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Dynamics of the Eating Behavior Hormone Levels Leptin and Ghrelin in Patients with Gastroesophageal Reflux Disease Depending on the Treatment Strategy

The aim of the work: to assess leptin and ghrelin levels in patients with gastroesophageal reflux disease, to assess their dynamics and to determine their relationship with body mass index.

Materials and Methods. The study included the examination and treatment of 90 patients with gastroesophageal reflux disease (GERD). Laparoscopic total fundoplication in a modification of the Short Floppy Nissen technique was performed in 35 patients. Drug therapy was prescribed to 35 patients. The comparison group consisted of 20 healthy volunteers. The groups of patients and healthy control subjects were matched for gender and age. Venous blood for the study was collected from patients in the comparison group, and in the groups before and after surgical or therapeutic treatment, as well as from all patients 2–3 months after treatment. Ghrelin (Ghrelin, ELISA, BMS2192, Ebioscience) and leptin (Leptin, Sensitiv ELISA, KAC2281, Ebioscience) were tested on the ImmunoChem-2100 enzyme-linked immunosorbent assay (ELISA) (USA).

Results. The median ghrelin level in healthy participants was 21.2 ng/mL [5.44; 22.7], leptin 0.8 ng/mL [0.22; 1.5], and body mass index (BMI) 21 kg/m² [18; 25]. In healthy volunteers, a strong positive correlation was observed between leptin levels and BMI ($r=0.666$, $p=0.001$). The correlation between ghrelin and leptin ($r=-0.373$, $p=0.105$) and between ghrelin and BMI ($r=-0.296$, $p=0.206$) was not statistically significant. Significant differences were found in concentrations of the metabolic hormones leptin and ghrelin, as well as in BMI, in patients with GERD compared to healthy volunteers. Changes in these parameters were also observed after surgical and therapeutic treatment. A positive correlation between leptin concentration and BMI was established in all groups. Patients had increased leptin levels and decreased ghrelin levels before surgery and before the start of therapy compared to the control group. After surgery, there was a significant increase in ghrelin levels compared to preoperative levels, while leptin concentrations decreased. A moderate negative correlation was found between ghrelin levels and BMI after therapeutic treatment. Correlation analysis demonstrated a consistent relationship between leptin and BMI in all study groups. The contribution of ghrelin to body weight regulation appeared to be more context-dependent, varying depending on the stage of treatment and the patient's energy status.

Conclusions. Patients with gastroesophageal reflux disease have an increased leptin levels and decreased ghrelin levels. The consistent association between leptin and body mass index in all groups confirms its key role as a marker of adipose tissue, while the greater variability of ghrelin indicates its sensitivity to changes in energy balance and to the effects of treatment. The more pronounced hormonal dynamics observed after surgery compared with therapeutic treatment suggests that antireflux surgery may affect not only reflux mechanisms but also metabolic regulation.

Key words: Gastroesophageal Reflux Disease; Laparoscopic Fundoplication; Eating Behavior; Ghrelin; Leptin.

Problem Statement and Recent Research

Analysis. Excess body weight is a major risk factor for the development of gastroesophageal reflux disease (GERD), influencing its clinical manifestations and treatment outcomes, including surgical interventions. Disruption of energy balance and alterations in eating behavior in patients with GERD are accompanied by changes in hormones regulating appetite and metabolism, among which leptin and ghrelin play a central role [1, 2].

Leptin, a hormone predominantly secreted by adipose tissue, participates in the regulation of energy balance, suppression of appetite, and control of excess body weight by reflecting long term energy stores in

the organism. Its role in maintaining the balance between energy intake and expenditure is supported by contemporary studies on weight regulation and human energy metabolism [3].

Ghrelin, synthesized primarily by gastric cells, acts as a potent stimulator of appetite and contributes to short term regulation of feeding behavior, with circulating concentrations rising before meals and declining after food intake. The dynamic interaction between ghrelin and leptin is considered an important component of energy homeostasis, particularly under conditions associated with changes in body weight [4].

