European Review for Medical and Pharmacological Sciences 2023; 27: 10605-10611

One-year follow-up of the oxidative stress profile of patients after laparoscopic sleeve gastrectomy

S. ŞIMŞEK[1], B. ER URGANCI[1], S. YILMAZ[2], M.R. AYKOTA[2], T. SARI[3], İ. AÇIKBAŞ[1]

[1]Medical Biology Department, Faculty of Medicine, Pamukkale University, Denizli, Turkey
[2]General Surgery Department, Faculty of Medicine, Pamukkale University, Denizli, Turkey
[3]Infectious Diseases Department, Faculty of Medicine, Pamukkale University, Denizli, Turkey

Abstract. – OBJECTIVE: Obesity is a major risk factor for developing a number of serious diseases, such as cardiometabolic changes and cancer. An increase in adipose tissue and a decrease in antioxidant capacity both contribute to the etiopathogenesis of these comorbidities. The most effective method in the treatment of morbid obesity is bariatric surgery. Laparoscopic sleeve gastrectomy (LSG) is the most preferred method in bariatric surgery today. In this study, the potential improvement effect of laparoscopic sleeve gastrectomy (LSG) surgery in the restoration of weight loss and endocrine and tissue-based deterioration was obtained by evaluating changes in oxidative stress, antioxidant agents, and lipid oxidation levels.

PATIENTS AND METHODS: Fifty patients who had LSG surgery were chosen, along with 50 healthy volunteers who were the same age and gender as these patients. Serum total antioxidant capacity measurement, total oxidant capacity measurement, malondialdehyde (MDA) for the measurement of lipid peroxidation degree, and superoxide dismutase (SOD) and glutathione peroxidase (GPx) levels for the measurement of antioxidant levels were measured colorimetrically with the help of a commercial kit.

RESULTS: Oxidative stress indices, MDA levels, and GPx levels of patients with morbid obesity treated with LSG were observed to decrease significantly compared to the preoperative period, while no significant changes were observed in SOD levels.

CONCLUSIONS: In patients with morbidly obese conditions, the weight control achieved by sleeve gastrectomy, which is a restrictive method and thus causes a decrease in adipose tissue, causes a decrease in oxidative stress and an increase in the antioxidant response.

Key Words:
Laparoscopic sleeve gastrectomy, Oxidative stress, MDA, SOD, GPx.

Introduction

Obesity is one of the most important health problems of the 21st century. According to the World Health Organization[1], 1.9 billion adults worldwide are considered overweight, and more than 650 million adults are obese. It is predicted that by 2030, 2.16 billion people (38%) will be considered overweight, and 1.12 billion (10%) will be obese.

Obesity is an important risk factor for the development of many serious diseases, including cardiometabolic changes and cancer. Among these pathologies, 44% of diabetes, 23% of ischemic heart disease, 7-41% of cancers, and metabolic syndrome are counted as clinical complications of obesity. Treatment of obesity is more expensive and time-consuming than expected or desired, especially for patients with morbid obesity (BMI>40%). Bariatric surgery is offered as the treatment of choice because of its long-lasting outcome and low risk. For this purpose, different surgical techniques showing different efficacy have been developed[2]. Despite diet and exercise, individuals with a BMI of 40 kg/m^2 or a BMI of 35 kg/m^2 and those with comorbidities (such as diabetes, hypertension, dyslipidemia, cardiovascular disease, and respiratory diseases) can be treated with bariatric surgery[3]. Laparoscopic sleeve gastrectomy (LSG) is the most frequently preferred surgical method today[4].

