, apolipoproteína A1 (Apo-A1) y factor activador de acetilhidrolasa (PAF-AH), y la cantidad de lipoproteína de baja densidad oxidada (Ox-LDL).* **Resultados:** *Observamos una mejora en los índices de riesgo aterogénico y en la funcionalidad del c-HDL después de la SG, así como aumentos en los niveles de c-HDL y Apo-A1 relacionados con el c-HDL seis meses después de la cirugía de obesidad. Además, la cantidad de triglicéridos séricos, la actividad de PON-1 y los índices de riesgo aterogénico disminuyeron significativamente en seis meses.* **Conclusión:** *Hasta donde sabemos, no hay ningún estudio en la literatura que examine los cambios dinámicos en los parámetros SG y PON-1, PAF-AH, Apo-A1 y Ox-LDL. Este estudio preliminar detectó dinámicamente una mejora en la función del c-HDL y una reducción en los índices de riesgo aterogénico después de SG.*

Palabras clave: *Obesidad. Colesterol vinculado a lipoproteínas de alta densidad. Apo-A1. Índices de riesgo aterogénico.*

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Introduction

Obesity has serious comorbidities that impair the quality of life and shorten the life span[1]. Today, obesity is accepted as a multifactorial chronic disease characterized by inflammation and dyslipidemia[2]. Obesity, which has become an epidemic today, can actually be positively affected by even small changes in body weight and living habits[3]. Sleeve gastrectomy (SG) surgery can reduce the incidence of obesity-related cardiovascular events in addition to weight loss in patients[4,5]. Some recent studies have demonstrated the effects of SG on atherosclerosis and vascular endothelial functions[5,6]. The functionality of high-density lipoprotein cholesterol (HDL-C) rather than its amount that can be measured in blood is very important in understanding the mechanism of recovery of patients with obesity and bariatric surgery. Numerous studies in the literature have shown that HDL-C functions are impaired by obesity[7,8].

HDL-C is a highly heterogeneous particle. Most of the more than 200 proteins in the HDL-C structure can be associated with obesity. HDL-C is a lipoprotein with antiatherogenic properties as well as anti-inflammatory and many other properties. The main function of HDL-C is to remove excess cholesterol from peripheral tissues and transport it to the liver for metabolism and excretion. In fact, HDL-C functions as an anti-infective, athero-protective, anti-apoptotic and anti-thrombotic agent, and reverses cholesterol flow[9,10]. Normal functional HDL-C has a high level of antioxidant activity, enzymes, and anti-inflammatory activity. Atherosclerotic lesions begin with the accumulation of oxidized low-density lipoprotein (Ox-LDL) in macrophages in the artery wall. Antioxidant molecules in the HDL-C structure, mainly paraoxonase-1 (PON-1), can prevent Ox-LDL oxidation in the arterial wall. Apolipoprotein A-1 (Apo-A1) is the main structural protein of HDL-C and makes up 70% of its proteins. Numerous studies have also shown that Apo-A1 is a powerful antioxidant and anti-inflammatory molecule[9-11].

Today, predictive index calculations such as the atherogenic index of plasma (AIP), atherogenic index (AI), and lipoprotein combined index (LPCI) have been developed to predict the risk of future cardiovascular disease (CVD). However, studies using the CVD risk predictive index in obese patients undergoing SG are few in the literature. In one study, a significantly higher AIP was reported in subjects with abdominal obesity than in subjects without abdominal obesity[12]. In another study, a higher AIP level was positively and strongly associated with obesity. In addition, the AIP index can decrease rapidly and significantly with obesity surgery[13,14].

However, there is no study yet showing the effect of SG surgery on functional molecules in the HDL-C structure, such as platelet-activating factor acetylhydrolase (PAF-AH), PON-1, or Apo-A1, which can show antiatherogenic activity. In addition, the possible relationship between the calculated atherogenic risk indices (AIP, AI, and LPCI) and SG surgery is not yet known. This preliminary study may contribute to the improvement of HDL-C functionality and understanding the possible antiatherogenic effects of SG surgery.