Alterations in the levels of these hormones have been described in various metabolic states, including

obesity, as well as following interventions that affect body weight and eating behavior. Studies have demonstrated that ghrelin and leptin concentrations may change after bariatric surgery, reflecting adaptive modifications in the regulatory mechanisms controlling appetite and energy metabolism [4].

However, data regarding the dynamics of these hormones in patients with GERD, particularly in the context of surgical treatment, remain limited [5]. Current evidence indicates that an imbalance between leptin and ghrelin is associated with excess body weight, obesity, and metabolic disturbances. Laparoscopic Nissen fundoplication remains one of the most effective treatments for GERD, providing durable antireflux control.

Nevertheless, the consequences of this intervention for the regulation of eating behavior, appetite, and hormonal balance have not been sufficiently investigated [5]. Furthermore, given the association between body weight changes and the progression of GERD, investigating leptin and ghrelin in this patient population may contribute to a better understanding of the pathophysiological mechanisms underlying the disease and its treatment.

The aim of the work: to assess leptin and ghrelin levels in patients with gastroesophageal reflux disease, to assess their dynamics and to determine their relationship with body mass index.

Materials and Methods. The study included the examination and treatment of 90 patients. Laparoscopic total fundoplication (TSF) modified by the Short Floppy Nissen technique was performed in 35 patients with gastroesophageal reflux disease (GERD). Drug therapy was prescribed to 35 patients with GERD, including two months of proton pump inhibitors and prokinetic agents. The comparison group consisted of 20 healthy volunteers. The patient and healthy control groups were matched by gender and age. Venous blood was collected for the study in the comparison group before and after surgical or therapeutic treatment and 2-3 months after treatment. Ghrelin (Ghrelin, ELISA, BMS2192, Ebioscience) and leptin (Leptin, Sensitiv ELISA, KAC2281, Ebioscience) were tested on the immunoenzyme complex ImmunoChem-2100 (USA) at the Department of Clinical Laboratory Diagnostics of Zaporizhzhia State Medical and Pharmaceutical University. The concentration of the indicators was expressed in ng/ml [6].

Inclusion criteria: GERD, consent for treatment (surgical or therapeutic), absence of general contraindications to treatment (surgical or therapeutic), absence of acute surgical pathology or other chronic gastrointestinal diseases in the acute stage.

Exclusion criteria are the absence of GERD, refusal of treatment (both surgical or therapeutic), the presence of acute surgical pathology or other chronic gastrointestinal diseases in the acute stage.

For the statistical evaluation of the study results, the Statistica for Windows 13 software package (StatSoft Inc., No. JPZ804I382130ARCN10-J) and GraphPad Prism version 11.0.0 (GraphPad Software, Boston, MA, USA) were used. All results were checked for compliance with the normal distribution law. Data with normal distribution were presented as mean \pm standard deviation ($M \pm SD$), whereas non-normally distributed data were described using the median and interquartile range (25th–75th percentiles). The data were presented as $x(x-x)$. Differences and interrelationships between groups were assessed using nonparametric methods, including the Mann – Whitney U-test and Spearman rank correlation analysis. Graphical visualization, including correlation plots, was performed using GraphPad Prism 11. Differences were considered statistically significant at $p < 0.05$. All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Results. Statistically significant differences in metabolic hormone concentrations and body mass index were identified between healthy controls and patients at various stages of treatment (Table).

Median ghrelin levels in healthy participants were 21.2 ng/ml [5.44; 22.7], leptin 0.8 ng/ml [0.22; 1.5], and body mass index (BMI) 21 kg/m² [18; 25] (Table).

In patients prior to surgical intervention, ghrelin concentrations were significantly lower than in controls at 12.21 ng/ml [0.83; 21.15] ($p < 0.001$). Following surgery, ghrelin levels increased significantly to 19.8 ng/ml [14.8; 31.5] ($p < 0.001$ vs preoperative values), approaching those observed in healthy participants. Similarly, patients before conservative therapy demonstrated reduced ghrelin levels of 14.6 ng/ml [3.9; 33.4] ($p < 0.01$ vs controls), whereas after therapy the median reached 18.4 ng/ml [4.0; 34.7], with no statistically significant differences from baseline (Table).

