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Modulation of appetite- and metabolism-regulating hormones after bariatric embolization, sleeve gastrectomy, and gastric plication

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Objective. The obesity is associated with profound disturbances in appetite-regulating and metabolic hormones including ghrelin, leptin, adiponectin, resistin, and insulin. Bariatric arterial embolization (BEA) has recently emerged as a minimally invasive alternative to established bariatric procedures. However, comparative endocrine evidence remains limited. The aim of the study was to assess postoperative changes in key metabolic and appetite-regulating hormones following laparoscopic sleeve gastrectomy (LSG), laparoscopic gastric plication (LGP), and bariatric embolization of the gastric arteries (BEA).

Methods. A total of 76 patients with obesity (BMI >35 kg/m^2) were assigned to LSG (n=32), LGP (n=37), or BEA (n=7). Serum concentrations of total ghrelin, leptin, adiponectin, resistin, insulin, and HbA1c were measured preoperatively 3 and 6 months after intervention using standardized ELISA assays. Statistical significance was set at $p<0.05$.

Results. All procedures resulted in significant improvements in hormonal and metabolic profiles within 6 months. Total ghrelin decreased by 55.6% after LGP, 69.7% after LSG, and 74.5% after BEA at 6 months (all $p<0.001$). Leptin significantly declined, while adiponectin increased in all groups with the most pronounced early (3-month) rise after BEA (+36.1%, $p=0.033$). Resistin, insulin, and HbA1c progressively decreased across all interventions indicating improved insulin sensitivity. Overall, BEA demonstrated the strongest early hormonal response, whereas long-term (6-month) effects were comparable across procedures.

Conclusion. Laparoscopic gastric plication, sleeve gastrectomy, and bariatric embolization provide substantial improvements in appetite-regulating and metabolic hormones in patients with obesity. BEA shows a particularly favorable early endocrine profile supporting its potential as a minimally invasive metabolic intervention.

Keywords: obesity, bariatric embolization, sleeve gastrectomy, gastric plication, ghrelin, leptin, adiponectin, resistin, insulin, metabolic hormones

Patients with obesity commonly exhibit disturbances in hormone secretion and metabolic homeostasis, among which ghrelin and leptin are considered the most relevant indicators. These two hormones play key and complementary roles in the regulation of energy balance. Leptin is a major mediator of long-term energy homeostasis suppressing the food intake and promoting weight loss (Friedman 2019). In contrast, ghrelin is a rapidly acting orexigenic hormone involved in the

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initiation of meals, enhancement of gastrointestinal motility, stimulation of hunger, and increased caloric consumption (Ibrahim Abdalla 2015).

With the global prevalence of obesity, the steadily rising understanding the mechanisms, by which hormones and neuroendocrine pathways influence energy balance has become a central focus of current research. In individuals with obesity, the circulating levels of the anorexigenic hormone leptin are paradoxically elevated, whereas the orexigenic hormone ghrelin levels are reduced. It is now well established that obese patients develop leptin resistance, which largely underlies this paradox and contributes to dysregulated appetite control (Montegut et al. 2021).

From adipose tissue-derived factors, adiponectin and resistin have recently gained attention as important modulators of metabolic and inflammatory processes. Adiponectin, secreted predominantly by adipocytes, circulates at reduced levels in individuals with obesity and type 2 diabetes mellitus (T2DM). It enhances fatty-acid oxidation, lowers plasma triglycerides, and improves glucose metabolism by increasing insulin sensitivity. Moreover, adiponectin exerts anti-inflammatory and potentially anti-atherogenic effects by inhibiting monocyte/macrophage migration and foam-cell formation. Reduced adiponectin concentrations are consistently observed in patients with coronary artery disease irrespective of body mass index (BMI) (Labounty et al. 2013).

Resistin, a member of the cysteine-rich secretory protein family known as resistin-like molecules (RELMs) or "found in inflammatory zone" (FIZZ) proteins, has been implicated in insulin resistance and obesity-related inflammation. Together with FIZZ1/RELMα and FIZZ2/RELMβ resistin is thought to contribute to the complex metabolic and immune dysregulation that characterizes obesity (Beltowski 2003).