There are some mechanisms to prevent cellular damage caused by oxidant molecules in the body. Antioxidants are substances that prevent the formation of free radicals, metabolize free radicals, or increase the scavenging of free radicals to prevent the oxidation of substances that can be oxidized, such as proteins, lipids,

Corresponding Author: B. Er Urgancı, Ph.D; e-mail: aber@pau.edu.tr

carbohydrates, and DNA in living cells. Normally, there is a balance between oxidants and antioxidants in our body, and it has a complex antioxidant system that fights endogenous or exogenous free radicals and related oxidative stress[5]. Antioxidants are divided into endogenous (enzymes and non-enzymes) and exogenous (external) sources. Antioxidants, which are endogenous enzymes, include superoxide dismutase (SOD), glutathione peroxidase (GPx), catalase (CAT), glutathione transferase (GST), glutathione reductase, and the mitochondrial oxidase system[6]. There are various studies[6-8] explaining the increase in oxidative stress observed in people with obesity. Abnormal formation of free oxygen radicals after meals, and changes in lipid and glucose metabolism are among the factors that play a role in the increase in oxidative stress[7].

In plasma, antioxidants interact with one another. In general, these compounds function as synergists. As a result of this interaction, the total antioxidant capacity is enhanced beyond the sum of the effects of the individual components. A decrease in one antioxidant can be compensated for by an increase in the other. Therefore, the measurement of total antioxidant capacity provides more valuable information than the measurement of individual antioxidants[8]. In cases where the total oxidant capacity (TOS) exceeds the total antioxidant capacity (TAS), oxidative stress and tissue damage develop.

Oxidative stress caused by ROS (reactive oxygen species) is one of the most important causes of a wide range of diseases because it changes metabolic homeostasis and controls cell growth and differentiation. So, the production of antioxidants to keep ROS from building up too much in cells and tissues has been linked to several diseases, such as cardiovascular disease and/or related metabolic disorders like dyslipidemia, diabetes, metabolic syndrome, and inflammatory processes. In many studies on obesity and oxidative stress, it has been found that the ratio of body fat to body mass index and the body mass index both go up with obesity[9].

In this study, we investigated the possible improvement effect of laparoscopic sleeve gastrectomy (LSG) operations in the correction of weight loss and endocrine and tissue-based deterioration by determining the changes in oxidative stress, antioxidant products, and lipid peroxidation levels on a cellular basis.

Patients and Methods

Sample Collection

This study was approved by the University Non-Invasive Clinical Research Committee under the number E-60116787-020-44670. In our study, we compared 50 LSG patients with the inclusion criteria of being between the ages of 18-65 and having a BMI between 35-45, with 50 healthy volunteers of the same age and gender who also did not have a comorbid disease. All patients and volunteers signed an informed consent to participate in this study. About 5-10 ml of peripheral vein blood was taken from patients who had LSG surgery before and 1 year after surgery and put into yellow-capped biochemistry tubes (BD, New Jersey, USA).

Serum Yield

To get the serum, blood from the patients and the control group was put in a biochemistry tube (BD, New Jersey, USA) and spun at 2,500 rpm for 10 minutes at +4°C. The obtained serums were stored at -80°C until the completion of the working samples.

Determination of TOS Levels

A commercial kit (Relassay, Gaziantep, Turkey) was used to determine the total oxidant capacity. The manufacturer's kit contains three chemicals: buffer, prochromogen, and standard. Basic steps:

1. 200 µl of buffer were added to each well of the 96-well plates.
2. Afterwards, 30 µl of the serum sample was added to it, and 30 µl of the standard was added to the other well. After 30 seconds, it was read at a wavelength of 660 nm with the help of an Eliza reader (Biotek, Vermont, ABD) (first reading: A1).
3. After reading, 10 µl of prochromogen was added to the mixture and mixed, and after 5 minutes, it was read again with Eliza plate reader at 660 nm wavelength (second reading: A2).
4. Then, using the first and second reading values (A1 and A2) obtained, the calculation was made in the Excel program according to the formulation determined by the manufacturer, and the TOS value was found.
5. The calculation is as follows: A2-A1= standard and sample (serum) ΔAbs Result = (ΔAbs Sample)/(ΔAbs Standard) * Standard Concentration.