Leptin concentrations were significantly higher in patients prior to surgery compared with healthy volunteers, reaching 1.63 ng/ml [0.6; 8.6] ($p < 0.001$). After surgical treatment, leptin levels decreased significantly to 0.97 ng/ml [0.2; 6.4] ($p < 0.01$ vs preoperative values). In the conservative treatment group, baseline leptin levels were also elevated

Table. Circulating ghrelin and leptin levels and body mass index in healthy volunteers and patients before and after surgical and therapeutic treatment

Group	Ghrelin, ng/ml	Leptin, ng/ml	BMI, kg/m ²
Healthy volunteers	21.2 [5.44; 22.7]	0.8 [0.22; 1.5]	21 [18; 25]
Before surgery	12.21 [0.83; 21.15]****	1.63 [0.6; 8.6]****	26 [18; 35]****
After surgery	19.8 [14.8; 31.5]####	0.97 [0.2; 6.4]##	24 [18; 32]#
Before therapy	14.6 [3.9; 33.4]**	1.8 [0.7; 6.8]****	27 [17; 33]****
After therapy	18.4 [4.0; 34.7]	1.6 [0.3; 7.5]	25 [16; 34]

Notes: 1. * – Statistically significant differences compared with the healthy volunteers and before surgery and therapy groups.
2. # – Statistically significant differences compared with the before and after surgery groups.

relative to controls at 1.8 ng/ml [0.7; 6.8] ($p < 0.001$). Following therapy, leptin remained relatively high at 1.6 ng/ml [0.3; 7.5], with no significant reduction observed (Table).

BMI was significantly higher in patients prior to surgery compared with healthy participants at 26 kg/m² [18; 35] ($p < 0.001$). After surgical treatment, BMI decreased significantly to 24 kg/m² [18; 32] ($p < 0.05$ vs preoperative values) but remained above control levels. A similar pattern was observed in the conservative treatment group, where BMI was 27 kg/m² [17; 33] before therapy ($p < 0.001$ vs controls) and showed no significant reduction after treatment at 25 kg/m² [16; 34] (Table).

Overall, patients were characterized by elevated BMI and hyperleptinemia accompanied by reduced ghrelin concentrations prior to treatment. Surgical intervention was associated with pronounced metabolic changes, including a significant increase in ghrelin together with reductions in leptin and BMI. In contrast, conservative therapy did not produce comparable hormonal or metabolic shifts.

Correlation analysis across the study groups demonstrated distinct association patterns. In healthy volunteers, a strong positive correlation was observed between leptin levels and BMI ($r = 0.666$, $p = 0.001$). Correlations between ghrelin and leptin ($r = -0.373$, $p = 0.105$) and between ghrelin and BMI ($r = -0.296$, $p = 0.206$) were not statistically significant (Figure 1, A).

Among patients prior to surgery, a moderate positive association between leptin and BMI was identified ($r = 0.511$, $p = 0.0017$), whereas correlations involving ghrelin remained nonsignificant, including ghrelin with leptin ($r = 0.076$, $p = 0.666$) and ghrelin with BMI ($r = -0.136$, $p = 0.435$) (Figure 1, B).

Following surgical treatment, the moderate positive leptin–BMI relationship persisted ($r = 0.485$, $p = 0.003$). However, correlations between ghrelin and

leptin ($r = 0.232$, $p = 0.179$) and between ghrelin and BMI ($r = -0.080$, $p = 0.650$) remained nonsignificant (Figure 1, C).

Before conservative therapy, leptin and BMI were also moderately positively correlated ($r = 0.487$, $p = 0.018$) (Figure 1, D), while ghrelin showed no significant associations with leptin ($r = 0.193$, $p = 0.377$) or BMI ($r = -0.235$, $p = 0.280$).

After therapy, a moderate positive correlation between leptin and BMI was maintained ($r = 0.499$, $p = 0.015$) (Figure 1, E). Additionally, a moderate inverse correlation emerged between ghrelin and BMI ($r = -0.508$, $p = 0.013$) (Figure 1, F), whereas the ghrelin–leptin correlation remained nonsignificant ($r = 0.034$, $p = 0.878$).