Method

Patients

Thirty consecutive patients aged 18-65 years, without diabetes or lipid disorders and diagnosed with morbid obesity were included in this study. All patients were evaluated by a multidisciplinary team (general surgeon, anesthesiologist, psychiatrist, and endocrinologist) for obesity-related comorbidities such as body mass index (BMI) $\geq$ 40 or BMI $\geq$ 35 and type II diabetes mellitus and hypertension.

Method

Blood samples from patients included in the study group were taken into vacuum-sealed, yellow-capped, gel biochemistry tubes between 08:00 and 09:30, following a 12-h night fast just before surgery. The blood samples were centrifuged at 4000 rpm for 10 min within half an hour, and their serum was separated. Hemolyzed serum samples were excluded from the study. Routine biochemistry measurements, glucose, triglycerides (TGs), total cholesterol (TC), HDL-C, and low-density lipoprotein cholesterol (LDL-C) were made without waiting. Serum samples were transferred to plastic-capped Eppendorf tubes and stored at −80°C until the day they were analyzed. The same procedures were repeated in post-operative patients at 3 and 6 months after surgery. Apo-A1, Ox-LDL, PON-1, and PAF-AH were analyzed from the stored samples. Beckman AU 5800® (Beckman Coulter Diagnostics, California, USA) for glucose, TG, TC, HDL-C, LDL-C parameters biochemistry auto analyzer and commercial diagnostic reagent kits (Beckman Coulter Inc, California, USA) of the same brand were used.

Quantitative enzyme-linked immunosorbent assay (ELISA) for Apo-A1, Ox-LDL, PON-1, and PAF-AH (Lp-PLA2) measurements YL Biont® (Shangai YL Biotech Co., Ltd., Shanghai, China) brand manual diagnostic reagent kits were used with the immunoanalytical method, ELISA. At the end of the experiment, optical density measurement was performed with an ELX 800 BioTek (BioTek®, ELX 800, San Francisco, USA) ELISA reader.

The following non-traditional atherogenic indices were calculated in the present study: AIP, AI, and LPCI. The AIP is calculated as the logarithmic transformation of the ratio of the TG level to HDL-C level and the formula is AIP = log10 (TG/HDL-C). The AIP indicates the risk of atherosclerosis according to the values obtained: an AIP of −0.3-0.1 indicates low risk, an AIP of 0.1-0.24 indicates medium risk, and > 0.24 indicates high risk[15,16]. AI is defined as the ratio of non-HDL-C level to HDL-C level and calculated using the formula AI = non-HDL-C/HDL-C. LPCI is calculated using the formula LPCI = (TC × TG × LDL)/HDL-C.

Statistical analysis

Statistical analysis of the data was performed using the MedCalc® Version 19.3 software. The compliance of the data to a normal distribution was evaluated with the Kolmogorov-Smirnov test. Data conforming to a normal distribution were expressed as median and interquartile range (IR), and data not conforming to a normal distribution were expressed as median and IR values. Since the number of subjects in both groups was < 30, a non-parametric test was used. The Wilcoxon test was used for countable data. Pearson's correlation test and regression analysis were used for correlation analysis. The value of $p < 0.05$ was considered statistically significant.

Results

The demographic characteristics of the patients are shown in table 1. The mean blood TG levels of the patients were 151 mg/dL pre-operatively (IR: 104.25-199) and 120 mg/dL (IR: 102.25-160.5) post-operatively (3th month) (p = 0.034). The blood TG values of the patients at the 6th post-operative month were 99 mg/dL (IR: 84.2-129.75) and were significantly lower than the pre-operative values (p < 0.001). As a result, the blood TG levels of the patients after SG showed a statistically significant decrease from the 3rd month, as shown in table 2.

