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REVIEW

Effect of Laparoscopic Sleeve Gastrectomy on Serum Adipokine Levels

Ľubica CIBIČKOVÁ[1,2], Michal GREGA[3], Roman DOHNAL[1,2], Jan SCHOVÁNEK[1,2]

[1]Department of Internal Medicine III – Nephrology, Rheumatology and Endocrinology, Faculty of Medicine and Dentistry, Palacky University, Olomouc, Czech Republic, [2]Department of Internal Medicine III – Nephrology, Rheumatology and Endocrinology, University Hospital Olomouc, Olomouc, Czech Republic, [3]Internal Department, Hospital Vítkovice, Czech Republic

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Summary

Bariatric procedures are considered to be the most effective treatment options for obesity. One of them is laparoscopic sleeve gastrectomy (LSG), which is nowadays very popular and widely used. LSG leads to weight loss and metabolic improvement and also changes adipokine levels, although it is just a restrictive operation. We describe changes in pro-inflammatory (leptin, resistin, visfatin and chemerin) and anti-inflammatory adipokines (adiponectin, omentin), with adiponectin and leptin being most studied. Their levels are markedly changed after LSG and this may partially explain the weight loss seen after LSG. Adipokines are closely connected to insulin resistance and chronic inflammation both being positively influenced after LSG. Leptin regulates amount of body fat, appetite, thermogenesis and metabolic rate and its levels are positively correlated with both weight and BMI changes after operation. Resistin influences insulin sensitivity, modulates body cholesterol trafficking and its changes after operation correlate with BMI, waist circumference, fat mass, LDL cholesterol and C-reactive protein. Chemerin, an important component of immune system, decreases after bariatric surgery and its levels correlate with BMI, triglyceride levels, and blood glucose. On the other hand, pro-inflammatory adipokine adiponectin, which influences fatty acid oxidation, browning of fat tissue and energy metabolism, is declining after LSG. This decline explains improvement of glucose status after bariatric surgery in patients with diabetes and is correlated with BMI loss, waist circumference and LDL cholesterol level. Effect of LSG goes beyond calory restriction and the changes of adipokines have a great impact on health status of the bariatric patients.

Key words

Sleeve gastrectomy • Adipokines • Leptin • Resistin • Adiponectin

Corresponding author

Ľubica Cibičková, Department of Internal Medicine III – Nephrology, Rheumatology and Endocrinology, University Hospital, I.P. Pavlova 6, 779 00 Olomouc, Czech Republic.
E-mail: cibickova@seznam.cz

Introduction

The prevalence of obesity has increased worldwide in the past 50 years, currently reaching pandemic levels [1]. Obesity represents a major health challenge because it increases the risk of diseases such as type 2 diabetes mellitus, non-alcoholic fatty liver disease, hypertension, myocardial infarction, stroke, dementia, osteoarthritis, obstructive sleep apnea and several types of cancer [1]. Obesity rates have increased in all ages and both sexes irrespective of geographical locality, ethnicity or socioeconomic status [2]. Current treatment methods, including lifestyle changes, diet and exercise, produce a weight loss of 5 % to 7 % on average [3]. Bariatric surgery is currently the most effective intervention to help obese individuals achieve significant and sustained weight loss and it is considered to be the gold standard strategy for treatment marked adiposity. The operation leads to dramatic and sustained weight loss, improvement in the quality of life and reduction in the risks of obesity-related disorders [4]. Among bariatric procedures,

laparoscopic sleeve gastrectomy (LSG) has gained popularity as the primary approach [5]. Together with Roux-en-Y gastric bypass (RYGB) it constitutes more than 80 % of the metabolic surgeries worldwide [4].

This restrictive bariatric procedure was first used as part of restrictive horizontal gastrectomy in the original Scopinaro-type biliopancreatic diversion [6]. This partial vertical gastrectomy served to reduce gastric capacity and initiate short-term weight loss while a malabsorptive component of the surgery provided long-term results [5]. Even though they could not undergo the second step, that is intestinal bypass, some patients were still able to reduce weight. That is why LSG has increasingly been used as a stand-alone bariatric procedure popular among both patients and gastric surgeons [5].

