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Secondary hyperparathyroidism and vitamin D deficiency in patients after laparoscopic sleeve gastrectomy

The purpose of this study is to investigate the vitamin D deficiency and secondary hyperparathyroidism (SHPT) in patients after laparoscopic sleeve gastrectomy (LSG). The study is based on a review of medical records and clinical data collected from patients with morbid obesity after laparoscopic sleeve gastrectomy.

The materials and methods. A total of 79 patients (without SHPT at the baseline) after LSG were included in the study, with a mean age of 40,7±11,4 years. All patients were divided into 2 groups depending on the receipt of additional vitamin and mineral therapy in the postoperative period. The first group (n=38) who did not receive additional therapy in the postoperative period. The second group (n=41) who received vitamins and trace elements (cholecalciferol 2 000 IU, calcium 1,000 mg and a multivitamin 1 tablet per day). Anthropometric parameters, ionized calcium (Ca2+), 25 hydroxycalciferol (25(OH)D), parathyroid hormone (PTH), were compared before and 1 year after LSG. The prevalence of vitamin D deficiency (< 20 ng/ml) and secondary hyperparathyroidism (> 88 pg/mL) at different time points was calculated.

Results. The median 25(OH)D levels in both groups were within normal limits, but in the first group the median was statistically significantly lower compared to the median of the same indicator in the second group (p=0.001). The number of cases of vitamin D hypovitaminosis was statistically significantly higher in the first group, 17 vs 5 patients, respectively (p=0.003). The overall prevalence of SHPT was recorded in 14 (17.7%) patients included in this study, with a statistically significant majority of cases in the first group 12 vs. 2 (p=0.005) patients, respectively. Post-operatively, PTH levels had a strong significant inverse relationship with 25(OH)D and Ca2+ levels (p<0.001).

Conclusion. Patients after laparoscopic sleeve gastrectomy have a risk of secondary hyperparathyroidism and vitamin D deficiency in the postoperative period. The higher levels of parathyroid hormone post laparoscopic sleeve gastrectomy significantly related to vitamin D deficiency and lower calcium levels. Additional vitamin and mineral therapy in the postoperative period after laparoscopic sleeve gastrectomy significantly reduces the risk of developing secondary hyperparathyroidism and vitamin D deficiency. This study also emphasizes the importance of routine testing for hyperparathyroidism and vitamin deficiency after laparoscopic sleeve gastrectomy.

Key words: Bariatric surgery, obesity, laparoscopic sleeve gastrectomy, ionized calcium, 25 hydroxycalciferol, parathyroid hormone, vitamin D deficiency, secondary hyperparathyroidism.

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Вторинний гіперпаратиреоз та недостатність вітаміну D у пацієнтів після лапароскопічної рукавної резекції шлунка

Мета дослідження. Вивчити недостатність вітаміну D та вторинний гіперпаратиреоз (ВГПТ) у пацієнтів після лапароскопічної рукавної резекції шлунка (ЛРРШ). Дослідження ґрунтується на аналізі медичної документації та клінічних даних зібраних у пацієнтів із морбідним ожирінням після лапароскопічної рукавної резекції шлунка.

Матеріали та методи. В дане дослідження було включено 79 пацієнтів (без ВГПТ до операції) після ЛРРШ, середній вік яких становив 40,7±11,4 років. Всі пацієнтки були розподілені на 2 групи залежно від отримання додаткової вітамінно-мінеральної терапії в післяопераційному періоді. Перша група (n=38), які не отримували додаткової терапії в післяопераційному періоді. Друга група (n=41), які отримували вітаміни та мікроелементи (холекальциферол 2 000 МО, кальцій 1 000 мг та полівітамінний препарат 1 таблетка на добу). Антропометричні показники, рівень іонізованого кальцію (Ca2+), 25-гідроксикальциферолу (25(OH)D), паратгормону (ПТГ) аналізували до та через 1 рік після ЛРРШ. Недостатність вітаміну D констатували при значенні 25(OH)D менше 20 нг/мл та вторинний гіперпаратиреоз при рівні ПТГбільше 88 пг/мл.