Table 1. General characteristics of the patients and comorbidities

General characteristics	Median, range
Age (in years)	37.05 (32.42-41.08)*
Sex (Female/Male) (%, n)	83.33% (25) / 16.67% (5)
Weight before surgery (kg)	121.52 (110.9-124.2)*
BMI (kg/m^2)	44.27 (41-45.8)*
Comorbidities	**(%) n**
Type 2 diabetes	23.33%, 7
Hypertension	16.67%, 5
Asthma	13.33%, 4
Migraine	6.67%, 2
Sleep apnea syndrome	3.33%, 1

Median and interquartile range. BMI: body mass index.

The pre-operative TC levels of the patients were within normal limits (188 mg/dL, IR: 159.5-212.5). Unlike TG values, TC values were 203 mg/dL (IR: 168.25-223.25) and 204 mg/dL (IR: 175.5-241.5) at the 3rd and 6th months post-operatively, respectively, yet this slight increase was not statistically significant. The blood HDL-C level of the patients increased from 40 mg/dL pre-operatively (IR: 36.5-47.25) to 44 mg/dL (IR: 39.75-48) at the 3rd post-operative month (p = 0.091). The increase in HDL-C continued 6 months post-operatively, and HDL-C levels were 48 mg/dL (IR: 45.75-51.5, p < 0.001 and p = 0.004) (Table 2).

Contrary to what was expected in pre-operative patients, the serum LDL-C level increased from 121 mg/dL (IR: 97.56-137) to 133 mg/dL (IR: 118.28-143) at the 3rd post-operative month. Moreover, this increase in LDL-C was statistically significant at 3 months (p = 0.008). At 6 months post-operatively, LDL-C increased to 134 mg/dL (IR: 118-163.25) and significantly increased compared to pre-operative values (p = 0.02). Although there was no statistically significant difference between the 3rd and 6th months (p = 0.1) (Table 2), the slight increase continued. Apo-A1 blood values, which are abundant in the HDL-C structure in pre-operative patients, averaged 2.28 mg/mL (IR: 1.97-3.47). This increase in Apo-A1 level was not statistically significant at 2.59 mg/mL (IR: 2.08-3.07) at the 3rd post-operative month (p = 0.47). However, the mean blood Apo-A1 level was found to be 2.61 mg/mL at 6 months after SG (IR: 2.31-3.04).

Table 2. Pre- and post-operative 3rd and 6th months statistical data of TG, TC, HDL-C, and LDL-C

Parameter (mg/dL)	Pre-operative			Post-operative 3rd month			Post-operative 6th month			p*		
	n	Median	IR	n	Median	IR	n	Median	IR	Pre-operative-post-operative 3rd month	Pre-operative-post-operative 6th month	Between post-operative 3rd and 6th months
TG	24	151	104.25-199	27	120	102.25-160.5	22	99	84.2-129.75	0.034	0.00046	0.027
TC	21	188	159.5-212.5	21	203	168.25-223.25	21	204	175.5-241.5	0.082	0.14	0.11
HDL-C	24	40	36.5-47.25	27	44	39.75-48	22	48	45.75-51.5	0.091	0.0007	0.0042
LDL-C	24	121	97.56-137	27	133	118.28-143	22	134	118-163.25	0.0083	0.02	0.1

*Wilcoxon test. TG: triglycerides; TC: total cholesterol; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; IR: interquartile range.

As a result, Apo-A1 levels increased at 6 months post-operatively compared to pre- and post-operative levels at 3 months (p = 0.028 and p = 0.05).

Apo-A1, Ox-LDL, PON-1, and PAF-AH values are shown in table 3. However, as shown in figure. 1, the Ox-LDL levels that we used as an oxidative stress (OS) marker in the blood at the 3rd post-operative month were 440.48 ng/L (IR: 400-659) in pre-operative patients, while the Ox-LDL level was 432.77 ng/L at the 3rd month. (IR: 382-517). This minimal decrease was not statistically significant (p = 0.46). However, the mean Ox-LDL level decreased to 426.98 ng/L (IR: 377-618) at 6 months post-operatively, but this decrease in Ox-LDL was not statistically significant compared to pre- and post-operative 3 months (Table 3 and Fig. 2).

