Comparative Assessment Of Leptin And Adiponectin Dynamics Following Different Bariatric Procedures

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ABSTRACT

This research focuses on evaluating changes in leptin and adiponectin concentrations in individuals undergoing bariatric surgery. These adipocytokines, which regulate fat metabolism and contribute to the progression of obesity, are key elements in its pathophysiology. The study enrolled 44 patients treated at the Endocrinology Department of TMA and Medion Family Hospital between 2022 and 2024. Participants were allocated into two cohorts: those who underwent gastric bypass (n = 19) and those who received sleeve gastrectomy (n = 25). Prior to and following the surgical intervention, biochemical indicators, hormonal profiles, and lipid metabolism parameters were analyzed. Twenty-four months post-operation, patients demonstrated a marked reduction in leptin and a notable rise in adiponectin, underscoring the positive metabolic impact of bariatric procedures in individuals with obesity.

Keywords: Leptin, adiponectin, bariatric surgery, obesity, weight loss, cytokines, metabolic health

Introduction. Cytokines that regulate lipid metabolism and contribute to the development of obesity play a significant role in its pathogenesis. Among the pro-inflammatory cytokines are leptin and adiponectin, whose circulating levels are altered in individuals with obesity. The study by Shanwen Charleen Yeo et al. confirms that cytokines such as leptin and adiponectin are key participants in the mechanisms underlying obesity, with their concentrations shifting in response to this condition [18].

The work of M. Borges-Canha and colleagues demonstrates that leptin and adiponectin levels undergo substantial alterations in patients with obesity, closely correlating with metabolic syndrome and insulin resistance [2]. The study by Duc-Vinh Pham and P. Park further shows that an imbalance of adipokines—particularly leptin and adiponectin—contributes to inflammatory processes and a range of obesity-related disorders [11]. Similarly, J. Ryu et al. report that adiponectin circulates in serum predominantly in trimeric, hexameric, and high-molecular-weight isoforms, with the latter exhibiting the greatest biological activity [13].

Moreover, the analysis by L. Ma and colleagues confirms that serum adiponectin concentrations in healthy adults typically range between 10–16 μg/mL [9]. Levels of adiponectin are frequently reduced in individuals with obesity, type 2 diabetes (T2DM), hypertension, dyslipidemia, and ischemic heart disease (IHD). A study conducted by Zahra Mazloum Khorasani et al. provides evidence that decreased adiponectin levels are associated with an elevated risk of IHD in patients with T2DM [8].

Additionally, the work of Xiaosi Hong and co-authors shows that individuals with obesity, dyslipidemia, and hypertension exhibit significantly lower adiponectin levels, which is linked to the development of T2DM [5]. The findings of P. Tangri and N. Tangri also support the notion that patients with obesity and T2DM

have markedly reduced adiponectin concentrations compared with healthy individuals, suggesting its contributory role in the pathogenesis of these conditions [15].

Adiponectin levels below 4.0 μg/mL are considered a predictor of increased risk for T2DM and dyslipidemia. The analysis by Zahra Mazloum Khorasani et al. corroborates that reduced adiponectin levels are associated with a heightened likelihood of developing T2DM and lipid disorders, thereby promoting the progression of atherosclerotic changes [8].

Another key cytokine is leptin, a peptide hormone involved in the regulation of energy metabolism. It is produced primarily by adipocytes and by enterocytes of the small intestine [1]. Leptin suppresses appetite, thereby reducing fat accumulation within adipocytes. This 16-kDa protein consists of 167 amino acids and is encoded by the LEP gene located on chromosome 7.

The hormone exerts its principal effects through receptors in the arcuate and ventromedial nuclei of the hypothalamus, as well as on dopaminergic neurons of the ventral tegmental area, thereby modulating feeding behavior. The influence of leptin on neurons in the ventromedial hypothalamus was further explored by Wiebe Venema and colleagues, who demonstrated that leptin activates the JAK2–STAT3 pathway in arcuate nucleus neurons, contributing to appetite suppression and the regulation of food intake [17].