Against this complex endocrine background, various bariatric procedures have been developed to induce weight loss and improve metabolic outcomes. In recent years, bariatric arterial embolization most commonly left gastric artery embolization aimed at modulating ghrelin-producing cells of the gastric fundus has emerged as a minimally invasive endovascular alternative to conventional bariatric surgery. Although its prevalence remains substantially lower than established procedures such as sleeve gastrectomy or Roux-en-Y gastric bypass, bariatric embolization is gaining attention due to its favorable safety profile, reduced procedural invasiveness, and potential applicability in patients who are not candidates for major abdominal surgery (Sangiorgi et al. 2021).

Despite promising early results regarding weight reduction and appetite suppression, the endocrine and metabolic mechanisms underlying the effects of bariatric embolization remain insufficiently understood. In particular, data on its postoperative influence on key hormonal regulators ghrelin, leptin, adiponectin, and resistin are limited and inconsistent. Given the central role of these hormones in energy homeostasis, glucose metabolism, and inflammatory pathways, evaluating their dynamics after bariatric embolization is critical for understanding the physiological basis of this intervention and for optimizing patient selection predicting metabolic outcomes and improving long-term efficacy.

The aim of this study was to evaluate the postoperative dynamics of key metabolic and appetite-regulating hormones ghrelin, leptin, adiponectin, and resistin following bariatric arterial embolization.

Materials and Methods

Study participants. The ethical principles included in the Declaration of Human Rights adopted in Helsinki in 1975 and revised in 2008, were fully respected in this study. The enrolled subjects participated in this study voluntarily and with completed and signed written informed consent. Study protocol was approved by the Ethics Committee (Protocol No. 80) of I. Horbachevsky Ternopil National Medical University of November 15, 2024.

The study included 76 individuals with obesity presenting clinical signs of metabolic syndrome (BMI>35 kg/m^2) and 48 subjects with normal body weight (BMI 18–25 kg/m^2). The comparison groups were formed to ensure similar distributions of age and sex with no statistically significant differences between them ($p>0.05$).

Anthropometric measurements like body weight, height, BMI, and waist-to-hip parameters were obtained using unified methodological standards. In the obese cohort, the mean body weight was 121.87±7.23 kg corresponding to an average BMI of 42.57±2.52 kg/m^2, which was markedly elevated relative to the control population ($p<0.001$). Obesity severity was determined in accordance with the IFSO (The International Federation for the Surgery of Obesity and Metabolic Disorders) categorization with class III obesity representing the predominant subgroup.

Surgical procedures. Patients were allocated into three groups according to the type of bariatric

intervention performed: laparoscopic sleeve gastrectomy (LSG, n=32), laparoscopic gastric plication (LGP, n=37), and bariatric embolization of the gastric arteries (BEA, n=7).

Laparoscopic sleeve gastrectomy (LSG). After standard abdominal preparation and establishment of pneumoperitoneum (15–17 mmHg), the stomach was mobilized from the pyloric region toward the angle of His. A 34 Fr calibration tube was introduced along the lesser curvature guiding the creation of a narrow gastric sleeve using endoscopic staplers (45/60 mm cartridges, 4.8 mm staples). The resulting gastric conduit had an approximate capacity of 120–150 mL. To reinforce the staple line, a continuous seromuscular over-sewing (V-Loc) was performed.

Laparoscopic gastric plication (LGP). Once pneumoperitoneum was achieved (15 mmHg), the greater curvature was fully mobilized. Gastric volume reduction was accomplished by placing inverting, non-resectional sutures (V-Loc 2.0), beginning 2–3 cm proximal to the pylorus and extending toward the fundus. One or several rows of sutures were applied creating a narrowed intragastric lumen with an estimated volume of 100–150 mL.

Bariatric embolization of the gastric arteries (BEA). Prior to intervention, CT angiography was performed to rule out variations in gastric vascular anatomy. Through transfemoral access, selective catheterization of the left gastric artery was carried out followed by embolization with 300–500 μm microspheres (Embosphere, Merit Medical) delivered via a high–flow microcatheter. Technical success was confirmed by post-embolization perfusion assessment. All patients received postoperative proton pump inhibitor therapy and were scheduled for endoscopic evaluation at 1 and 3 months after the procedure.