Результати. Медіана рівня 25(OH)D в обох групах була в межах норми, проте у першій групі медіана була статистично достовірно нижчою порівняно із медіаною цього ж показника у другій групі (p=0,001). Кількість випадків гіповітамінозу вітаміну D була статистично достовірно вищою у першій групі, 17 проти 5 пацієнтів відповідно (p=0,003). Загальна поширеність ВГПТ була зафіксована у 14 (17,7%) пацієнтів, які були включені в дане дослідження, зі статистично значущою більшістю випадків у першій групі – 12 проти 2 (p=0,005) пацієнтів відповідно. Після операції рівень ПТГ мав сильний достовірний зворотній зв'язок із рівнями 25(OH)D та Ca2+ (p<0,001).

Висновок. Пацієнти після лапароскопічної рукавної резекції шлунка мають ризик розвитку вторинного гіперпаратиреозу та дефіциту вітаміну D у післяопераційному періоді. Підвищення рівня паратгормону після лапароскопічної рукавної резекції шлунка статистично достовірно пов'язане із дефіцитом вітаміну D та зниженням рівня кальцію. Додаткова вітамінно-мінеральна терапія у післяопераційному періоді після лапароскопічної рукавної резекції шлунка достовірно знижує ризик розвитку вторинного гіперпаратиреозу та дефіциту вітаміну D. Це дослідження також підкреслює важливість рутинного тестування на гіперпаратиреоз та вітамінну недостатність після лапароскопічної рукавної резукції шлунка.

Ключові слова: баріатрична хірургія, ожиріння, лапароскопічна рукавна резекція шлунка, іонізований кальцій, 25-гідроксикальциферол, паратгормон, недостатність вітаміну D, вторинний гіперпаратиреоз.

Introduction. Over the past decades, the problem of obesity has grown to pandemic levels around the world [1]. Numerous meta-analyses have confirmed the benefits of bariatric surgery over conservative methods of treating obesity. After surgery, it is possible to achieve a stable long-term weight loss and compensation for comorbidities associated with obesity [2].

In general, bariatric surgeries are classified as restrictive, malabsorptive, and combined, depending on the surgical procedure performed on the stomach and the bypass stage on the small intestine. Sleeve gastrectomy is a restrictive procedure. Since 2014, it has become the most performed bariatric procedure on all continents [3].

Unfortunately, in addition to a positive result in weight loss and compensation for associated comorbidities, in the long-term postoperative period, most patients after malabsorptive and combined bariatric surgery may suffer from deficiencies of vitamins and minerals, including calcium, vitamin D, vitamin B12, iron, folic acid, copper, selenium, and zinc. These deficiency conditions can lead to a cascade of metabolic disorders in the organism, that can lead to severe reversible damage to the organism [4].

Although sleeve gastrectomy does not have an intestinal bypass component, it preserves the passage of food and nutrients through the duodenum and proximal jejunum, but deficiency conditions can occur due to a decrease in food intake and accelerated evacuation from the stomach [5]. Moreover, it has already been confirmed that obesity itself may be associated with abnormal levels of certain microelements and hormones, in particular vitamin D, calcium and parathyroid hormone (PTH) [6]. It has also been reported that weight loss is the risk factor for bone loss and can significantly increase the probability of pathological bone fractures [7].

Several studies have analyzed the relationship between bariatric surgery and the development of secondary hyperparathyroidism (SHP), which is the result of calcium malabsorption. Thus, the incidence of secondary hyperparathyroidism in patients after Roux-en-Y gastric bypass (RYGB) occurs in 40% of cases, and in patients after biliopancreatic diversion this rate can be 100% [8].

SHPT due to increased calcium resorption from bones can lead to osteopenia, osteoporosis and osteomalacia over time. Reduced bone density, especially in overweight patients, leading to pathological bone fractures, resulting in increased rates of invalidization of this category of patients.

The literature describes incidents of vitamin D hypovitaminosis and SHPT after bariatric surgery, but most publications are focused on the analysis of results after malabsorptive surgery. At the same time, there are currently few publications regarding this pathological condition after LSG.