In LSG, a laparoscopic procedure, the stomach is resected vertically and approximately two thirds are removed [7]. It results in a narrow, tubular stomach. This partial removal leads to food intake restriction. The mechanisms of action through LSG produces early satiety, hunger control, and improvement in metabolic parameters go beyond simple caloric restriction. Potential mechanisms that contribute to the positive effects of this procedure are currently studied. This has been supported by observation that rats pair-fed to their sleeve-gastrectomized counterparts did not exhibit a comparable weight loss [8]. Metabolic improvement after LSG has been associated with different pathophysiologic mechanisms unrelated to weight loss such as increased gastric emptying and intestinal transit, bile acid metabolism, small intestine refactoring, alteration of the gut microbial community and the inflammatory response, and activation of some hormonal mechanisms [6,7]. The mechanism behind the metabolic effects of different bariatric surgeries including LSG has been attributed to changes in the enteroendocrine peptide hormone secretion (as PYY and GLP-1) and the reduction in the hedonic value for food and hunger than just structural alterations [9]. Changes in the secretion of hunger hormones (e.g. ghrelin) and satiety gut hormones have been described in detail recently [7] and they show similar results after just restrictive procedure as LSG and restrictive – malabsorptive procedure as RYGB [5]. However, there are different research outcomes regarding adipokines after bariatric surgery in the literature and the evidence is not consistent. It is difficult to conclude the exact change in adipokines because they were measured at different time point after bariatric surgery and because some authors mix results of different bariatric operations.

In our review we have focused on the change of adipose tissue-derived hormones (adipokines) after LSG. In our center, we take care mainly of patients after LSG while it is the most used bariatric procedure in our conditions. Although it is only a restrictive bariatric procedure, we have very good experience with this method as a way for weight reduction. In many studies, patients underwent various bariatric procedures and the results concerning adipokine levels were mixed together. However, we have only concentrated on adipokines after LSG and selected studies that study this kind of operation. For this purpose, we have made a literature search based on PubMed database.

Adipokines are hormones produced by adipose tissue, which is no longer regarded as a passive depot for storing excess energy. Adipokines are secreted according to signals modulating appetite, insulin sensitivity, glucose homeostasis, energy expenditure, inflammation and immunity [10]. Abnormal shifts in adipokine levels lead to chronic state of low-grade inflammation and are associated with obesity-related diseases such as insulin resistance, type 2 diabetes, hyperlipidemia, stroke, hypertension, atherosclerosis and different types of cancer [11]. Adipocytes secrete proinflammatory cytokines which have substantial role in the pathogenesis of insulin resistance and systemic inflammation. It is known, that metabolic surgery can significantly reduce white adipose tissues mass and has been shown to downregulate the serum inflammatory mediators interleukin 6 (IL-6) and tumor necrosis factor (TNF-alpha) [7,12]. Inflammatory mediators are produced by macrophages which infiltrate white adipose tissue in obese patients. Such macrophages predominantly present the M1 pro-inflammatory phenotype and promote inflammation by releasing TNF-alpha and IL-6, thus contributing to insulin resistance [13]. The change in IL-6 levels positively correlates with BMI after metabolic surgery [14].

Effects of LSG on diabetes mellitus shows that it is more than a restrictive bariatric surgery procedure, because there are higher type 2 diabetes mellitus remission rates after LSG than after other restrictive techniques such as laparoscopic adjustable gastric banding [6]. Furthermore, this improvement occurred soon after surgery when significant weight loss had not yet been achieved [6]. These findings are attributed to changes in the gut hormonal mechanisms as improvement in chronic inflammation mentioned above.

We start our review with leptin, which is the most studied adipokine with potent anorexic and anti-diabetic roles [4].