Determination of TAS Levels

A commercial kit (Relassay, Gaziantep, Turkey) was used to determine the total antioxidant capacity. The kit contains three chemicals: buffer, prochromogen and standard. Basic steps:

1. 200 µl of buffer was added to each well of the 96-well plates.
2. Afterwards, 12 µl of serum sample was added to it, and 12 µl of standard was added to the other well. In addition, molecular-grade water (H_2O) was added to three wells. After 30 seconds, it was read at a wavelength of 660 nm with the help of an Eliza reader (first reading: A1).
3. After reading, 30 µl of prochromogen was added to the mixture and mixed, and after 5 minutes, it was read again with the Eliza Plate reader at 660 nm wavelength (second reading: A2).
4. Then, using the first and second reading values (A1 and A2) obtained, the calculation was made in the Excel program according to the formulation determined by the manufacturer, and the TAS value was found.
5. The calculation is as follows: A2-A1=standard and sample (serum) ΔAbs Result = (ΔAbs H2O) – (ΔAbs Sample) / (ΔAbs H2O) – (ΔAbs standard)

In addition, the oxidative stress index was determined using the formula "OSI = TOS/TAS*1/10".

Measurement of MDA, SOD, and GPx Levels

MDA for the measurement of lipid peroxidation degree and SOD and GPx levels for the measurement of antioxidant levels were measured colorimetrically with the help of a commercial kit (BT Lab, Shangai, China).

In this system based on the biotin double antibody (sandwich) principle, the wells are coated with specific antibodies. Standards were prepared as serial dilutions of 1/2, 1/4, 1/8, 1/16, and 1/32 from the 1x stock standard, respectively. 50 µl of standard and 50 µl of streptavidin-HRP were added to the standard well. 40 µl of sample + 10 µl of antibody + 50 µl of streptavidin-HRP were added to the sample wells. After 60 minutes of incubation at 37°C, washing was performed with the washing solution. First, 50 µl of chromogen solution A and 50 µl of chromogen solution B were added to the wells, respectively. After 10 minutes of incubation at 37°C in the dark, 50 µl of stop solution was added (blue color turned yellow). The color change and absorbances were determined at 450 nm wavelength.

Statistical Analysis

According to the power analysis we have done, it has been calculated that 80% power can be reached at the 95% confidence level when at least 41 patients are included in the study. Since comparisons were planned with the control group, the patient group was included in the same way as the number of people.

The data were analyzed with the SPSS 25.0 package program (IBM Corp., Armonk, NY, USA). Continuous variables are given as mean ± standard deviation, and categorical variables as numbers and percentages. The relationships between continuous variables were examined with Spearman's correlation analyses, and the differences between categorical variables were examined with the Chi-square analysis. $p<0.05$ was considered significant.

Results

When the oxidative stress index results between the groups were examined, it was observed that the stress indexes of the patients decreased significantly at the 1st year after bariatric surgery ($p=0.0081$). No significant difference was observed between the control group and the LSG surgery group ($p>0.05$) (Table I).

When the MDA levels between the groups were examined, it was observed that the MDA levels of the patients decreased significantly in the control after the 1st year from the bariatric

Table I. Oxidative stress indices of preoperative, postoperative 1st year follow-up and control groups.

Group	Mean	Standard deviation	Standard error	p
Preoperative	0.3891	0.2653	0.03829	> 0.05
Postoperative 1 year	0.2827	0.09314	0.01344	0.0081
Control	0.3188	0.1001	0.0143	

Table II. MDA levels of the preoperative, postoperative 1st year follow-up and control groups.

Group	Mean	Standard deviation	Standard error	p
Preoperative	6.32 nmol/ml	4.099	0.6044	< 0.001
Postoperative 1 year	1.712 nmol/ml	0.825	0.1273	< 0.001
Control	8.286 nmol/ml	5.177	0.7717	

surgery (p<0.0001). A significant difference was observed between the control group and the LSG surgery group (p<0.001) (Table II).