The aim of the study. To analyze the effect of sleeve gastrectomy on the development of vitamin D hypovitaminosis and secondary hyperparathyroidism 1 year after surgery.

Materials and methods. This retrospective study was based on the analysis of the results of treatment of 79 obese patients who underwent laparoscopic sleeve gastrectomy (LSG).

The inclusion criteria were: 1) patients' age from 19 to 70 years; 2) BMI from 35 to 70 kg/m^2; 3) preoperative levels of vitamin D, ionized calcium and parathyroid hormone within normal limits; 4) absence of organic pathology according to ultrasound of the parathyroid glands; 5) patients strictly followed the recommendations for diet and additional therapy of vitamins and trace elements in the postoperative period; 6) written consent to the processing of personal data was obtained.

Exclusion criteria: 1) patients who had a preoperative abnormality of one of the indicators (vitamin D, ionized calcium or parathyroid hormone); 2) concomitant pathology that could affect calcium-phosphorus metabolism, including chronic kidney disease and type 2 diabetes mellitus; 3) postmenopausal women; 4) complications that occurred in the early postoperative period.

The patients included in this study were divided into 2 groups depending on the receipt of additional vitamin and mineral therapy in the postoperative period. Thus, the first group of the study included 38 patients who did not receive additional therapy in the postoperative period after LSG. The second group included 41 patients who received vitamins and trace elements (cholecalciferol 2 000 IU, calcium 1,000 mg and a multivitamin 1 tablet per day) after LSG.

The data evaluated (except for age and sex) included: preoperative body weight, body mass index (BMI), and excessive body weight (EBW); postoperative body weight, BMI, percentage of excessive body weight loss (%EBWL), and percentage of total body weight loss (%TBWL). Body weight and excess body weight in kilograms (kg), BMI in kg/m^2, %BWL and %TBWL in percent.

Laboratory parameters analyzed in this study were ionized calcium (Ca2+) (mmol/L), 25 hydroxycalciferol (25(OH)D) (ng/mL) and parathyroid hormone (PTH) (pg/mL).

Laboratory studies were performed using an EasyLyte Calcium biochemical analyzer (Medica, USA) and a CL-1000i chemiluminescence analyzer (Mindray, China).

The diagnosis of secondary hyperparathyroidism (SHPT) was made in case of elevated PTH levels above 88 pg/ml. Hypovitaminosis of vitamin D was diagnosed in case of 25-hydroxycalciferol decrease below 20 ng/ml. The normal level of ionized calcium was in the range of 1.11–1.33 mmol/l.

Anthropometric and laboratory parameters were evaluated before surgery and 1 year after surgery.

The technique for performing LSG was the same in the study groups. The traditional scheme of trocar placement was used to perform the operation. After the formation of the pneumoperitoneum, the first step was to mobilize the stomach. Using the LigaSure electrosurgical instrument on a 12 mm (36 Fr) calibration probe, the great curvature and the bottom of the stomach were mobilized with electrical ligation of the gastric branches of the right and left gastroesophageal vessels, short vessels and the posterior gastric artery with the obligatory crossing of the gastroduodenal ligament and visualization of the left leg of the diaphragm. The latter is a criterion for the adequacy of mobilization in the gastric floor. The initial level of mobilization of the large curvature was at a distance of 4 cm from the pylorus. After that, the calibration probe was passed into the duodenum and its position along the minor curvature was ensured. A staged vertical gastric resection was performed using the Endo GIA linear suturing device (Medtronic). The gastric resection was performed on a 12 mm (36 Fr) calibration probe from a level of 4 cm from the pylorus (the initial point of mobilization) to the angle of Hiss, ensuring a gastric tube width of up to 2 cm and a controlled retreat of the staple suture line from the esophagogastric junction by 1 cm. The resection stage of the operation was performed with moderate lateral traction of the large curvature of the stomach by the assistant strictly behind the line of its mobilization. During the operation, each patient was tested for gastric tube leakage with methylene blue solution through a nasogastric tube and the operation was completed by drainage of the abdominal cavity.