The PON-1 enzyme level in the HDL-C structure showed a significant decrease compared to pre-operative levels at the 3rd and 6th months. For example, at the 3rd post-operative month, its level was 23.64 ng/mL (IR: 17.98-35.38) (p = 0.038). The decrease continued, and the mean PON-1 values decreased to 20.88 ng/mL (IR: 17.33-27.17) in the 6th post-operative month, and a significant decrease was found according to the pre-operative values (p = 0.05). According to the results, the blood PON-1 enzyme level decreased in pre-operative patients in the 3rd month, and the decrease slowed down in the 6th month and maintained the same level. Statistically, the amount of PON-1 did not show a significant change between the 3rd and 6th months post-operatively (p = 0.85). There was a strong statistically significant correlation between PON1, Ox-LDL, and Apo-A1. The amount of Ox-LDL and PON-1 enzyme in the correlation calculations is shown in table 4. The reduction in PON-1 is compensatory, perhaps due to the reduced need for Ox-LDL. As a result, the distribution of Ox-LDL and PON-1 values can be seen in figures 2 and 3. However, there was no statistically significant change in the antioxidant enzyme PAF-AH levels in the blood HDL-C structure after SG in the 6th month (p = 0.5).

One of the most important findings is the atherogenic risk indices that decreased with SG. As a result, the calculated atherogenic risk index values continued to decrease in pre-operative patients from the 3rd month and reached their lowest levels at the 6th month. In particular, AIP dropped from a high-risk level to a low-risk level.

Similarly, AI (p = 0.012) and LPCI (p = 0.042) levels decreased significantly post-operatively (Table 5).

Table 3. Changes in Apo-A1, Ox-LDL, PON-1, and PAF-AH pre-operative and 3rd and 6th months post-operative

Parameter	Pre-operative			Post-operative 3rd month			Post-operative 6th month			p*		
	n	Median	IR	n	Median	IR	n	Median	IR	Pre-operative-Post-operative 3rd month	Pre-operative-Post-operative 6th month	Between post-operative 3rd and 6th months
Apo-A1 (mg/mL)	30	2.28	1.97-3.47	30	2.59	2.08-3.07	30	2.61	2.31-3.04	0.47	0.028	0.05
Ox-LDL (ng/L)	30	440.48	400-659	30	432.77	382-517	29	426.98	377-618	0.46	0.13	0.58
PON-1 (ng/mL)	29	23.64	17.98-35.38	29	21.69	19.09-27.59	30	20.88	17.33-27.17	0.038	0.05	0.85
PAF-AH (ng/mL)	30	5.25	3.78-7.42	30	5.06	3.29-6.25	30	5.19	3.1-6.89	0.092	0.5	0.87

*Wilcoxon test. Ox-LDL: oxidized low-density lipoprotein; PON-1: Paraoxonase-1, PAF-AH: Platelet-activating factor acetylhydrolase, IR: interquartile range; Apo-A1: Apolipoprotein A-1.