When the SOD levels between the groups were examined, there was no significant difference in the SOD levels of the patients in the 1st-year control after bariatric surgery (p=0.873). A significant difference was observed between the control group and the LSG surgery group (p<0.001) (Table III).

Considering the GPx levels between the groups, a significant difference was observed in the GPx levels of the patients after 1 year from the bariatric surgery (p<0.001). While a significant difference was observed between the control group and the preoperative GPx levels (p<0.001), no significant difference was observed in the postoperative 1st-year control (p=0.6996) (Table IV).

Discussion

In this study, patients with morbid obesity who were treated with LSG had significantly lower oxidative stress indexes, MDA levels, and GPx levels compared to before the surgery. However, SOD levels did not change in a significant way.

Obesity is one of the most important risk factors for systemic diseases like type 2 diabetes, coronary heart disease, and liver diseases[2]. This is shown by the chronic inflammatory process that happens when there is more fat tissue, more oxidative stress, and less antioxidant capacity. In many ways, obesity causes the body's oxidative load to rise, which makes antioxidant capacity less effective. Changing the balance between oxidative stress and antioxidant capacity in favor of the antioxidant system greatly reduces the risk of hyperlipidemia, diabetes, cardiovascular disease, and inflammatory diseases, which are all parts of the metabolic syndrome. Reducing fat mass in patients undergoing surgery improves not only their appearance but also their metabolism[7].

The main energy source in the body is white adipose tissue. White adipose tissue is a storage form, but the main mechanism controlling food intake is the hormone leptin secreted from adipocytes. White adipose tissue cells enlarge as a result of consuming an excessive number of calories. The increase in adipocyte size serves as a depot, with the formation of approximately 10% of new adipocytes annually[10]. The increase in size of adipocytes is characterized by an increase in

Table III. SOD levels of the preoperative, postoperative 1st year follow-up and control groups.

Group	Mean	Standard deviation	Standard error	p
Preoperative	85.5 pg/ml	15.85	2.445	< 0.001
Postoperative 1 year	68.18 pg/ml	18.41	2.841	0.873
Control	389.6 pg/ml	260.2	36.8	

Table IV. GPx levels of the preoperative, postoperative 1st year follow-up and control groups.

Group	Mean	Standard deviation	Standard error	p
Preoperative	92.52 µU/ml	3.669	0.573	< 0.001
Postoperative 1 year	66.44 µU/ml	18.2	2.573	0.6996
Control	69.04 µU/ml	19.65	2.778	

inflammatory cell infiltration[11]. Parameters such as excessive fat accumulation, increased cellular stress, metabolic dysfunction, inflammation, and hypoxia increase the oxidative load[12]. Chronic inflammation plays an important role in the development of obesity-related metabolic complications by impairing microvascular structures and vascular endothelial function in connection with insulin resistance[13]. Visceral fat deposition carries a greater risk than subcutaneous fat deposition for metabolic syndrome and insulin resistance because proinflammatory cytokines and mediators accumulate more in visceral adipose tissue[14].