Statistical processing methods. Statistical data processing was performed using the methods of variation and descriptive statistics using the SPSS Statistics statistical analysis package: An IBM Company, version 23. Before starting the data analysis, all indicators were checked for normality of distribution using the Shapiro-Wilcoxon test and for equality of variances using the Levene's criterion. Descriptive statistics were used in the study: mean (M) and standard deviation (SD) (for normal distribution) or median (Me) and interquartile range [25–75%] (for non-normal distribution). Statistically significant differences in relative values were assessed using the χ^2 Pearson's square test with the Yates correction. To evaluate statistically significant differences in the mean values of quantitative traits subject to the law of normal distribution, parametric methods of evaluation in independent groups were used (Student's t-test). For non-normal distribution, non-parametric methods of evaluation (Mann-Whitney U test) were used to determine the statistical significance of differences in the mean values of quantitative traits between two independent groups. To assess the degree of dependence between the variables, the Spearman's rank correlation coefficient (rs) was used in the distribution of indicators that differed from the normal distribution. Differences in the results were considered statistically significant at $p < 0.05$, which provides a 95% probability level.

Results. The average age of all patients was 40.7±11.4 years, ranging from 19 to 67 years. Patients in the first group were statistically significantly younger compared to patients in the second group, 36.9±11.2 years and 43.8±10.7 years, respectively (t=2.8; p=0.005). Anthropometric parameters, laboratory parameters and the nature of concomitant pathology of 79 patients included in this study are presented in Table 1.

The ratio of men to women in the first group was 17/21 and 26/15 in the second group, respectively. This frequency distribution by sex in the study groups did not differ significantly (χ^2=2.1; p=0.15).

Patients in the first group were slightly lighter than patients in the second group. Thus, the median body weight and BMI in the first group were 130 kg [124.5–152.8] and 44.7 kg/m^2 [41–50.1], and in the second group 152 kg [125.7–174.5] and 47.7 kg/m^2 [42.9–56.7], respectively ($p < 0.05$).

When analyzing laboratory parameters before surgery, it was found that the level of PTH in the second group was statistically significantly higher (U = 545; p = 0.02), although in both groups no deviations from the norm of this indicator were recorded in any case. There was no statistically significant difference in Ca2+ and 25(OH)D levels between the groups before surgery ($p > 0.05$).

Despite the fact that the patients of the first group were younger than the patients of the second group, after analyzing the frequency distribution of patients by the nature of concomitant pathology and the physical status of patients (according to the ASA scale), there was no statistically significant difference between the groups ($p > 0.05$).

The tactics of patient management in the early postoperative period were the same. Enteral nutrition was started from day zero of the postoperative period with a gradual increase. There were no cases of complications among the patients included in the study.

Patients in the second group took additional vitamins and minerals after surgery (cholecalciferol 2 000 IU, calcium 1 000 mg and a multivitamin 1 tablet per day).

Analyzing the anthropometric parameters of patients 1 year after surgery, it was found that the median body weight and BMI of patients in the second group were slightly higher compared to those of patients in the first group, 110 kg [84–116.5] and 31.6 kg/m^2 [29.4–37.6] and 94 kg [84.8–106.3] and 30.8 kg/m^2 [29.6–34.2], respectively ($p < 0.05$). However, given the rate of excess body weight loss, there was no statistically significant difference between the groups. Thus, the median %BWL in the first group was 60.5% [57.1–63.4] and in the second group –

