Comparison of preoperative and postoperative serum thiol/disulfide levels in patients undergoing laparoscopic sleeve gastrectomy for morbid obesity

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Abstract. – OBJECTIVE: Obesity, a prevalent chronic disease, results from an imbalance between energy intake and expenditure. The oxidative stress associated with obesity stems from an imbalance between reactive oxygen species and the cell's antioxidant defense system. Oxidative stress can cause many diseases. The assessment of thiol/disulfide balance, a biochemical test, can be used to detect oxidative stress. The aim of this study is to determine the changes in oxidative stress associated with obesity after obesity surgery by assessing the thiol/disulfide levels.

PATIENTS AND METHODS: The study was conducted with 40 volunteer patients with a body mass index (BMI) above 40 who underwent obesity surgery at Harran University Hospital General Surgery. Thiol and disulfide levels and other blood parameters were measured from the preoperative and postoperative 2nd and 6th-month blood samples of the patients. BMI was calculated by recording the weights and heights of the cases. Patients with diseases that could affect oxidative stress measurements and those using medication were excluded from the study, and the analyses were performed accordingly.

RESULTS: The results showed a statistically significant decrease in native thiol, disulfide, reduced thiol, oxidized thiol, glucose, ALT (alanine aminotransferase), ALP (alkaline phosphatase), total cholesterol, HDL (high-density lipoprotein), triglyceride, and BMI values between the preoperative, 2-month postoperative, and 6-month postoperative measurements ($p<0.05$).

CONCLUSIONS: Restrictive methods such as sleeve gastrectomy in individuals with morbid obesity led to weight control and a decrease in adipose tissue, reducing oxidative stress and increasing antioxidant response.

Key Words:
Obesity, Oxidative stress, Surgery.

Introduction

Obesity is a health condition characterized by excessive fat accumulation in the body. Obesity means that a person's weight is above the accepted healthy ranges for their age, gender, and general health. Obesity can lead to some complications with adverse health effects and increase the risk of serious diseases, such as heart disease, diabetes, high blood pressure, respiratory problems, joint conditions, and some types of cancer[1,2].

The increase in reactive oxygen species (ROS) can directly or indirectly cause damage in different organs and is known to play a role in the pathogenesis of many diseases[3-5]. The relationship between oxidative stress and various diseases, including myocardial infarction, neurological diseases, asthma, diabetes mellitus, rheumatoid arthritis, and cancer, has been demonstrated in various studies[6-8].

It has been reported that oxidative stress may be an underlying mechanism for the development of obesity and may contribute to obesity-related metabolic disorders[9]. Some scholars[10] on obesity and oxidative stress have reported that obesity increases oxidative stress in relation to body fat percentage and BMI. There are several mechanisms by which obesity produces ROS. One mechanism is the mitochondrial and peroxisomal oxidation of fatty acids, and another mechanism is the excessive oxygen consumption resulting in free radical production in the mitochondrial respiratory chain[10,11].

Thiols are organic compounds that contain a sulfhydryl group (-SH), which consists of a hydrogen atom attached to a carbon atom and a sul-

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fur atom. Thiols react with oxidants *via* disulfide (RSH) bonds to form disulfide bonds (RSSR)[12]. This bond is called a disulfide bridge. Oxidative stress converts the sulfhydryl group of thiols into disulfide. The disulfide bonds formed are reduced to thiol groups under suitable conditions and become sulfhydryl. Thus, dynamic thiol/disulfide homeostasis is achieved[13]. It has been reported by Erel and Neselioglu[14] that the reduced thiol concentration increases, the native thiol (SH) concentration decreases, and the disulfide (SS) values increase correlatively under oxidative stress conditions.

Increased oxidative stress in obese subjects may further contribute to the development of atherosclerosis or other cardiovascular ailments[15,16]. In our study, we aimed to investigate whether weight control is achieved through bariatric surgery in morbidly obese individuals and, consequently, the reduction of adipose tissue leads to a decrease in oxidative stress and/or an increase in antioxidant response.