Oxidative stress is the most important underlying cause of changes in the body in the case of chronic obesity[15]. Patients who are obese have dysfunctional adipose tissue because they make too many proinflammatory cytokines. This is because there is a link between oxidative stress and systemic inflammation. In many clinical studies[14,15], the end products of free radicals like malondialdehyde, lipid hydroxyperoxidase, F2-isoprostanese, and other biomarker measurements have shown that there is a positive correlation between obesity and oxidative stress. In some studies[16], it has been reported that there is a positive correlation between free radicals formed because of oxidative stress and BMI. On the contrary, there is an opposite relationship between body fat and antioxidant capacity. As a matter of fact, studies[17] have reported that reducing the amount of reactive oxygen species (ROS) improves steatohepatitis, dyslipidemia, and insulin resistance. Hypertrophic adipocytes, especially adipokine products, are the main source of ROS that impair white adipose tissue function[18]. Some studies[19] have reported that the increase in ROS causes the development of mitochondrial dysfunction and insulin resistance in relation to obesity. The high amount of ROS in adipocytes suppresses the anti-inflammatory, anti-atherogenic, and insulin-sensitivity properties of adiponectin. Therefore, it is understood that there is an inverse relationship between oxidative stress and adiponectin. Increased ROS levels in adipocytes result in increased proinflammatory cytokine (IL-6, PAI-1), macrophage chemoattractant factor-1 (MCP-1) levels, and decreased adiponectin levels. Increased adipose tissue causes obesity to progress to metabolic syndrome *via* M1 macrophage and T cell accumulation, as well as proinflammatory cytokines such as IL-1, TNF-, IL-17, and IL-6. Pro-inflammatory immune cells formed by lymphocytes, monocytes, macrophages, and natural killer cells with an increase in adipose tissue cause permanent and low-grade inflammation in patients with obesity[20].

Furukawa et al[21] analyzed lipid peroxidation and H_2O_2 production in adipose tissue in CCF mice with obesity to determine whether obesity-related oxidative stress is reduced. They reported that lipid peroxidation and H_2O_2 production were specifically increased in white adipose tissue, but not in the liver, muscle, or aorta. Aminooxidases, which are expressed as the main source of H_2O_{2n} in adipose tissue, are not thought to be very effective in the development of mitochondrial dysfunction and insulin resistance because their activities in white adipose tissue are very limited in individuals with obesity. While hydrogen peroxide inhibits lipolysis, it plays a role in insulin resistance by increasing lipid synthesis, glucose uptake, and glucose transport to the tissues.

The antioxidant system keeps free radicals from doing damage by using enzymes like superoxide dismutase (SOD), glutathione peroxidase (GPX), glutathione S-transferase (GST-A4), and catalase to get rid of ROS[22]. Curtis et al[23] wrote in a study that GST-A4 makes patients who are overweight less resistant to insulin. Carotenoids, antioxidants, and fat-soluble vitamins (like vitamins A, C, and E) are all stored in adipose tissue. Patients who are overweight have lower levels of glutathione, TAS, and lipophilic vitamins. On the other hand, they have higher levels of LDL and VLDL lipoproteins. It has been said[24] that lipid peroxidation is the cause of atherosclerosis in overweight patients who do not have diabetes.

In the study by Cabrera[25], in which the changes in oxidative stress after Roux-en-Y gastric bypass surgery (RYGB) were examined, oxidation markers in individuals with obesity were higher than those in the control group, and antioxidant defense indexes were lower. Although there was a significant decrease in oxidation markers 12 months after RYGB, they were still higher than the control group. A higher antioxidant defense was also observed in association with the decrease in inflammatory markers after surgery. These findings support the view that adipose tissue is a potential treatment target for the control and prevention of obesity-related pathologies.

According to Peng and Murr[26], RYGB may have a different effect on oxidative stress and total antioxidant capacity on metabolic activities in the liver than restrictive surgeries alone.

Cătoi[27] reported that while there was an in-

crease in NO level and a decrease in total antioxidant response in the 6th postoperative month in patients who underwent vertical band gastroplasty, at the end of the first year, there was a decrease in NO level and an increase in total antioxidant response.

Bawahab[28] reported that plasma activities of antioxidant markers are high in morbidly obese individuals and that the presence of chronic inflammation and oxidative stress in obesity will result in an increase in superoxide radicals and a decrease in SOD activity as an adaptive response. In the study, the weight loss achieved by sleeve gastrectomy decreased the stimulating effect on SOD, GPx, GST, and vitamin C by inhibiting the production of ROS.

In our study, we showed that sleeve gastrectomy, which is a restrictive method, reduced the oxidative stress index and GPx levels in morbidly obese individuals at the end of the 1st year.