Table 1

Characteristics of patients included in this study before surgery

Indicator	Total	Group 1 (without additional therapy)	Group 2 (with additional therapy)	p
Number of patients	79	38	41	
Age, years	40.7±11.4	36.9±11.2	43.8±10,7	t=2.8; p=0.005[1]
Gender (male/female)	43/36	17/21	26/15	c
Body weight, kg	140 [125-165.3]	130 [124.5-152.8]	152 [125.7-174.5]	U=642.5 p=0.04[3]
BMI, kg/m^2	45,9 [41.9-52.7]	44.7 [41-50.1]	47.7 [42.9-56.7]	U=639.5 p=0.04[3]
Overweight, kg	69.9 [59-93.3]	65.5 [58-85.5]	81.5 [60-102.5]	U=657 p=0.05[3]
Ca2+, mmol\l	1.26 [1.24-1.28]	1.26 [1.22-1.28]	1.26 [1.24-1.28]	U=765 p=0.9[3]
25(OH)D, ng/ml	42.3 [37.6-44.8]	40.6 [35.4-44.3]	43.2 [38.8-45.7]	U=617 p=0.1[3]
PTH, pg/ml	43.2 [33.5-52.6]	37.4 [31.3-48.1]	48.7 [37.8-53.2]	U=545 p=0.02[3]
Comorbidities, n (%)				
Type 2 diabetes mellitus, n (%)	23 (29.1%)	11 (28.9%)	12 (29.3%)	c
Arterial hypertension, n (%)	59 (74.7%)	28 (73.7%)	31 (75.6%)	c
Non-alcoholic fatty liver disease, n (%)	55 (69%)	26 (68.4%)	29 (70.7%)	c
Dyslipidemia, n (%)	66 (83.5%)	31 (81.6%)	35 (85.4%)	c
Obstructive sleep apnea syndrome, n (%)	49 (62%)	21 (55.3%)	28 (68.3%)	c

Data are presented as M ± SD, Me [IQ 25-75] or n (%)
Methods of statistical analysis between groups:
1 – by Student's t-test
2 – by the criterion of χ^2 Pearson's square with Yates' correction
3 – by the Mann-Whitney test

Table 2

Clinical characteristics of patients 1 year after surgery

Indicator	Total	Group 1 (without additional therapy)	Group 2 (with additional therapy)	p
Number of patients	79	38	41	
Body weight, kg	95 [84-111]	94 [84.8-106.3]	101 [84-116.5]	U=660 p=0.24[1]
BMI, kg/m^2	30.8 [29.4-35]	30.8 [29.6-34.2]	31.6 [29.4-37.6]	U=700 p=0.43[1]
%BWL, %.	60.8 [57.1-65.5]	60.5 [57.1-63.4]	61.6 [57.1-67.7]	U=654,5 p=0.22[1]
%TBWL, %.	31.5 [28.9-34.1]	31.1 [26.9-33.3]	31.6 [29.7-36.1]	U=566,5 p=0.04[1]
Ca2+, mmol\l	1.17 [1.14-1.21]	1.17 [1.14-1.21]	1.17 [1.15-1.22]	U=771 p=0.9[1]
Hypocalcemia, n (%)	6 (7.6%)	5 (13.2%)	1 (2.4%)	c
25(OH)D, ng/ml	37.8 [15.4-41.3]	32.3 [11.7-38.7]	40.2 [35.6-43.6]	U=347 p=0.001[1]
Vitamin D hypovitaminosis, n (%)	22 (27.4)	17 (44.7%)	5 (12.2)	c
PTH, pg/ml	63.4 [53.4-71.8]	68.8 [62.9-96.9]	53.7 [36.6-64,6]	U=245 p=0.001[1]
SHPT, n (%)	14 (17.7%)	12 (31.6%)	2 (4.9%)	c

Data are presented as M ± SD, Me [IQ 25-75] or n (%).
Methods of statistical analysis between groups:
1 – by the Mann-Whitney test
2 – by the criterion of χ

61.6% [57.1–67.6], respectively (U = 654.5; p = 0.22). Taking into account the classification proposed by R.B. Reinhold to assess the effectiveness of bariatric surgery, it can be stated that the rates of excess weight loss reached the level of a satisfactory result (Table 2).

At 1 year after surgery, hypocalcemia was recorded in 5 (13.2%) patients of the first group and 1 (2.4%) patient of the second group (χ^2=1.9; p=0.2). At the same time, the median and interquartile range of Ca2+ levels in both groups were within the normal range and no statistically significant difference was recorded (U=771; p=0.9).