Patients and Methods

Study Design

This study was conducted with 40 cases aged 18 and 65 who underwent laparoscopic sleeve gastrectomy surgery between June and November 2022. This cohort study entailed a retrospective analysis of data collected prospectively. Age, sex, height, weight, body mass index (BMI), and laboratory data were analyzed using the obesity follow-up forms.

After obtaining informed consent forms from obese patients with a BMI over 40 who applied to the Department of General Surgery at Harran University Hospital, serum obtained from blood samples taken during preoperative routine tests and serum from routine blood samples taken at the 3rd and 6th months after the surgery were used. The patient's height and weight values were recorded. Their BMI was calculated using the ratio of weight to height (kg/m²), and their weight loss rates in the 3rd and 6th months were calculated.

Blood samples were obtained from obese patients with a BMI of 40 or above who applied to the Department of General Surgery at Harran University Hospital. The serum samples were collected from their preoperative routine blood tests after obtaining informed consent forms and from routine blood tests taken at 3 and 6 months postoperatively. The patients' height and weight values were recorded, and body mass index (BMI) was calculated using the weight and height ratios (kg/m²).

Patients with cardiovascular disease, cerebrovascular disease, proteinuria at the level of nephrotic syndrome, liver, and kidney disease, and malignancy were excluded from the study. In addition, those who use antioxidant drugs that may affect oxidative stress measurements, those who take vitamin supplements, n-acetyl-cysteine users, those taking lipid-lowering therapy, and smokers were excluded from the study.

Blood samples were obtained between 8 a.m. and 10 a.m. after 8-10 hours of fasting. The samples were then centrifuged at 1,500 rpm for 10 minutes. Separated serum samples were immediately frozen and stored at -80°C until analyzed. All thiol/disulfide parameters were studied in the same samples. Serum concentrations of native and total thiol and ratios of disulfide and native and total thiol were determined by a spectrophotometric method using an automatic clinical chemical analyzer (Roche, Cobas 501, Mannheim, Germany) as previously described by Erel and Neselioglu[14].

Plasma thiol concentrations were determined using 5, 5'-dithio-bis (2-nitrobenzoic acid) (DTNB) as described by Erel and Neselioglu[14]. Absorbances were measured at 412 nm against blank samples without DTNB. Plasma thiol concentration was calculated using 1.36 x 104 M-1 x cm-1 as the molar absorption coefficient. Intra- and interassay coefficients of variation for analyses were 4.5% and 4.0%, respectively. Results were expressed as µM plasma thiols.

Fasting blood sugar was measured with the hexokinase method after 10 h of fasting, followed by measurement of total cholesterol, total bilirubin, amylase, triglyceride, high-density lipoprotein (HDL) cholesterol and low-density lipoprotein (LDL) cholesterol, and total cholesterol. Triglycerides were measured with the enzymatic calorimetric test, HDL cholesterol was measured using the selective inhibition method, and LDL cholesterol was measured with the homogeneous enzymatic calorimetric test using an automatic analyzer (Advia 1650; Bayer, Fernwald, Germany). This study was approved by the Institutional Ethical Review Board of Harran University Hospital (Project No: E-76244175-050.04.04-144275) according to the Declaration of Helsinki and written informed consent was obtained from each participant before inclusion.

Statistical Analysis

The data of 40 morbidly obese individuals participating in the study were evaluated using the Windows operating system SPSS (version 17, Chicago, IL, USA) program. The Kolmogorov-Smirnov test was applied to determine whether each parameter followed a normal distribution. For parameters that were found to follow a normal distribution, One-Way ANOVA and Independent Samples Student's *t*-test were applied. For parameters that did not follow a normal distribution, the Mann-Whitney U Test and the Kruskal-Wallis Test were applied.

For intergroup correlation analyses of quantitative data, the Pearson Correlation Analysis Test was used. The data were presented as mean values ± standard deviation (SD). Results were considered statistically significant if $p<0.05$ in the analysis.

Results

The data of 40 patients included in the study were analyzed for preoperative, postoperative 2nd month, and postoperative 6th month, as shown in Table I. The mean age of the cases was 37 (19-54). 31 (77%) of the cases were female and 9 (23%) were male. It was determined that the changes in total thiol, AST, GGT, TBIL, LDL, and amylase values were not statistically significant ($p>0.05$). However, according to the analysis, it was found that the changes in native thiol, disulfide, reduced thiol, oxidized thiol, glucose, ALT, ALP, total cholesterol, HDL, triglycerides, and BMI values were statistically significant between the preoperative, postoperative 2nd month, and postoperative 6th-month findings ($p<0.05$).