Discussion. The aim of the present study was to investigate leptin and ghrelin levels in patients with gastroesophageal reflux disease and to evaluate their dynamics depending on the selected treatment strategy. The conducted analysis allowed not only the characterization of changes in the concentrations of these hormones but also the determination of their relationship with fat mass indicators and body mass index.

Significant differences in the concentrations of the metabolic hormones leptin and ghrelin, as well as BMI, were identified in patients with GERD compared with healthy volunteers. Changes in these parameters were also observed following surgical and therapeutic treatment. These findings are consistent with well described mechanisms of endocrine regulation of energy homeostasis.

A positive correlation between leptin concentration and BMI was established across all groups, reflecting the fundamental physiological relationship between fat mass and leptin secretion. Leptin is the principal adipokine synthesized by adipocytes, and its concentration correlates with adipose tissue volume, as demonstrated in classical studies in which leptin

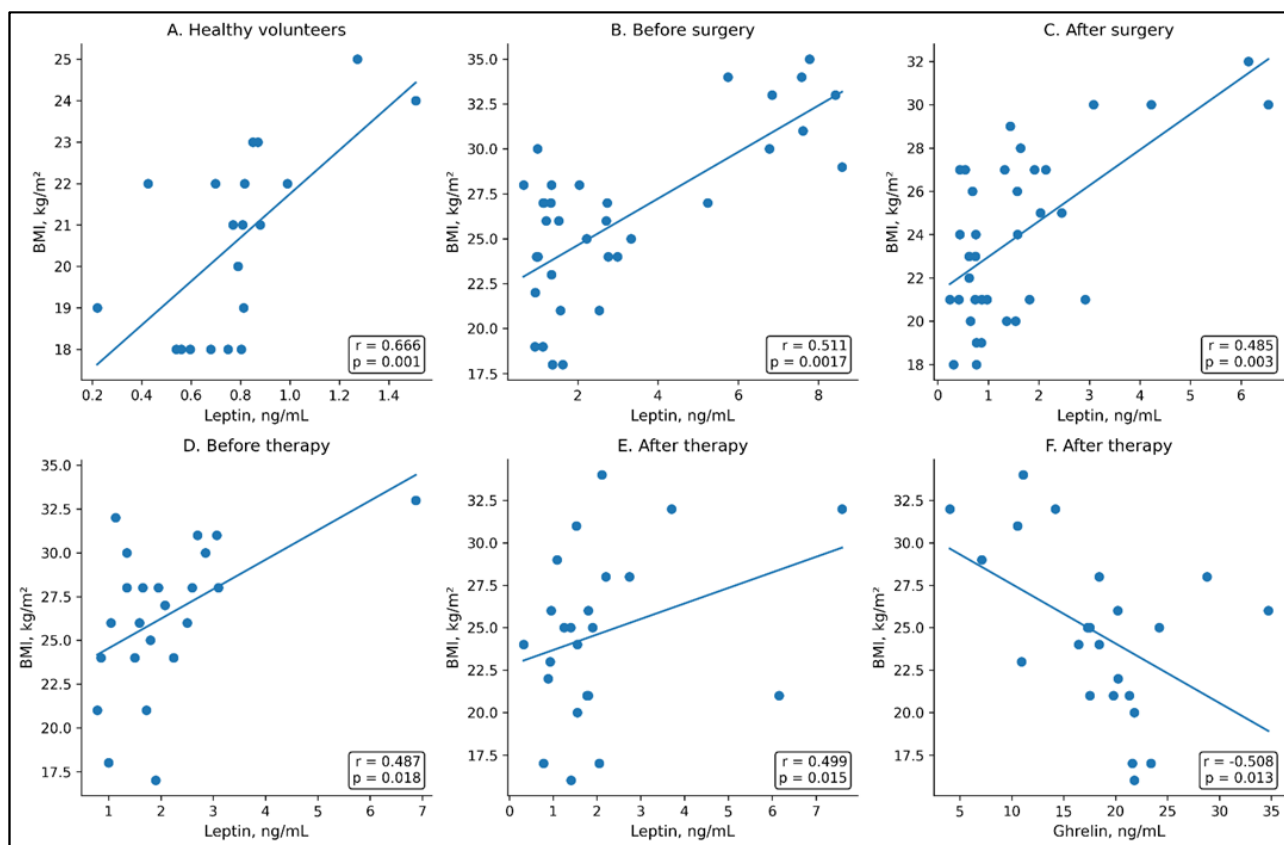


Figure 1. Correlation analysis between metabolic hormones and body mass index across study groups. Panels A – E demonstrate significant positive correlations between leptin levels and BMI in healthy individuals, before and after surgery, and before and after conservative therapy. Panel F shows a moderate inverse correlation between ghrelin and BMI after therapy. Other correlations involving ghrelin were not statistically significant and are not shown.

increased linearly with rising BMI regardless of weight category including normal weight, overweight, and obesity. These observations are supported by a large study showing that leptin increases while ghrelin decreases with increasing BMI, highlighting the close association between leptin and body fat mass and its role in metabolism [7].

Additionally, patients prior to surgical intervention and before the initiation of therapy exhibited elevated leptin levels and reduced ghrelin levels compared with controls. These hormonal shifts are characteristic of conditions associated with increased fat mass and impaired energy balance. It is well established that elevated leptin in obesity reflects increased adipocyte mass, whereas ghrelin is typically reduced, which is consistent with the concept of compensation for decreased sensitivity to this hormone during metabolic stress [8].

Following surgical intervention, a significant increase in ghrelin levels compared with the preoperative state was observed, whereas leptin concentrations decreased. These changes in hormonal

profiles reflect metabolic adaptation in response to weight reduction and are consistent with literature reporting increased ghrelin alongside decreased leptin after surgical procedures, including bariatric interventions [9]. Moreover, surgical interventions on the stomach have been shown to be associated with alterations in hormones involved in appetite regulation, including ghrelin and leptin. These changes are considered one possible explanation for the metabolic effects of surgical treatment [4, 10, 11].