The central physiological role of leptin is to regulate adipose tissue mass by modulating hunger, energy expenditure, physical activity, and overall energy balance [7].

In obesity, reduced sensitivity to leptin—analogous to insulin resistance in type 2 diabetes—results in an impaired ability to perceive satiety despite excessive energy stores and elevated circulating leptin levels. Experimental work by M. Niimi further demonstrates that mutations in the leptin gene or its receptors can diminish leptin sensitivity, contributing to the development of obesity and related metabolic disturbances [10].

The primary function of leptin is the regulation of adipose tissue mass through central modulation of appetite, energy expenditure, physical activity, and overall energy balance. Its secondary functions include influencing energy utilization. In the lateral hypothalamus, leptin suppresses hunger by counteracting orexigenic mediators such as neuropeptide Y and anandamide. In the medial hypothalamus, it enhances satiety by promoting the synthesis of α-MSH, a potent appetite-suppressing peptide. Notably, leptin-induced appetite suppression is long-acting, unlike the rapid, short-lived effects of cholecystokinin (CCK) or the more gradual inter-meal suppression mediated by PYY3-36.

A deficiency of leptin or its receptor results in uncontrolled hyperphagia and subsequent obesity [4]. Fasting or adherence to very low-calorie diets leads to a reduction in leptin levels. Importantly, leptin concentrations fluctuate more dramatically in response to decreased energy intake than to increased caloric consumption. These dynamic changes in leptin, driven by abrupt shifts in energy balance, appear to correlate more closely with appetite regulation and subsequent food intake than with adipose tissue mass per se.

The present study aims to investigate the complex roles of adiponectin and leptin within the context of obesity and its associated metabolic disturbances. A deeper understanding of the mechanisms through which these cytokines act will enhance our insight into the pathophysiology of obesity and support the development of targeted therapeutic strategies.

Aim of the Study. To evaluate the changes in leptin and adiponectin levels in patients before and after bariatric procedures, specifically gastric bypass and sleeve gastrectomy.

Materials and Methods. The study was conducted at the Endocrinology Department of TMA and the Medion Family Hospital, where bariatric surgeries were performed. A total of 44 patients who underwent bariatric surgery between 2022 and 2024 were included. The mean age of participants was 38.59±8.77 years; 4 were men (9.1%) and 40 were women (90.9%). The average body weight was 108.88±15.70 kg, and the preoperative BMI was 40.19±5.97 kg/m^2.

Patients were divided into two groups based on the surgical method: gastric bypass (n = 19) and sleeve gastrectomy (n = 25). The follow-up period for all participants was 24 months.

Results. A total of 44 patients participated in the study, of whom 90.9% were women and 9.1% were men. The mean age was 38.59 years, with an average body weight of 108.88 kg and a preoperative BMI of 40.19 kg/m^2. Patients were assigned to two groups based on the type of bariatric intervention performed.

Table 1 demonstrates that, in the sleeve gastrectomy group, leptin levels decreased from 8.25±2.38 ng/mL to 4.75±1.16 ng/mL (P<0.001), whereas adiponectin levels increased from 7.97±1.06 ng/mL to 13.49±2.61 ng/mL (P<0.001). These shifts reflect the reversal of leptin resistance and a notable improvement in metabolic status associated with postoperative weight reduction.

Table 1
Leptin and Adiponectin Levels in Patients Undergoing Sleeve Gastrectomy

Parameter	Before surgery (Mean ± SD)	After 24 months (Mean ± SD)	P
Leptin (ng/mL)	8,25±2,38	4,75±1,16	P<0,001
Adiponectin (ng/mL)	7,97±1,06	13,49±2,61	P<0,001

Note: The comparison of differences was performed between the groups before and after the surgical intervention, respectively. * – P<0.05, ** – P<0.001.