When the source of the detected differences was examined using the Wilcoxon rank test, a significant decrease was observed in glucose, ALP, and BMI values in the postoperative 2nd-month period compared to the preoperative period ($p=0.039$, $p=0.011$, $p=0.000$, respectively).

Regarding the differences observed between the preoperative and postoperative 6th-month findings, it was found that the patients showed an increase in native thiol, reduced thiol, and HDL levels in the postoperative 6th month ($p=0.003$, $p=0.014$, $p=0.021$, respectively), while disulfide, oxidized thiol, glucose, ALP, total cholesterol, triglycerides, and BMI values showed a decreasing trend ($p=0.038$, $p=0.014$, $p=0.014$, $p=0.000$, $p=0.000$, $p=0.002$, $p=0.000$, respectively).

As for the differences observed between the postoperative 2nd-month and postoperative 6th-

Table I. Comparison of the preoperative and postoperative findings of the patients.

	Preop mean ± SD	Postop 2nd month mean ± SD	Postop 6th month mean ± SD	p^1	p^2	p^3	p^4
Native thiol (µmol/L)	273.4 ± 67.4	309.6 ± 70.8	367.9 ± 105.5	**0.003****	0.121	**0.003****	**0.004****
Total thiol (µmol/L)	421.1 ± 101.1	446.8 ± 111.9	461.0 ± 79.0	0.162	0.789	0.158	1.000
Disulfide (µmol/L)	73.8 ± 33.4	68.6 ± 37.8	46.5 ± 31.3	**0.049***	1.000	**0.038***	0.294
Reduced thiol (µmol/L)	65.9 ± 2.1	70.8 ± 12.0	79.3 ± 14.2	**0.021***	0.739	**0.014***	0.247
Oxidized thiol (µmol/L)	17.1 ± 6.1	14.6 ± 6.0	10.4 ± 7.1	**0.021***	0.738	**0.014***	0.247
Glucose (mg/ dl)	97.8 ± 20.2	91.2 ± 11.5	89.7 ± 6.8	**0.019***	**0.039***	**0.014***	0.731
AST (U/L)	27.4 ± 14.7	27.9 ± 26.8	22.2 ± 8.1	**0.045***	1.000	0.141	0.458
ALT (U/L)	22.8 ± 7.5	22.3 ± 10.5	21.1 ± 6.7	0.407	1.000	0.730	1.000
ALP (U/L)	70.7 ± 25.5	63.1 ± 21.7	56.1 ± 21.2	**0.000****	**0.011***	**0.000****	**0.009****
GGT (U/L)	25.1 ± 12.4	21.9 ± 16.7	21.1 ± 12.9	0.300	0.873	0.354	1.000
Total Bilirubin (mg/dL)	0.76 ± 1.09	0.71 ± 1.19	0.74 ± 1.34	0.726	1.000	1.000	1.000
T. Cholesterol (mg/dL)	132.3 ± 36.4	113.7 ± 30.3	96.7 ± 30.8	**0.000****	0.000	**0.000****	**0.000****
HDL (mg/dL)	39.9 ± 6.6	40.4 ± 5.1	42.7 ± 6.4	**0.020***	1.000	**0.021***	0.187
LDL (mg/dL)	77.4 ± 58.9	59.2 ± 26.5	65.5 ± 23.0	0.082	0.181	0.682	0.385
Triglyceride (mg/dL)	131.9 ± 62.2	116.5 ± 41.9	102.1 ± 22.4	**0.002****	0.066	**0.002****	**0.020***
Amylase (U/L)	56.1 ± 23.6	52.3 ± 18.5	51.9 ± 12.7	0.423	0.658	0.609	1.000
BMI (kg/m²)	46.3 ± 3.6	42.3 ± 3.0	36.3 ± 2.9	**0.000****	**0.000****	**0.000****	**0.000****

AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, ALP: Alkaline phosphatase, GGT: Gamma-glutamil transferase, HDL: High density lipoprotein, LDL: Low density lipoprotein, BMI: Body mass index, T. Cholesterol: Total cholesterol. $*p < 0.05$, $**p < 0.001$, p^1: Repeated measures, p^2: Preop-Postop 2nd month, p^3: Preop-Postop 6th month; p^4: Postop 2nd month-Postop 4th month, $p2$, p^3, p^4: Wilcoxon rank.

month findings, it was determined that native thiol levels increased in the postoperative 6th-month (p=0.004), while ALP, total cholesterol, triglycerides, and BMI values showed a decreasing trend (p=0.009, p=0.000, p=0.020, p=0.000, respectively).

Discussion

Oxygen molecules, which are very important for life, form free radicals that are highly reactive intermediates during energy production. The antioxidant defense system increases its main effect in case of free radical formation and tries to neutralize the formed free radicals. The balance between free radicals and antioxidants is necessary for physiological function[17]. If the balance is disturbed by radical formation, the body may face many diseases[18].

Oxidative stress is the most important underlying cause of changes in the body in the case of chronic obesity[10]. Adipose tissue dysfunction occurs in obese patients due to excessive production of proinflammatory cytokines because there is a positive correlation between oxidative stress and systemic inflammation.

There are various studies explaining the increase in oxidative stress observed in obese individuals. 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[19]. The activities of antioxidant enzymes such as superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx) significantly decrease with the increase in adipose tissue[20]. It has been reported that lipid peroxidation increases in plasma lipoproteins, erythrocyte membrane lipids and various tissues of obese individuals. Whether this increase is due to enzymatic lipid peroxidation (arachidonic acid pathway) or non-enzymatic pathway is still not fully understood[21].

In some studies[22], oxidant and antioxidant compounds have been examined to determine the oxidative stress state. Recent studies have focused on the role of thiols (RSH) in oxidative stress. Thiols are important antioxidant compounds that react with free radicals to provide protection against oxidative stress[22].

In a study by Uzun et al[23] the levels of RSH were found to be lower in obese individuals compared to normal-weight individuals. Vincent et al[24] reported that although not statistically significant, obese individuals had higher levels of total thiols and protein thiols compared to normal-weight individuals. Another study[25] reported a decrease in thiol concentrations with aging.

Stefanović et al[26] found that the total RSH levels of obese individuals were lower than those of normal-weight individuals. Positive correlations between free radicals generated as a result of oxidative stress and body mass index have also been reported in some studies[27,28].

In our study, we observed a statistically significant decrease in the levels of native thiols, reduced disulfides, and disulfides after surgery (p<0.05). Although the total thiol level gradually increased in each follow-up, it was not statistically significant (p>0.05). We observed a reduction in oxidative stress associated with weight loss in our patients due to decreased body mass index.

In a study conducted in 2017, Bawahab et al[29] reported higher plasma activity of antioxidant markers in morbidly obese individuals, stating that the presence of chronic inflammation and oxidative stress in obesity leads to an increase in superoxide radicals and a decrease in SOD activity as an adaptive response. They showed that sleeve gastrectomy, which achieves weight loss, inhibits the production of reactive oxygen species (ROS) and reduces the stimulating effect on SOD, GPx, GST, and vitamin C. Cătoi et al[30] reported an increase in NO levels and a decrease in total antioxidant response at 6 months after vertical banded gastroplasty, followed by a decrease in NO levels and an increase in total antioxidant response at one year.

We have found that our study is in line with the general consensus in the literature, which suggests a decrease in oxidative stress with weight loss. However, there is limited research evaluating the reduction in oxidative stress following obesity surgery by assessing the imbalance of thiol-disulfide equilibrium. Therefore, we believe that our study contributes significantly to this topic.

Conclusions

Increased reactive oxygen radicals in obese individuals lead to oxidative stress. The negative effects caused by free radicals are eliminated by the cell's antioxidant defense system and antioxidants obtained through nutrition. However, in cases of prolonged obesity, antioxidant system enzymes are lower. This imbalance between oxidant and antioxidant defense systems in obesity leads to cell damage and the development of various diseases, such as type 2 diabetes, cardiovascular diseases, and cancer. In morbidly obese individuals, weight control achieved through restrictive methods, such as sleeve gastrectomy, results in a reduction in oxidative stress and an increase in antioxidant response, due to the decrease in adipose tissue.