Pro-inflammatory adipokines

Leptin

Leptin is a polypeptide hormone secreted by adipose tissues and stomach fundus cells. It is synthesized in white adipose tissue proportionally to the amount of body fat, reduces food intake and body weight through actions in the central nervous system [6]. Leptin suppresses appetite and increases the metabolic rate and thermogenesis through binding to leptin receptors expressed in the hypothalamus [15]. After high-calorie food consumption, serum leptin level would be increased to keep the balance of energy control and body weight [12]. The amount of leptin is proportional to body fat content. In contrast to that, in obesity, leptin levels are usually normal or slightly elevated relative to the amount of adipose tissue. That is why a decreased sensitivity to leptin has been suggested, resulting in an inability to detect satiety despite high energy stores [15,16]. Reducing leptin resistance rather than a mere increase in its plasma levels may reduce adipose tissue mass and obesity [15].

It is unclear whether the improvement in leptin resistance plays a direct role in weight loss after LSG. While related genes seem to increase its expression, an effect on leptin sensitivity after LSG was not confirmed in rats [17]. Recent studies suggest that the reversal of leptin resistance could be regulated by protein availability [6]. A human study performed by Mazahreh *et al.* showed decreased leptin resistance. They measured serum leptin and leptin receptor levels and calculated a leptin resistance index (estimated by adjusting the leptin-to-BMI ratio to the corresponding leptin receptor concentration) in patients 12 months after LSG. They found a significant reduction of the BMI, leptin-to-BMI ratio and leptin resistance index in patients after LSG [15]. According to the authors, feedback loops that regulate the levels of leptin and its receptor are dysregulated in obese patients. Bariatric surgery could restore these feedback loops, resulting in the improvement of leptin resistance by downregulating leptin expression and/or upregulating leptin receptor levels/activity [15].

Reduction in leptin levels after LSG has been proved in both rats [8] and humans also in a recent meta-analysis [12]. Leptin levels decreased insignificantly within four months [18], but significantly within six months [19], 12 months [11,14] or 18 months from LSG [20] and remained lower (54 % from baseline) even after four years [21]. Leptin changes were observed to be positive correlated with both weight and BMI changes [11], suggesting that a change in weight is associated with a change in leptin [14,21]. BMI loss was found to be an independent predictor of serum leptin level decrease [14]. Leptin levels decreased not only after LSG, but there is a comparable reduction in serum leptin levels after either RYGB or LSG [12].

Resistin

Resistin is a cysteine-rich protein identified by screening for the genes that are induced during differentiation of adipocytes [10]. It received its name from its apparent induction of insulin resistance in mice. In obese mice, serum levels of resistin are markedly increased and can be decreased by use of thiazolidinediones that increase insulin sensitivity [22]. Moreover, neutralization of resistin activity (by injecting antibodies against resistin) decreased blood glucose levels and improved insulin sensitivity in obese, insulin-resistant mice, and the injection of resistin into mice worsened glucose tolerance and induced insulin resistance [22]. However, data on the role of resistin in human insulin sensitivity and obesity are controversial and are not as convincing as those in rodents. It may play a causal role in the development of insulin resistance and type 2 diabetes but this remains to be confirmed [23].

Resistin is also thought to act as a modulator of body cholesterol trafficking that increases LDL cholesterol and enhances degradation of liver LDL receptors, thus contributing to atherosclerosis pathogenesis [13]. In white adipose tissue, resistin promotes pro-inflammatory cytokine production through the resistin receptor and is found to be increased in obesity [24]. Lower levels were seen in lean individuals compared to obese patients before bariatric surgery [11,31].