Laboratory data. Blood samples were collected in the morning after an overnight fast. The plasma glycated hemoglobin (HbA1c) concentration was determined using commercial assay kits (Roche Diagnostics) on a Hitachi automated analyzer. Serum concentrations of selected hormones were measured using the following ELISA kits: Leptin (LDN Labor Diagnostics Nord GmbH & Co. KG, Germany), Human Ghrelin (Thermo Fisher Scientific, USA), Human Adiponectin (Thermo Fisher Scientific, USA), OxLDL (Thermo Fisher Scientific, USA), and Resistin (Human ELISA Kit, Thermo Fisher Scientific, USA). All assays were performed on the Multiskan FC microplate analyzer (SkanIt Software, version 4.1.0.43) at a wavelength of 620 nm.

Statistics. Statistical comparison of surgical outcomes (BMI, TWL, EWL hormone levels) among LSG, LGP, and GAE groups was performed using one-way ANOVA or Kruskal-Wallis test, depending on data distribution. Paired t-test or Wilcoxon signed-rank test was used for within-group pre/post comparisons. Categorical variables were analyzed using the chi-square test and $p<0.05$ was considered statistically significant.

Results

The indicators of orexigenic and anorexigenic hormone imbalance as well as the metabolic syndrome markers were analyzed in patients prior to different types of bariatric procedures depending on obesity grade (Tables 1–3).

Ghrelin dynamics. Analysis of total ghrelin concentrations (ng/mL) at 3 and 6 months postoperatively demonstrated a significant decline in all three study groups (Figure 1). After LGP, ghrelin decreased by 41.03% at 3 months ($p<0.001$) from 747.31 to 440.72 ng/mL and by 55.62% at 6 months ($p<0.001$). Following LSG, total ghrelin decreased by 50.61% at 3 months ($p<0.001$) and by 69.73% at 6 months ($p<0.001$). The most pronounced reduction was observed after BEA, by 59.92% at 3 months ($p<0.001$) and by 74.49% at 6 months ($p<0.001$) (Figure 1).

Leptin dynamics. Postoperative serum leptin levels (ng/mL) showed a significant decline across all groups (Figure 2). After LGP, leptin decreased by 10.61% at 3 months ($p>0.05$) and by 31.04% at 6 months ($p=0.032$). Following LSG, leptin decreased by 43.49% at 3 months ($p=0.0005$) and by 47.73% at 6 months ($p=0.0001$). In the BEA group, a reduction of 17.18% was observed after 3 months ($p>0.05$) and 24.72% after 6 months ($p=0.0280$).

Adiponectin dynamics. Serum adiponectin (μg/mL) increased progressively at 3 and 6 months in all

Table 1
Preoperative hormonal markers of metabolic syndrome before laparoscopic gastric plication (LGP) in patients with grade I obesity

Indicators	LGP (n=5)
HbA1c (%)	5.54±0.21
Leptin (ng/mL)	18.28±2.71
Total ghrelin (ng/mL)	1157.64±10.34
Adiponectin (μg/mL)	7.74±1,02
Resistin (ng/mL)	7.76±0.97
Insulin (μIU/mL)	21.36±3.21

Table 2
Preoperative hormonal markers before laparoscopic gastric plication (LGP), laparoscopic sleeve gastrectomy (LSG), and bariatric embolization of the gastric arteries (BEA) in patients with grade II obesity

Indicators	LGP (n=13)	LSG (n=8)	BEA (n=7)	p-value
HbA1c (%)	6.38±0.14	5.94±0.12	5.97±0.15	0.754
Leptin (ng/mL)	27.58±3.21	31.57±4.03	32.08±3.67	0.778
Total ghrelin (ng/mL)	718.02±8.65	911.29±12.6	784.09±8.87	0.859
Adiponectin (μg/mL)	6.35±0.98	6.81±0.67	6.51±0.84	0.845
Resistin (ng/mL)	8.32±0.65	8.36±0.56	8.19±0.77	0.889
Insulin (μIU/mL)	23.43±2.78	26.45±3.14	26.11±3.42	0.847

groups (Figure 3). After LGP, adiponectin increased by 29.52% at 3 months (p>0.05) and by 39.15% at 6 months (p=0.0176). After LSG, the increase was 32.59% at 3 months (p>0.05) and 43.05% at 6 months (p=0.007). BEA resulted in the most favorable early dynamics, 36.05% at 3 months (p=0.033) and 40.0% at 6 months (p=0.015).