Conflict of Interest
The authors declare that they have no conflict of interests.

Ethics Approval
The study was approved by the Ethics Committee of the Medical Faculty of the Harran University and was performed in accordance with the Helsinki Declaration (Approval date and number: 20.06.2022-2022/12-17).

Availability of Data and Materials
The data generated and analyzed during the study are available from the corresponding author. They are not available publicly.

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Authors' Contribution
All authors contributed to the study's conception and design. Material preparation, data collection, and analysis were performed. The first draft of the manuscript was written by [HA and FK]. All authors read and approved the final manuscript. The first author [HA] had the idea for the article, and all authors reviewed the manuscript and data analysis and drafted and/or critically revised the work.

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References

1) Kim DS, Scherer PE. Obesity, diabetes, and increased cancer progression. Diabetes Metab J 2021; 45: 799-812.
2) Garcia-Sânci Ifz A, Gomez-Hermosillo L, Casillas-Moreno J, Pacheco-Moises F, Campos-Bayardo TI, Român-Rojas D, Miranda-Diaz AG. Prevalence of hypertension and obesity: profile of mitochondrial function and markers of inflammation and oxidative stress. Antioxidants 2023; 12: 165.
3) Rotariu D, Babes EE, Tit DM, Moisi M, Bustea C, Stoicescu M, Radu AF, Vesa CM, Behl T, Bungau AF, Bungau SG. Oxidative stress-Complex pathological issues concerning the hallmark of cardiovascular and metabolic disorders. Biomedicine & Pharmacotherapy 2022; 152: 1-15.
4) Panda P, Verma HK, Lakkakula S, Merchant N, Kadir F, Rahman S, Jeffree MS, Lakkakula BVKS, Rao PV. Biomarkers of oxidative stress tethered to cardiovascular diseases. Oxid Med Cell Longev 2022; 1-15.
5) Kazancioglu L, Batcik S, Arpa M, Kazdal H, Koyuncu T, Bilgin H, Erel Ö. The relationship between intraoperative body temperature and thiol/disulfide balance in geriatric patients undergoing elective transurethral prostate resection surgery with spinal anesthesia. Eur Rev Med Pharmacol Sci 2023; 27: 8523-8530.
6) Tauffenberger A, Magistretti PJ. Reactive oxygen species: beyond their reactive behavior. Neurochem Res 2021; 46: 77-87.
7) Truong L, Zheng YM, Kandhi S, Wang YX. Overview on interactive role of inflammation, reactive oxygen species, and calcium signaling in asthma, COPD, and pulmonary hypertension. Lung Inflammation in Health and Disease, Springer 2021; 2: 147-164.
8) Nakamura H, Takada K. Reactive oxygen species in cancer: Current findings and future directions. Cancer Sci 2021; 112: 3945-3952.
9) Çolak E, Pap D. The role of oxidative stress in the development of obesity and obesity-related metabolic disorders. J Med Biochem 2021; 40: 1-9.
10) Martinez-Martinez E, Cachofeiro V. Oxidative stress in obesity. Antioxidants (Basel) 2022: 639-641.
11) Zhou Y, Li H, Xia N. The interplay between adipose tissue and vasculature: Role of oxidative stress in obesity. Front Cardiovasc Med 2021; 8: 1-14.
12) Georgescu SR, Mitran CI, Mitran MI, Matei C, Popa GL, Erel O, Tampa M. Thiol-disulfide homeostasis in skin diseases. J Clin Med 2022; 11: 1-23.
13) Izgi M, Basaran B, Bayir T, Akca B, Celebioglu B. The effect of remifentanil and dexmedetomidine on dynamic thiol/disulfide balance in patients undergoing coronary artery bypass surgery: a prospective case-control study. Eur Rev Med Pharmacol Sci 2023; 27: 9050-9057.
14) Erel O, Neselioglu S. A novel and automated assay for thiol/disulphide homeostasis. Clin Biochem 2014; 47: 326-332.
15) Izzo C, Vitillo P, Di Pietro P, Visco V, Strianese A, Virtuoso N, Ciccarelli M, Galasso G, Carrizzo A, Veechiono C. The role of oxidative stress in cardiovascular aging and cardiovascular diseases. Life 2021; 11: 1-42.