Six, 12 and also 18 months after surgery, a significant decrease in resistin concentrations was reported, which reached the values of normal-weight subjects [11,14,19,20]. No correlations between changes in resistin levels and improvements in anthropometric and metabolic parameters were observed in a study by Marantos *et al.* [14]. In a recent study, resistin levels were notably associated with age, weight and BMI while

a weak negative association was seen in the case of LDL cholesterol and vitamin D [11]. At the same time, there was no statistical difference in resistin levels between morbidly obese patients and healthy subjects of normal weight, or between obese patients before and after weight loss [32]. Resistin levels were even higher in non-obese patients compared to obese patients and in obese patients resistin levels further decrease after LSG [31]. Another study showed a significant positive correlation of resistin with BMI and plasma glucose levels in diabetic women while for non-diabetic women and lean persons, no such correlation was found [33]. In humans, resistin is located on a chromosome region that has not been linked to susceptibility for obesity [22]. There are also many other hormones that affect insulin resistance and resistin may not be a major determinant of insulin resistance. This might explain the above-mentioned conflicting resistin results. De Luis *et al.* explain this data inconsistency by a possible link between chronic inflammation and resistin [10]. They have found significant correlations of resistin levels and BMI, waist circumference, fat mass, LDL cholesterol and C-reactive protein [10]. In their multivariate analysis, only C-reactive protein remained as an independent predictor for resistin concentration [10]. As already mentioned above, adipose tissue is known to be infiltrated by M1 macrophages producing inflammatory markers and this fact could explain these findings. Additionally, there are studies about the reducing effects of thiazolidinediones on resistin levels in mice, as mentioned above [10].

Visfatin

Visfatin is a pro-inflammatory adipokine that is preferentially expressed in visceral adipose tissue as compared to subcutaneous adipose tissue. It plays a role in insulin sensitivity and its production is increased in obesity and correlates with visceral adiposity [13]. Total cholesterol, LDL-cholesterol and resistin levels are known to be elevated in patients with visfatin levels above the median value [25]. It remains to be established whether visfatin production is a compensatory response to tissue-specific insulin resistance of more simply a marker of tissue-specific inflammatory-cytokine action. De Luis showed that visfatin is related in a negative way to insulin resistance and relation was detested with inflammatory markers such as TNF-alpha [25]. However, studies on the influence of LSG on this adipokine are, to the best of our knowledge, lacking.

Chemerin

Chemerin is an important component of the immune system and potent chemoattractant adipokine that is secreted by white fat tissue. Chemerin has a major contribution in body weight hemostasis and energy balance through regulation of adipogenesis and insulin sensitivity [26]. As adipose tissues expand and chronic low-grade inflammation develops in obese individuals, the level of serum chemerin elevates markedly [27]. Chemerin elevated levels are associated with many obesity-related metabolic and inflammatory disorders, such insulin resistance, high blood pressure, cardiovascular diseases, metabolic syndrome and Chron´s disease [4,26]. Moreover, it was found that serum chemerin levels correlated positively with BMI, triglyceride levels, and blood glucose, which reflects its importance as a proxy for the metabolic state in obese patients [4]. However, little research investigated the impact of bariatric operations on serum chemerin concentrations. In a recent meta-analysis, its values declined after LSG and were linked to alterations in glucose profile and BMI [4]. Both LSG and RYGB led to significant, comparable decrease in serum chemerin levels [4,28].

Table 1. Pro-inflammatory adipokines after laparoscopic sleeve gastrectomy

Name	Function	Effect of LSG
Leptin	Regulates amount of body fat, appetite, thermogenesis and metabolic rate	↓
Resistin	Insulin sensitivity, modulator of body cholesterol trafficking	↓
Visfatin	Insulin sensitivity	Not known
Chemerin	Component of immune system	↓

Anti-inflammatory adipokines

Adiponectin

White adipose tissue browning plays a crucial role in energy metabolism. Adiponectin is expressed exclusively by adipocytes and it is known to upregulate the expression of browning genes [29]. It is inversely correlated with the degree of visceral adiposity [13] and low circulating adiponectin levels were found in obesity. Adiponectin plays an anti-inflammatory role and promotes insulin sensitivity by increasing fatty acid oxidation, thus regulating lipoprotein metabolism and inhibiting hepatic gluconeogenesis [13].