Resistin dynamics. Serum resistin (ng/mL) declined after all procedures (Figure 4). After LGP, the reductions were 11.67% at 3 months (p>0.05) and 20.95% at 6 months (p=0.0002). After LSG, resistin decreased by 12.35% at 3 months (p=0.0178) and by 21.72% at 6 months (p=0.002). Following BEA, reductions were 5.62% at 3 months (p>0.05) and 13.19% at 6 months (p=0.0173).

Insulin dynamics and insulin resistance. A significant reduction in insulin levels (μIU/mL) was observed at both time points in all groups indicating improvement of insulin resistance (Figure 5). After LGP, insulin decreased by 26.69% at 3 months (p=0.0001) and by 38.97% at 6 months (p<0.001). After LSG, the reductions were 21.94% at 3 months (p<0.001) and 39.54% at 6 months (p<0.001). BEA resulted in decreases of 21.45% at 3 months (p=0.0098) and 28.07% at 6 months (p=0.0015).

HbA1c dynamics. Consistent with the improvements in leptin and insulin sensitivity, a significant decrease in glycated hemoglobin was observed (Figure 6). After LGP, HbA1c declined by 11.17% at 3 months (p=0.0305) and by 19.05% at 6 months (p=0.0008). After LSG, the reductions were 6.55% at 3 months (p>0.05) and 12.60% at 6 months (p=0.0012). BEA resulted in decreases of 5.53% at 3 months (p>0.05) and 11.73% at 6 months (p=0.0037).

A significant improvement in metabolic syndrome-related biochemical markers was observed 6 months after all three types of bariatric interventions. The trajectories of hormonal and metabolic changes were generally comparable between laparoscopic procedures and BEA. Based on the magnitude of reduction in leptin and total ghrelin and the increase

Table 3
Preoperative hormonal markers before laparoscopic gastric plication (LGP) and laparoscopic sleeve gastrectomy (LSG) in patients with grade III obesity

Indicators	LGP (n=19)	LSG (n=24)	p-value
HbA1c (%)	6.03±0.09	6.18±0.11	0.882
Leptin (ng/mL)	56.74±2.67	55.77±3.01	0.756
Total ghrelin (ng/mL)	659.38±5.78	650.96±6.23	0.849
Adiponectin (μg/mL)	5.86±0.56	5.81±0.47	0.892
Resistin (ng/mL)	8.63±0.72	9.33±0.88	0.889
Insulin (μIU/mL)	28.45±2.98	29.61±3.3	0.854

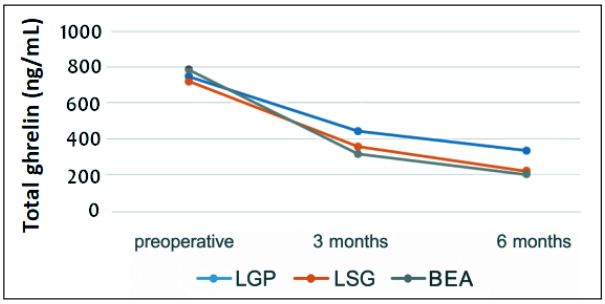

Figure 1. Response of total ghrelin (ng/mL) to bariatric procedures, laparoscopic gastric plication (LGP), laparoscopic sleeve gastrectomy (LSG), and bariatric embolization of the gastric arteries (BEA) 3 and 6 months after these procedures.

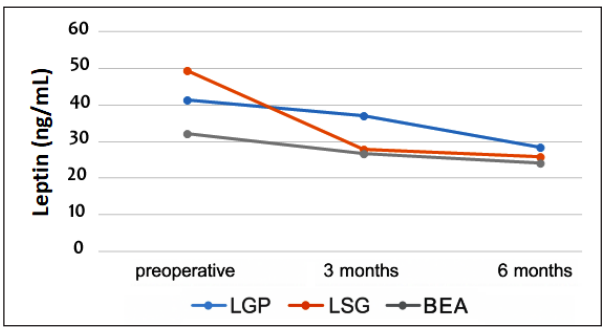

Figure 2. Response of leptin (ng/mL) to different bariatric procedures, laparoscopic gastric plication (LGP), laparoscopic sleeve gastrectomy (LSG), and bariatric embolization of the gastric arteries (BEA) 3 and 6 months after these procedures.