The median 25(OH)D levels in both groups were within normal limits, but in the first group the median was statistically significantly lower compared to the median of the same indicator in the second group (U=347; p=0.001). In addition, the number of cases of vitamin D hypovitaminosis was statistically significantly higher in the first group, 17 vs 5 patients, respectively (χ^2=8.8; p=0.003).

SHPT was recorded in 14 (17.7%) patients included in this study, with a statistically significant majority of cases in the first group 12 vs. 2 (χ^2=7.9; p=0.005) patients, respectively. Also, the difference in the median PTH level between the groups reached a statistically significant level (U=245; p=0.001).

In patients who had an increase in PTH levels, 1 year after surgery, according to the control ultrasound of the parathyroid glands, no organic pathology was detected in any of the cases, which in turn allowed to confirm the diagnosis of SHPT.

One year after surgery, in patients with normal PTH values, vitamin D hypovitaminosis was recorded in 8 (12.3%) patients, whereas in the group of patients with SHPT, this disorder was detected in all patients (χ^2=39.8; p=0.001) (Table 3).

At the same time, the median calcium level in patients with HPT did not differ statistically from that in patients with normal SHPT values (U=389; p=0.4). Similarly, there was no statistically significant difference in the number of episodes of hypocalcemia (χ ^{2}=2.6; p=0.1).

After correlation analysis of the parameters in the group of patients with SHPT, it was found that PTH levels had a strong significant inverse relationship with 25(OH)D and Ca2+ levels (rs=0.73; p=0.003 and rs=0.62; p=0.017) (Fig. 1).

Discussion. Whether nutritional deficiencies and endocrine side effects persist over the long term following the LSG has yet to be established. As the age of patients seeking bariatric surgery increases, the skeletal consequences of secondary hyperparathyroidism, such as osteoporosis and osteomalacia, become increasingly important [9]. Our study is demonstrate that SHPT occur at 1 years following the LSG in 14 (17.7%) patients, despite patients being supplemented with calcium and vitamin D.

Preoperative and postoperative vitamin D deficiencies appear in 68 and 80% of patients respectively as secondary hyperparathyroidism is frequent after bariatric surgery [4, 8]. It is reported to be common in malabsorptive procedures, appearing in biliopancreatic diversion patients after 1 year, and to a lesser extent in RYGB patients. Although LSG is considered a pure restrictive procedure, vitamin D deficiency can still occur. Up to 53 % of patients are found to have increased serum PTH post-obesity surgery [5, 10]. Despite receiving vitamin D supplementation after gastric bypass surgery, vitamin D deficiency with hyperparathyroidism is seen to continue to 50 % of patients [11]. The reasons remain unknown but may be attributed to various factors in dietary intake, season of the year, and socioeconomic status. Deficiency of vitamin D will in turn lead to

Table 3

Clinical characteristics of patient groups, depending on the presence of SHPT

Indicator	Group with SHPT	Group without SHPT	p
Number of patients	14	65	
Age, years	42 [27.5-54]	40 [32.5-48]	U=428 p=0.7[1]
Gender (male/female)	7/7	36/29	c
Body weight before surgery, kg	132.5 [124.5-151.3]	143 [124.5-166]	U=398 p=0.4[1]
Body weight after surgery, kg	95.5 [84.7-108.3]	95 [84-111.5]	U=427 p=0.7[1]
BMI before surgery, kg/m^2	46.4 [42.5-50.2]	45.5 [41.6-54.5]	U=435 p=0.8[1]
BMI after surgery, kg/m^2	32.4 [29.3-34.4]	30.6 [29.4-35.4]	U=429 p=0.7[1]
%BWL, %.	59.7 [54.1-62.1]	61.2 [49.9-65.8]	U=338 p=0.1[1]
%TBWL, %.	29.9 [26.5-33.9]	31.6 [29-34.5]	U=354 p=0.2[1]
PTH, pg/ml	100.5 [97.4-107.3]	61.2 [49.9-65.8]	U=1 p=0.001[1]
Ca2+, mmol\l	1.16 [1.12-1.21]	1.18 [1.15-1.21]	U=389 p=0.4[1]
Hypocalcemia, n (%)	3 (21,4%)	3 (4,6%)	c
25(OH)D, ng/mL	9.2 [11.3-12.2]	39.7 [33.4-41.6]	U=45 p=0.001[1]
Vitamin D hypovitaminosis, n (%)	14 (100%)	8 (12,3%)	c