Table 2 demonstrates similar trends in patients who underwent gastric bypass surgery. Leptin levels decreased from 8.54±2.38 ng/mL to 4.87±1.04 ng/mL (P<0.001), while adiponectin levels increased from 8.32±0.87 ng/mL to 14.18±2.67 ng/mL (P<0.01). These findings confirm the effectiveness of bariatric surgery in normalizing key hormones involved in lipid metabolism.

The study conducted by M. Salman and his team shows that following Roux-en-Y gastric bypass, leptin levels decline significantly, whereas adiponectin levels rise, indicating restoration of metabolic balance and improvement of hormonal status [14]. Furthermore, the work of X. Unamuno and colleagues also demonstrates that one year after gastric bypass, leptin concentrations decrease and adiponectin levels increase, contributing to an improved metabolic profile [16]. Similarly, the research by Gisele Farias et al. confirms that two years after gastric bypass, leptin levels continue to decline while adiponectin levels rise, reflecting a substantial enhancement of the patients' metabolic state [3].

Table № 2
Leptin and Adiponectin Levels in Patients Undergoing Gastric Bypass Surgery

Parameter	Before Surgery (Mean ± SD)	After 24 Months (Mean ± SD)	P-value
Leptin (ng/mL)	8,54±2,38	4,87±1,04	P<0,001
Adiponectin (ng/mL)	8,32±0,87	14,18±2,67	P<0,01

Note: The comparison of differences was performed between the groups before and after the surgical intervention, respectively. * – P<0.05, ** – P<0.001.

Our findings show a pronounced decrease in leptin levels and a substantial rise in adiponectin levels two years after bariatric surgery, aligning closely with results reported in previous studies.

Research conducted by S. Hosseini and colleagues demonstrated that following sleeve gastrectomy (SG) and Roux-en-Y gastric bypass (RYGB), leptin concentrations declined significantly, while adiponectin levels increased markedly. They reported that both SG and RYGB produced greater reductions in leptin and notable elevations in adiponectin compared with baseline, changes that were strongly associated with improved metabolic parameters [6].

Furthermore, the study by M. Rafey et al. showed that 12 months after laparoscopic sleeve gastrectomy, leptin levels dropped from 40.7±24.9 to 30.9±30.5 ng/mL, whereas adiponectin levels rose from 4.49±1.6 to 8.93±6.36 μg/mL. These results corroborate our observations of a significant decrease in leptin and a substantial increase in adiponectin following surgery [12].

Additionally, the investigation by M. Salman and colleagues confirmed that both RYGB and LSG lead to marked reductions in leptin and increases in adiponectin 12 months after surgery, indicating restoration of hormonal balance and enhancement of the patients' metabolic profiles [14].

Thus, our results are consistent with findings from other researchers, reinforcing the evidence for the beneficial effects of bariatric surgery on leptin and adiponectin levels and, consequently, on the metabolic health of patients with obesity. These hormonal shifts are fundamental to understanding the mechanisms underlying metabolic improvement and the reduction in the risk of obesity-related comorbidities following surgical interventions.

Conclusion. Our study demonstrated that bariatric surgery has a substantial impact on leptin and adiponectin levels in patients with obesity, contributing to significant improvements in metabolic health. Two years after surgery, leptin levels had markedly decreased, while adiponectin levels had significantly increased in patients who underwent both sleeve gastrectomy and gastric bypass. These hormonal shifts indicate a reversal of leptin resistance and an overall enhancement of metabolic status.

The observed improvements in hormonal parameters confirm the effectiveness of bariatric procedures in normalizing adipokine regulation associated with lipid metabolism and inflammatory processes. Our findings align with previous research, showing that surgical intervention helps restore hormonal balance and improve the lipid profile in individuals with obesity.

Thus, bariatric surgery represents an effective therapeutic approach for improving metabolic health in obese patients through substantial reductions in leptin levels and increases in adiponectin levels. These changes not only contribute to weight reduction but also decrease the risk of metabolic and cardiovascular diseases associated with obesity.

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