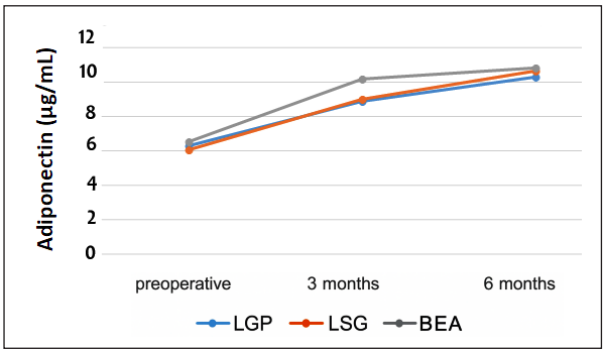

Figure 3. Response of adiponectin (µg/mL) to different bariatric procedures, laparoscopic gastric plication (LGP), laparoscopic sleeve gastrectomy (LSG), and bariatric embolization of the gastric arteries (BEA) 3 and 6 months after these procedures.

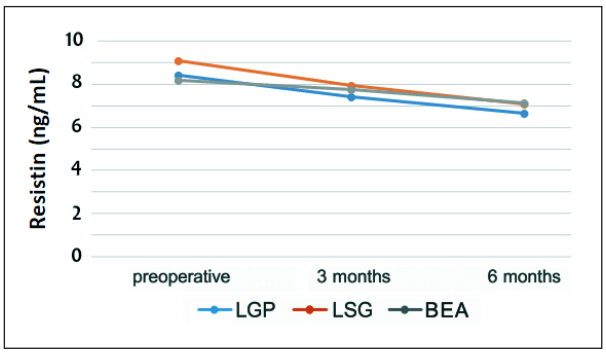

Figure 4. Response of resistin (ng/mL) to different bariatric procedures, laparoscopic gastric plication (LGP), laparoscopic sleeve gastrectomy (LSG), and bariatric embolization of the gastric arteries (BEA) 3 and 6 months after these procedures.

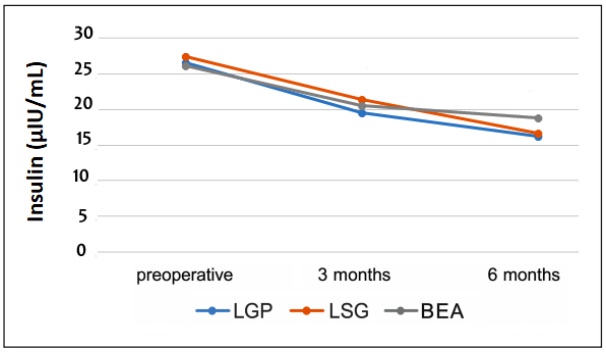

Figure 5. Response of insulin (µIU/mL) to different bariatric procedures, laparoscopic gastric plication (LGP), laparoscopic sleeve gastrectomy (LSG), and bariatric embolization of the gastric arteries (BEA) 3 and 6 months after these procedures.

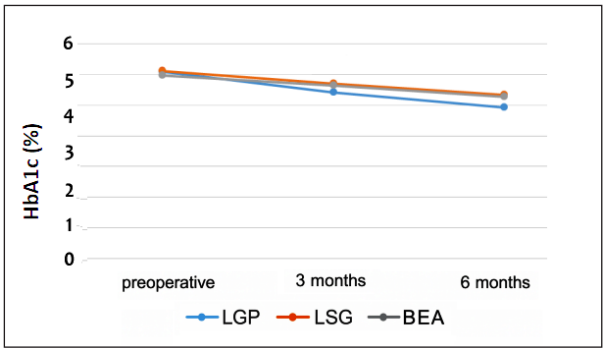

Figure 6. Response of glycated hemoglobin (HbA1c) (%) to different bariatric procedures, laparoscopic gastric plication (LGP), laparoscopic sleeve gastrectomy (LSG), and bariatric embolization of the gastric arteries (BEA) 3 and 6 months after these procedures.

in adiponectin, BEA demonstrated the most favorable early (3-month) hormonal response ($p<0.001$).