Most studies have shown a significant increase of adiponectin after LSG, leading to upregulation of the expression of the browning genes in adipocytes [29]. Only one small study failed to show significant changes in adiponectin levels at four years after LSG [21], probably due to small sample size. While increased adiponectin was observed as early as four months after LSG, leptin did not rise so shortly after the surgery [18]. A decreased leptin-to-adiponectin ratio was driven primarily by an increase in adiponectin rather than a reduction in leptin, with these changes being proportional to the reduction in weight after surgery [18].

Six, 12 and 18 months after surgery, significantly elevated adiponectin was observed [11,14,19,20] and these results are proven also by recent large meta-analysis [12]. The levels almost doubled [11] but were significantly lower than those in lean individuals [14]. Adiponectin changes were negatively correlated with the BMI and weight loss [11,14]. Moreover, the change in adiponectin levels negatively correlated with improvements in LDL and glycated hemoglobin levels 12 months after surgery, thus suggesting a correlation between serum adiponectin levels and determinants of metabolic syndrome [14]. BMI loss, waist circumference, LDL level and glycated hemoglobin level reduction were independent predictors of serum adiponectin level increase [14]. Adiponectin was the adipokine that best positively correlated with the overall improvement in the metabolic profile [14]. The revealed inverse correlation between adiponectin concentration and BMI in obese patients shows that adipose tissue with increasing fat mass decreases fatty acid oxidation in muscles and the liver and this apparently contributes to lipid accumulation in the cells [11]. Moreover, adiponectin is an insulin-sensitizing hormone and its increased levels after surgery can partly explain why blood glucose status can be improved after bariatric surgery in patients with diabetes [12].

Omentin

Omentin is an anti-inflammatory adipokine with insulin-sensitizing effects whose levels are decreased in obesity and diabetes and inversely correlated with BMI [13,30]. It is also associated with cardiovascular disease and expressed throughout the body (the heart, lungs, ovaries and placenta) but the main site of its production is visceral tissue [30]. It has been studied in patients following another type of bariatric surgery – biliopancreatic diversion with duodenal switch – but not in those undergoing LSG. Omentin increased as early as 24 hours post-surgery, with changes maintained up to one year [30].

Table 2. Anti-inflammatory adipokines after laparoscopic sleeve gastrectomy

Name	Function	Effect of LSG
Adiponectin	Fatty acid oxidation, browning of fat tissue, energy metabolism	↑
Omentin	Insulin sensitivity	Not known

Conclusions

In last twenty years, our knowledge about various adipokines has rapidly increased and parallel to that popularity of bariatric surgery rises, LSG being one of the most carried. Accumulating evidence suggests that bariatric surgery improves the production of anti-inflammatory adipokines and decreases the production of pro-inflammatory ones. Among them, adiponectin and leptin are preferably explored and understood. Leptin levels drop rapidly soon after bariatric surgery and then continue to decline during the follow-up; in conformity with this finding, anti-inflammatory hormone adiponectin levels rise after LSG. Resistin and omentin dynamics

show less agreement and visfatin has been little studied in bariatric patients so far. Although we do not understand all mechanism and all changes in adipokines, we see the positive effects of bariatric surgery in our daily praxis. Bariatric surgery not only helps in weight reduction, but also improves the metabolic state including glucose metabolism and chronic inflammation which both are linked to obesity. LSG goes beyond calory restriction and although being just a restrictive method we see great impact of this procedure on health status of the bariatric patients. It is even more effective than endoscopic sleeve gastroplasty not only in weight reduction but also in adipocyte levels change [9]. Nevertheless, not all patients profit from this bariatric procedure in the same extent. Some patients are able to keep the weight loss and metabolic improvement while the others need another bariatric operation because of weight regain. New studies are needed to elucidate the role of adipokines to better understand this paradox.

Conflict of Interest

There is no conflict of interest.

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