Furthermore, a moderate inverse correlation between ghrelin levels and BMI was identified after therapeutic treatment, which may reflect partial restoration of the physiologically expected relationship between the hunger hormone and body weight parameters. Similar phenomena have been described in studies where changes in ghrelin were associated with weight dynamics during non-surgical treatment, emphasizing the importance of ghrelin as a marker of short-term energy homeostasis [12].

Our results also demonstrate that leptin levels are closely associated with the amount of adipose tissue

and remain a relatively stable parameter across all studied groups. In contrast, ghrelin concentrations exhibit greater variability and respond more rapidly to surgical and therapeutic interventions. These differences are well explained by the physiology of these hormones. Leptin is produced by adipocytes, and therefore its level is primarily determined by adipose tissue volume and changes gradually [13]. Ghrelin, which is predominantly synthesized in the stomach, participates in appetite regulation and can change rapidly depending on nutritional status, body weight, and the functional state of the gastrointestinal tract and after surgical intervention on the gastric fundus [14]. Consequently, leptin reflects long term metabolic characteristics, whereas ghrelin is more sensitive to current metabolic alterations and may respond more quickly to treatment [15].

Finally, correlation analysis demonstrated a persistent association between leptin and BMI across all study groups, underscoring the intergroup stability of metabolic mechanisms regulating fat mass. At the same time, the contribution of ghrelin to body weight regulation appeared more context dependent, varying according to treatment stage and the patient's energy state. This multidimensional regulation is consistent with contemporary concepts of hormonal appetite control, in which leptin and ghrelin function as components of an integrated yet complex signaling network influencing energy homeostasis, body weight, and metabolic adaptation to weight change [5, 16].

Conclusions. The present study demonstrated that patients with gastroesophageal reflux disease exhibit an altered hormonal profile characterized by elevated leptin and reduced ghrelin levels, suggesting a potential link between the disease and metabolic disturbances. The consistent association between leptin and body mass index across all groups confirms its key role as a marker of adipose tissue, whereas the greater variability of ghrelin indicates its sensitivity to changes in energy balance and to the effects of treatment. The more pronounced hormonal dynamics observed after surgical intervention compared with therapeutic treatment suggest that antireflux surgery may influence not only reflux mechanisms but also metabolic regulation. Overall, these findings support the relevance of considering appetite regulating hormones in the investigation of the pathophysiology of gastroesophageal reflux disease and in the evaluation of treatment effectiveness.