Antioxidant molecules that contribute to the scavenging process of free oxygen radicals in plasma can be endogenous (such as uric acid, albumin, and thiol compounds) or exogenous (such as ascorbic acid and vitamin E). Therefore, consumption of foods rich in antioxidants (olive oil, vegetables, fruits, tea, wine, etc.) may cause an increase in plasma antioxidant capacity. The sum of endogenous and exogenous antioxidant compounds constitutes the total antioxidant activity in the extracellular fluid. Plasma levels of antioxidants may decrease due to their consumption during acute oxidative stress. Cooperation between different antioxidants provides stronger protection against attack by ROS than a single compound. Therefore, TAS may provide more useful biological information than the measurement of each individual antioxidant parameter to evaluate the cumulative effect of all antioxidants in plasma and body fluids[29].

Conclusions

In this study, we aimed to determine the oxidative stress indices by evaluating the TOS and TAS, as well as measuring the compounds with oxidative and antioxidant properties separately to measure the antioxidant system and oxidative stress.

As a result, in individuals with morbid obesity, weight control achieved by sleeve gastrectomy, which is a restrictive method and thus causes a decrease in adipose tissue, causes a decrease in oxidative stress, and an increase in the antioxidant response. The increase in antioxidant response is likely to be due to decreased consumption of antioxidant compounds due to reduced oxidative stress.

Conflict of Interest

The authors have no relevant financial or non-financial interests to disclose.

Funding

This study was supported by the Scientific Research Coordination Unit of Pamukkale University, under project No. 2019BSP024.

Authors' Contribution

SŞ, SY, MRA, TS, and IA designed the experiments. SŞ and BEU performed data analysis. SŞ and BEU wrote the manuscript.

Ethics Approval

This study was approved by the University Non-Invasive Clinical Research Committee under the number E-60116787-020-44670.

Informed Consent

All patients and volunteers signed informed consent to participate in this study.

ORCID ID

Selda Şimşek: 0000-0002-1585-7379
Buket Er Urgancı: 0000-0002-5339-3835
Sevda Yılmaz: 0000-0002-1309-0805
Muhammed Raşid Aykota: 0000-0003-1862-6186
Tuğba Sarı: 0000-0002-9902-9879
İbrahim Açıkbaş: 0000-0001-7483-1147

References

1) WHO. Obesity and Overweight, 2021. Available at: https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight.
2) Colquitt JL, Pickett K, Loveman E, Frampton GK. Surgery for weight loss in adults. Cochrane Database syst rev 2014; 8: Cd003641.
3) Karnak İ. Obezite tedavisinde cerrahinin yeri. Katkı Pediatri Dergisi 2000; 21: 554-573.
4) Sista F, Carandina S, Andreica A, Zulian V, Pietroletti R, Cappelli S, Balla A, Nedelcu M, Clementi M. Long-term results of laparoscopic gastric sleeve: the importance of follow-up adherence.

Eur Rev Med Pharmacol Sci 2022; 26: 6691-6699.