Discussion

The present study demonstrates that LSG, LGP, and BEA all produced substantial improvements in appetite-regulating and metabolic hormones within 6 months. These findings are broadly consistent with previous evidence that bariatric interventions modulate orexigenic and anorexigenic pathways and improve metabolic homeostasis.

The marked reduction in total ghrelin observed after LSG and LGP corresponds closely with earlier studies reporting suppression of fundus-derived ghrelin following restrictive procedures. Sethi et al. (2018) have described significant long-term reductions in ghrelin after LSG, which were associated with sustained weight loss and appetite suppression supporting the pattern observed in our cohort. More recent physiological investigations have further confirmed the regulatory interaction between ghrelin and post-operative glucose tolerance, gut hormones, and appetite control. Hedback et al. (2024) have demonstrated that suppression of ghrelin after LSG contributes to improved incretin secretion and satiety, which aligns with the magnitude of hormonal changes seen in our study.

The endocrine effects of BEA observed in our cohort, particularly the rapid and pronounced decline in ghrelin levels, are consistent with early interventional findings from the GET LEAN trial, in which left gastric artery embolization resulted in clinically meaningful appetite suppression and significant reductions in circulating ghrelin (Syed et al. 2016). Together, these findings support the hypothesis that BEA exerts its weight-reducing effects, at least in part, through modulation of appetite-regulating

hormones. These comparable patterns support the hypothesis that BEA mimics the endocrine effects of fundal modification achieved surgically, while providing a less invasive alternative.

Changes in leptin observed in the present study also reflect previously published findings. Leptin levels typically decline with postoperative reductions in adiposity and improved leptin signaling. Nozari et al. (2023) have demonstrated that improvements in leptin sensitivity after bariatric surgery correlate not only with weight loss, but also with broader metabolic and neurocognitive benefits. Similar declines in leptin across LGP, LSG, and BEA in our cohort suggest parallel improvements in adipose-tissue function.

The increase in adiponectin and decline in resistin observed across all bariatric procedures are consistent with recent adipokine-focused analyses. Several studies including those by Sebunova et al. (2022) and Fiorotti et al. (2024) have documented that rising adiponectin and falling resistin are reliable markers of enhanced metabolic and cardiometabolic health after bariatric surgery. The trajectories identified in our study fit well within these established patterns reinforcing the role of adipokines as dynamic biomarkers of metabolic recovery.

Furthermore, the interplay among ghrelin, leptin, and insulin observed in our cohort echoes the regulatory networks described by Skuratovskaia et al. (2021) who have demonstrated that postoperative hormonal shifts involve coordinated changes in incretins, glucagon, and insulin. This supports the interpretation that improvements in insulin and HbA1c in our patients reflect not only the weight reduction, but also an integrated endocrine response to bariatric intervention.

Recent systematic reviews including the metabolomics-focused analysis by Vaz et al. (2021) have emphasized that bariatric procedures trigger broad metabolic remodeling involving lipid oxidation, inflammation, and insulin sensitivity pathways. The results of our study align with this evolving evidence demonstrating parallel improvements across multiple metabolic biomarkers regardless of intervention type.

Notably, BEA displayed the strongest early endocrine response among the three modalities examined. Given the limited number of comparative studies directly evaluating BEA against surgical techniques, our findings contribute novel evidence supporting its endocrine efficacy. However, the smaller sample size in the BEA group warrants caution and larger controlled trials are necessary to validate these patterns and assess long-term durability.

Conclusion

Bariatric interventions including laparoscopic gastric plication, sleeve gastrectomy and bariatric embolization of the gastric arteries led to significant improvements in orexigenic and anorexigenic hormone balance, insulin sensitivity, and metabolic syndrome-related biochemical markers within 6 months of follow-up. All procedures were associated with a consistent reduction in total ghrelin, leptin, resistin, insulin, and HbA1c, accompanied by an increase in adiponectin levels. Although the overall metabolic trajectories were comparable across surgical and endovascular techniques, bariatric embolization demonstrated the most pronounced early (3-month) hormonal response characterized by greater reduction in ghrelin and leptin and a more rapid increase in adiponectin. These findings support the effectiveness of both surgical and minimally invasive approaches in improving endocrine-metabolic regulation in patients with obesity and highlight bariatric embolization as a promising modality for early modulation of appetite-regulating hormones.

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Conflict of interest: *The authors declare no conflict of interest.*

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