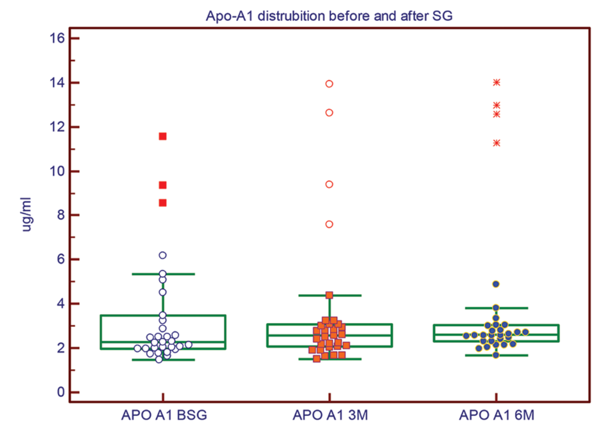

Figure 1. *Apolipoprotein A-1 distribution by pre- and post-operative 3rd and 6th months. In the box-and-whisker plot, the central box represents the values from the lower to upper quartile (25-75 percentile). The middle line represents the median. The horizontal line extends from the minimum to the maximum value, excluding outside and far-out values, which are displayed as separate points.*

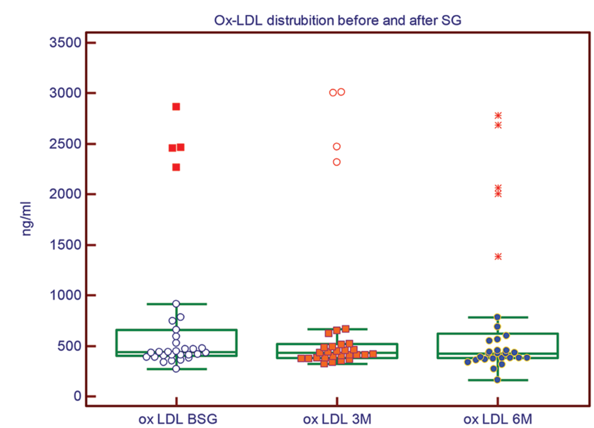

Figure 2. *Oxidized low-density lipoprotein distribution by pre- and post-operative 3rd and 6th month. In the box and whisker plot, the central box represents the values from the lower to upper quartile (25-75 percentile). The middle line represents the median. The horizontal line extends from the minimum to the maximum value, excluding outside and far-out values, which are displayed as separate points.*

Discussion

The main findings of this study were that HDL-C and Apo A1, which are indicators of HDL-C function, increased significantly after SG, and patients had a better lipid profile than pre-operative values. Furthermore, there was a strong correlation between Ox LDL and PON-1 as a result of reduced OS. Furthermore, new atherogenic risk indices AIP, AI, and LPCI, which were not included in the literature for SG and were shown comprehensively for the 1st time in the current study,

Table 4. Correlation of Ox-LDL and the amount of PON-1 enzyme

n	Ox-LDL pre-operative	Ox-LDL post-operative 3rd month	Ox-LDL post-operative 6th month
PON-1 pre-operative			
Correlation coefficient	0.932		
Significance level (p)	< 0.0001		
n	29		
PON-1 Post-operative 3rd month			
Correlation coefficient		0.895	
Significance level (p)		< 0.0001	
n		29	
PON-1 Post-operative 6th month			
Correlation coefficient			0.925
Significance level (p)			< 0.0001
n			29

Ox-LDL: oxidized low-density lipoprotein; PON-1: paraoxonase-1.

Table 5. Statistical improvement of AIP, AI, and LPCI

Parameters	Pre-operative	Post-operative 3rd month	Post-operative 6th month
non-HDL	3.86	4.66	4.17
AIP	0.22 (High risk)	0.1 (Low risk)	< 0.1 (Low risk)
AI	3.64	3.68	3.31*
LCPI	25.42	21.01	19.20**

*p = 0.012. **p = 0.042. AIP: atherogenic index of plasma (low risk < 0.1, intermediate risk 0.1-0.21, high risk > 0.21); AI: atherogenic index; LPCI: lipoprotein combined index. All values calculated mmoL/L.