5) Halliwell B. Free radicals, antioxidants, and human disease curiosity, cause, or consequence? Lancet 1994; 344: 721-725.
6) Neeraj PJ, Sing S, Singh J. Role of free radicals and antioxidants in human health and disease. Int J Curr Res Rev 2013; 5: 14-22.
7) Fejfer K, Buczko P, Niczyporuk M, Ładny JR, Hady HR, Knaś M, Waszkiel D, Klimiuk A, Zalewska A, Maciejczyk M. Oxidative Modification of Biomolecules in the Nonstimulated and Stimulated Saliva of Patients with Morbid Obesity Treated with Bariatric Surgery. Biomed Res Int 2017; 4923769.
8) Koracevic D, Koracevic G, Djordjevic V, Andrejevic S, Cosic V Method for the measurement of antioxidant activity in human fluids. J Clin Pathol 2014; 54: 356-361.
9) Kahraman FU, Torun E, Osmanoğlu NK, Oruçlu S, Özer ÖF. Serum oxidative stress parameters and paraoxonase-1 in children and adolescent sex posed to passive smoking. Pediatr Int 2017; 59: 68-73.
10) Spalding KL, Arner E, Westermark PO. Dynamics of fat cell turnover in humans. Nature 2008; 453: 783-787.
11) Weisberg SP, McCannD, DesaiM, Rosenbaum M, Leibel RL, Ferrante AW. Obesity is associated with macrophage accumulation in adipose tissue. J Clin Inves 2003; 112: 1796-1808.
12) Qatanani M, LazarMA. Mechanisms of obesity-associated insulin resistance: many choices on themenu. Genes Dev 2007; 21: 1443-1455.
13) Gregor MF, Hotamisligil GS. Inflammatory mechanisms in obesity. Annual Rev Immunol 2011; 29: 415-445.
14) Choe SS, Huh JY, Hwang IJ, Kim JI, Kim JB. Adipose Tissue Remodeling: Its Role in Energy Metabolism and Metabolic Disorders. Front Endocrinol (Lausanne) 2016; 7: 30
15) Roberts CK, Sindhu KK. Oxidative stres and metabolic syndrome. Life Sciences 2009; 84: 705-712.
16) Sankhla M, Sharma TK, Mathur K. Relationship of oxidative stress with obesity and its role in obesity induced metabolic syndrome. Clin Lab 2012; 58: 385-392.
17) Houstis N, Rosen ED, Lander ES. Reactive oxygen species have a causal role in multiple forms of insulin resistance. Nature 2006; 440: 944-948.
18) DePauw A, Tejerina S, Raes M, Keijer JT. Mitochondrial (dys)function in adipocyte (de) differentiation and systemic metabolic alterations. Am J Pathol 2009; 175: 927-939.
19) Fujita K, Nishizawa H, Funahashi T, Shimomura I, Shimabukuro M. Systemic oxidative stress is associated with visceral fat accumulation and the metabolic syndrome. Circ J 2006; 70: 1437-1442.
20) Osborn O, Olefsky JM. The cellular and signaling networks linking the immune system and metabolism in disease. Nat Med 2012; 18: 363-337.
21) Furukawa S, Fujita T, Shimabukuro M. Increased oxidative stress in obesity and its impact on metabolic syndrome. J Clin Invest 2004; 114: 1752-1761.
22) Galinier A, Carrière A, Fernandez Y. Adipose tissue proadipogenic redox changes in obesity. J Biol Chem 2006; 281: 12682-12687.
23) Curtis JM, Grimsrud PA, Wright WS. Down regulation of adipose glutathione S-tansferaseA4 leads to increased protein carbonylation, oxidative stress, and mitochondrial dysfunction. Diabetes 2010; 59: 1132-1142.
24) Landrier JF, Marcotorchino J, Tourniaire F. Lipophilic micronutrients and adipose tissue biology. Nutrients 2012; 4: 1622-1649.
25) Cabrera E. Reduction in Plasma Levels of Inflammatory and Oxidative Stress Indicators After Roux-En-Y Gastric Bypass. Obes Surgery 2010; 20; 42-49.
26) Peng Y, Murr MM. Roux –en-Y gastric bypass improves hepatic mitochondrial function in obese rats. Surg Obes Relat Dis 2013; 9: 429-435.
27) Cătoi A. NitricOxide, Oxidant Status and Antioxidant Response in Morbidly Obese Patients: the Impact of 1-Year Surgical Weight Loss. Obes Surgery 2013; 23: 1858-1863.
28) Bawahab M. Effects of Weight Reduction After Sleeve Gastrectomy on Metabolic Variables in Saudi Obese Subjects in Aseer Province of Kingdom of Saudi Arabia. Obes Surgery 2017; 27: 2005-2014.
29) Serafini M, Del Rio D. Understanding the association between dietary antioxidants, redox status and disease: is the Total Antioxidant Capacity the right tool? Redox Rep 2004; 9: 145-152.