16) Silveira Rossi JL, Barbalho SM, Reverete de Araujo R, Bechara MD, Sloan KP, Sloan LA. Metabolic syndrome and cardiovascular diseases: Going beyond traditional risk factors. Diabetes Metab J 2022; 38: 35-42.

17) Adetuyi BO, Adebayo PF, Olajide PA, Atanda OO, Oloke JK. Involvement of free radicals in the ageing of cutaneous membrane. WNOFNS 2022; 43: 11-37.

18) Unsal V, Cicek M, Sabancilar İ. Toxicity of carbon tetrachloride, free radicals and role of antioxidants. Rev Environ Health 2021; 36: 279-295.

19) Khalili F, Vaisi-Raygani A, Shakiba E, Kohsari M, Dehbani M, Naseri R, Asadi S, Rahimi Z, Rahimi M, Rahimi Z. Oxidative stress parameters and keap 1 variants in T2DM: Association with T2DM, diabetic neuropathy, diabetic retinopathy, and obesity. J Clin Lab Anal 2022; 36: 1-9.

20) Mullins CA, Gannaban RB, Khan MS, Shah H, Siddik MAB, Hegde VK, Reddy PH, Shin AC. Neural underpinnings of obesity: the role of oxidative stress and inflammation in the brain. Antioxidants 2020; 9: 1-21.

21) Kaneko T, Mita Y, Nozawa-Kumada K, Yazaki M, Arisawa M, Niki E, Noguchi N, Saito Y. Antioxidant action of persulfides and polysulfides against free radical-mediated lipid peroxidation. Free Radic Res 2022; 56: 677-690.

22) Erel O, Erdoğan S. Thiol-disulfide homeostasis: an integrated approach with biochemical and clinical aspects. Turk J Med Sci 2020; 50: 1728-1738.

23) Uzun H, Konukoglu D, Gelisgen R, Zengin K, Taskin M. Plasma protein carbonyl and thiol stress before and after laparoscopic gastric banding in morbidly obese patients. Obes Surg 2007; 17: 1367-1373.

24) Vincent HK, Vincent KR, Bourguignon C, Braith RW. Obesity and postexercise oxidative stress in older women. MSSE 2005; 37: 213-219.

25) Dröge W. Aging-related changes in the thiol/disulfide redox state: implications for the use of thiol antioxidants. Exp Gerontol 2002; 37: 1333-1345.

26) Stefanovic A, Kotur-Stevuljevic J, Spasic S, Bogavac-Stanojevic N, Bujisic N. The influence of obesity on the oxidative stress status and the concentration of leptin in type 2 diabetes mellitus patients. Diabetes Res Clin Pract 2008; 79: 156-163.

27) Vincent HK, Taylor AG. Biomarkers and potential mechanisms of obesity- induced oxidant stress in humans. Int J Obes 2006; 30: 400-418.

28) Sankhla M, Sharma TK, Mathur K, Rathor JS, Butolia V, Gadhok AK. Vardey SK, Sinha M, Kaushik GG. Relationship of oxidative stress with obesity and its role in obesity induced metabolic syndrome. Clin Lab 2012; 58: 385-392.

29) Bawahab MA, Assiri AS, Maksoud WA, Patel A, Kadoumi O, Zaman GS, Alessih RMK, Haider SS. Effects of weight reduction after sleeve gastrectomy on metabolic variables in Saudi obese subjects in Aseer Province of Kingdom of Saudi Arabia. Obes Surg 2017; 27: 3065-3074.

30) Câtoi AF, Pârvu A, Galea RF, Pop ID, Mureşan A, Câtoi C. Nitric oxide, oxidant status and antioxidant response in morbidly obese patients: the impact of 1-year surgical weight loss. Obes Surg 2013; 23: 1858-1863.