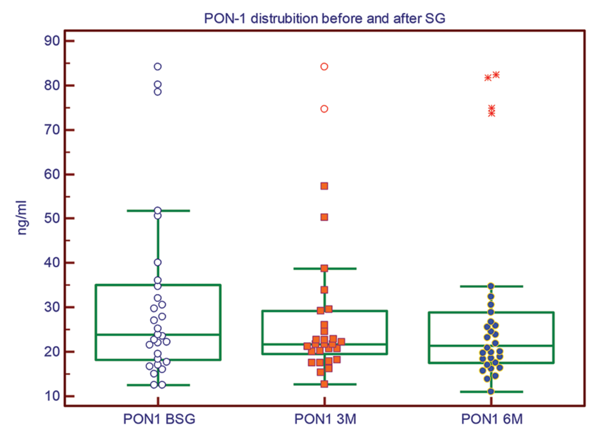

Figure 3. *Paraoxonase-1 enzyme distribution by pre- and post-operative 3rd and 6th months. In the box-and-whisker plot, the central box represents the values from the lower to upper quartile (25-75 percentile). The middle line represents the median. The horizontal line extends from the minimum to the maximum value, excluding outside and far-out values, which are displayed as separate points.*

decreased after SG surgery. Thus, the significant improvement in atherogenic risk indices after SG surgery may demonstrate the strong corrective effect of SG on lipid metabolism in patients with obesity. In the 6th month after SG, the AIP value of the patients decreased from the risk level to the lower risk level. The patients in the pre-operative group had a high risk of AIP, such as 0.22. In addition, the AI value and especially the LPCI values decreased significantly at the end of the 6th month. This decrease in atherogenic risk indices is actually an indication that HDL-C functions have improved significantly.

Today, every patient with morbid obesity should be examined for lipid metabolism. Increased TGs, mixed dyslipidemia associated with LDL-C and Ox-LDL, and increased atherogenic risk should be expected in obesity[15]. Increased adipose tissue in obese individuals contributes to low-grade inflammation and OS[3,16]. Significantly decreased TG levels and increased HDL-C and Apo-A1 levels after SG surgery determined in this study are perhaps the most important reasons for the decrease in atherogenic risk indices. In this study, the most important contribution to HDL-C functions after SG was the significant increase in the amount of Apo-A1 after 6 months.

Conversely, SG surgery can cause a clear reduction in chronic inflammation and atherogenic risk indices by decreasing plasma oxidative markers, including Ox-LDL and PAF-AH enzyme activity, as shown in this study. Our results show that the proatherogenic lipid profile and increased atherogenic risk indices (AIP, AI, and LCPI) characteristic of morbid obesity patients improve after SG[12-14].

In general, an increase in HDL-C was observed in the 3rd month after SG. There was also a significant decrease in TG levels, which reduced dyslipidemia. However, there was no statistically significant change in TC and Ox-LDL after SG surgery. Increasing systemic bile acid levels and changing bile acid composition after SG can affect lipid absorption, but the molecular mechanism is not understood. In addition to the surfactant role of bile after SG, bile acids also function as signaling molecules for a number of cellular nuclear receptors and plasma membrane receptors[17]. SG is a bariatric surgery technique that preserves the small intestine (especially the jejunum), so it may cause a significant increase in HDL-C[4,18].

In our study, we did not find any statistically significant changes in Ox-LDL and PAH enzyme levels in the blood for up to the 6th month. However, the Ox-LDL levels in the blood of patients with SG did not change significantly. In fact, this result should be expected, since the enzyme PAF-AH in the blood is mainly contained in LDL-C content of more than 80%, but < 20% is associated with HDL-C. Decreased chronic inflammation and decreased production of OS in the blood after SG can contribute to the stabilization of the levels of Ox-LDL and PAF-AH in the blood[19].