Data are presented as M ± SD, Me [IQ 25-75] or n (%).
Methods of statistical analysis between groups:
1 – by the Mann-Whitney test
2 – by the criterion of χ

decreased calcium absorption. This may result in low calcitriol, which favors fat accumulation [4].

Signs and symptoms of vitamin D deficiency include osteomalacia, osteoporosis, arthralgia, myalgia, fasciculation, and depression. Considering the high prevalence of preoperative deficiency, all candidates for obesity surgery should undergo 25-hydroxy vitamin D screening. Awareness should be noted that serum calcium may be low or normal with a decrease in serum phosphorus and increase in serum alkaline phosphatase [12].

The clinical significance of prolonged hyperparathyroidism in bariatric surgery population is unknown. Johnson et al. found that bone mineral density decreased after the first year post-RYGB but then stabilized. A 2016 meta-analysis of 11 studies, looking at changes in bone metabolism after bariatric surgery, echoed Johnson et al. in that bone mineral density was significantly reduced after gastric bypass with elevations in both urinary N-telopeptide; however, most studies had short-term follow-up, between 6 and 12 months [10]. In a small study of 30 individuals undergoing gastric bypass and 20 controls, Yu and colleagues recently demonstrated that ongoing bone mineral density losses persist up to 24 months of follow-up [10]. It is still unclear if this reduction in bone mineral density translates

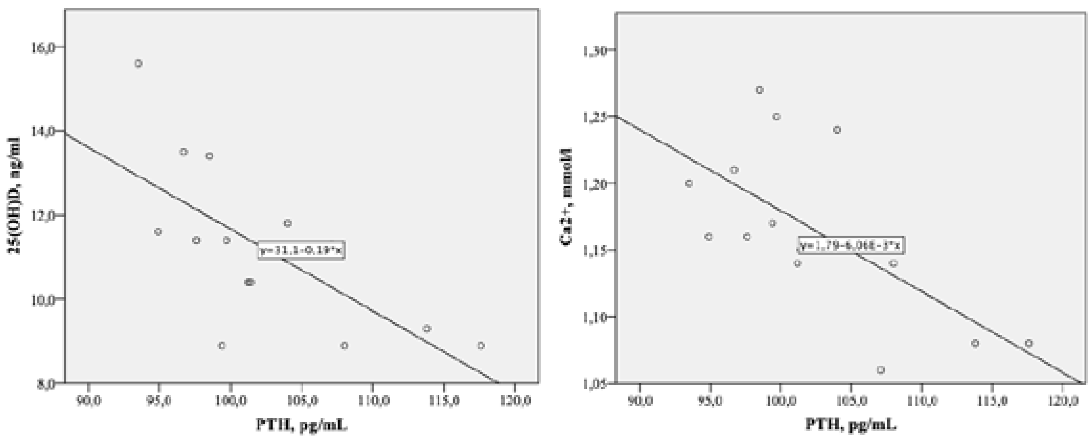

Fig. 1. Graphical representation of the correlation between PTH and 25(OH)D (A) and between PTH and Ca2+ (B) in the group of patients with SHPT

into an increased risk of fragility fractures [12]. Parsing out the effect of prolonged secondary hyperparathyroidism on bone metabolism and fracture risk is made difficult by the fact that >10% weight loss itself, regardless of etiology, is associated with a 1 to 2% bone loss, which can confound results [13]. The relative risk increase for any fracture following bariatric surgery has been reported to be between 1.38 and 2.30 times compared to controls [6]. Also, the sites of bone mineral density loss are differentially distributed, with fracture sites shifting from classic obesity patterns to ones more correlated with osteoporosis after surgery [13].