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

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Authors' contribution. Yevhen Haidarzhi – research concept and design, collection and assembly of data, data analysis and interpretation, writing the article, final approval of the article. Sergii Pavlov – collection and assembly of data, data analysis and interpretation, critical revision of the article, final approval of the article. Mykola Golovko – critical revision of the article, final approval of the article.

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Динаміка рівнів гормонів харчової поведінки лептину та греліну в пацієнтів із гастроєзофагеальною рефлюксною хворобою залежно від стратегії лікування

Мета роботи: оцінити рівні лептину та греліну в пацієнтів із гастроєзофагеальною рефлюксною хворобою, проаналізувати динаміку та визначити їх зв'язок з індексом маси тіла.

Матеріали і методи. Дослідження включало обстеження та лікування 90 пацієнтів із гастроєзофагеальною рефлюксною хворобою (ГЕРХ). Лапароскопічну тотальну фундоплекіацію у модифікації за методикою Short Floppy Nissen було проведено 35 хворим. Медикаментозну терапію призначили 35 пацієнтам. Групу порівняння склали 20 здорових добровольців. Групи пацієнтів та здорових людей із контрольної групи були зіставні за статтю та віком. Венозну кров для дослідження збирали у хворих із групи порівняння та в групах до та після хірургічного або терапевтичного лікування, а також у всіх пацієнтів через 2–3 місяці після лікування. Грелін (Ghrelin, ELISA, BMS2192, Ebioscience) та лептин (Leptin, Sensitiv ELISA, KAC2281, Ebioscience) тестували на імуноферментному комплексі ImmunoChem-2100 (США).

Результати. Медіанний рівень греліну в здорових учасників становив 21,2 нг/мл [5,44; 22,7], лептину – 0,8 нг/мл [0,22; 1,5], а індекс маси тіла (ІМТ) – 21 кг/м² [18; 25]. У здорових добровольців спостерігалася сильна позитивна кореляція між рівнем лептину та ІМТ ($r=0,666$, $p=0,001$). Кореляція між греліном та лептином ($r=-0,373$, $p=0,105$) та між греліном й ІМТ ($r=-0,296$, $p=0,206$) не була статистично значущою. У пацієнтів із ГЕРХ, порівняно зі здоровими добровольцями, було виявлено значні відмінності в концентраціях метаболічних гормонів лептину та греліну, а також в ІМТ. Зміни цих параметрів також спостерігалися після хірургічного та терапевтичного лікувань. Позитивна кореляція між концентрацією лептину та ІМТ була встановлена в усіх групах. У пацієнтів до хірургічного втручання та до початку терапії спостерігався підвищений рівень лептину та знижений рівень греліну порівняно з контрольною групою. Після хірургічного втручання було значне підвищення рівня греліну, порівняно з доопераційним станом, тоді як концентрація лептину знизилася. Після терапевтичного лікування було виявлено помірну негативну кореляцію між рівнем греліну та ІМТ. Кореляційний аналіз продемонстрував стійкий зв'язок між лептином та ІМТ у всіх дослідних групах. Внесок греліну в регуляцію маси тіла виявився більш залежним від контексту, змінюючись залежно від стадії лікування та енергетичного стану пацієнта.

Висновки. У пацієнтів із ГЕРХ спостерігається підвищений рівень лептину та знижений рівень греліну. Постійний зв'язок між лептином та ІМТ в усіх групах підтверджує його ключову роль як маркера жирової тканини, тоді як більша мінливість греліну вказує на його чутливість до змін енергетичного балансу та до впливу лікування. Більш виражена гормональна динаміка, що спостерігається після хірургічного втручання, порівняно з терапевтичним лікуванням, свідчить про те, що антирефлюксна хірургія може впливати не лише на механізми рефлюксу, але й на метаболічну регуляцію.

Ключові слова: гастроєзофагеальна рефлюксна хвороба; лапароскопічна фундоплекіація; харчова поведінка; грелін; лептин.

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