According to the literature, HDL-C levels increase by 47% compared to pre-operative SG values 10 years after SG surgery. In our study, the follow-up period after SG was up to 6 months, but even in this short time, the HDL-C ratio increased by 29% compared to pre-operative values. Therefore, an increase in HDL-C values indicates a statistically significant improvement in patients with obesity[20,21]. PON-1, which falls in the blood after SG, is actually a multifunctional enzyme associated with HDL-C, contrary to what we expected. PON-1 exerts antiatherogenic effects by increasing cholesterol influx from macrophages to HDL with ATP binding cassette transporter 1. Studies have shown that increased PON-1 activity is associated with a reduction in the incidence of major cardiovascular events[9,22]. HDL-C's most important antiatherogenic effect is thought to occur through macrophage-cholesterol flow and the reverse cholesterol transport system[10]. In addition, HDL-C improves endothelial function by increasing nitric oxide production and acts as a protective agent against oxidation and inflammation with Apo-A1- activity[10,22]. The main antiatherogenic property of functional HDL-C is inhibition of LDL-C oxidation[9]. Apo-A1, lecithin-cholesterol acyltransferase,and PON-1 enzymes functionally inhibit LDL oxidation in the HDL-C structure[9,10,22]. In this study, the amount of PON-1 enzyme found in the blood of patients after SG decreased after the 3rd month. This enzyme, which acts as an antioxidant in the structure of HDL-C, can mainly function in preventing the formation of Ox-LDL in patients after SG, and there may be a compensatory decrease in the need for PON-1. In fact, a lower level of chronic inflammation, low atherogenic activity, and much less oxidant molecule production should be expected as a result of the reduction in active adipose tissue in patients after bariatric surgery[23,24]. The PON-1 enzyme, which acts as an antioxidant in the structure of HDL-C, mainly prevents the formation of Ox-LDL in patients after SG. Finally, the decrease in PON-1 needs after SG surgery may be due to the decrease in radicals of OS synthesis[25,26].

The response of each bariatric surgical procedure to obesity-related dyslipidemia is different. Previous studies have reported a significant decrease in TG and a significant increase in HDL-C and Apo-A1 in patients undergoing SG[26-29]. The improvement in insulin sensitivity and the effect of this improvement on lipoprotein lipase activity may be explained by the meaningful and effective decrease in TG levels after SG[26-29]. Another finding in our study is that although TC did not change significantly, LDL-C increased significantly from the early post-operative period to the 3rd month. Fortunately, SG surgery can balance the increase in LDL-C inflammation and OS by reducing oxidative and microvascular function markers in adipose tissue[30,31]. Finally, in this study, Ox-LDL levels did not change significantly immediately after surgery and remained without a significant increase at the 6-month follow-up. In one study, a significant increase in HDL-C and Apo-A1 and a decrease in LDL-C and Ox-LDL were observed after the other SG[30-32].

Conclusion

According to our study results, HDL-C levels were increased in patients who had undergone SG surgery. In addition to this numerical increase, the increase in Apo-A1, which is part of the HDL-C function, may be very significant. In addition, the decrease in atherogenic risk indices due to the decrease in Ox-LDL and adipose tissue and the improvement of HDL-C functions after SG may be significant for the anti-atherosclerotic effect of SG surgery and reduce the risk of stroke and cardiovascular events in patients over time. The insufficient number of studies showing the relationship of atherogenic risk indices with SG makes this study a preliminary study, being the first study to report a decrease in atherogenic risk indices after SG surgery and an increase in HDL-C and Apo-A1 serum levels at 3 and 6 months. It is also a preliminary study showing that patients' HDL-C function improved after SG and significantly reduced TGs and Ox-LDL in the blood. However, it has some limitations; it has a limited number of subjects and longer patient follow-up is not possible.

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Conflicts of interest

The authors declare no conflicts of interest.

Ethical considerations

Protection of human and animal. The authors declare that no experiments involving humans or animals were conducted for this research.

Confidentiality, informed consent, and ethical approval. The authors have obtained approval from the Ethics Committee for the analysis of routinely obtained and anonymized clinical data, so informed consent was not necessary. Relevant guidelines were followed.

Declaration on the use of artificial intelligence. The authors declare that no generative artificial intelligence was used in the writing of this manuscript.

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