Despite a successful supplementation regimen, some patients remain at risk for bone resorption [14]. One potential explanation is that vitamin D levels in obese individuals need to be higher to minimize the potential for developing hyperparathyroidism. This is supported by the current study in which we demonstrated that patients with elevated PTH very rarely had vitamin D levels above 35 ng/mL. Inadequate vitamin D absorption may also play a role. Some studies suggest that calcium malabsorption is a limiting factor. Johnson et al. found a significant increase in mean PTH values among patients undergoing a long-limb bypass (Roux >100 cm) versus short-limb bypass (<100 cm) [12]. Reidt et al. reported a significant decrease in true fractional calcium absorption following RYGB, however, it remained within normal range [15].

The number of cases of vitamin D hypovitaminosis was statistically significantly higher in the group who did not receive additional therapy in the postoperative period, 17 vs 5 patients, respectively. In our study, we found a correlation between PTH levels and vitamin D and iCa levels, which are risk factors for secondary hyperparathyroidism.

There is no agreement on recommendations for vitamin D therapy after bariatric surgery as few evidence-based regimens exist.

With severe deficiency, 50 000–150 000 IU of vitamin D3 per day can be sufficient with oral calcitriol if necessary. Some studies have recommended 5000 IU per day and 50 000 IU 2 times per day for prophylaxis and maintenance in RYGB and biliopancreatic diversion patients correspondingly [16, 17]. Proper dosage may widely vary in each individual patient, and 25-hydroxy vitamin D levels should be monitored 2 weeks after initiation. These levels should be repeated up to every 3 months in the first year after surgery. Suggested supplementation for prevention after surgery is generally 2 000 IU per day of oral vitamin D2 or D3. For patients who underwent malabsorptive procedures, vitamin D levels of ≥100 nmol/L have been demonstrated to be effective in prevention of secondary hyperparathyroidism [8].

Regular supplementation with vitamins and minerals after bariatric surgery is one of the most important components of the prevention of metabolic disorders in the postoperative period.

Conclusion. Patients after laparoscopic sleeve gastrectomy have a risk of secondary hyperparathyroidism and vitamin D deficiency in the postoperative period. The higher levels of parathyroid hormone post laparoscopic sleeve gastrectomy significantly related to vitamin D deficiency and lower calcium levels. Additional vitamin and mineral therapy in the postoperative period after laparoscopic sleeve gastrectomy significantly reduces the risk of developing secondary hyperparathyroidism and vitamin D deficiency. This study also emphasizes the importance of routine testing for hyperparathyroidism and vitamin deficiency after laparoscopic sleeve gastrectomy.

Further research and investigation are needed to validate these findings and explore effective strategies for preventing the secondary hyperparathyroidism and vitamin deficiency in patients after sleeve gastrectomy.

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Kalashnikov O.O. – idea, purpose, study design, data interpretation, statistical analysis, manuscript preparation, literature search, funds collection;

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Todurov I.M. – idea, purpose, data interpretation, manuscript preparation, funds collection;

Orlyk O.S. – data collection, data interpretation;

Hrynevych A.A. – data collection, literature search.

REFERENCES

1. Swinburn B.A., Sacks G., Hall K.D., McPherson K., Finegood D.T., Moodie M.L., Gortmaker S.L. The global obesity pandemic: shaped by global drivers and local environments. Lancet. 2011;378(9793):804-814. https://doi.org/10.1016/s0140-6736(11)60813-1.

2. Gloy V.L., Briel M., Bhatt D.L., Kashyap S.R., Schauer P.R., Mingrone G., Bucher H.C., Nordmann A.J. Bariatric surgery versus non-surgical treatment for obesity: a systematic review and meta-analysis of randomized controlled trials. BMJ. 2013;347:f5934. https://doi.org/10.1136/bmj.f5934.

3. Angrisani L., Santonicola A., Iovino P., Vitiello A., Higa K., Himpens J., Buchwald H., Scopinaro N. IFSO Worldwide Survey 2016: Primary, Endoluminal, and Revision Procedures. Obes Surg. 2018;28(12):3783-3794. https://doi.org/10.1007/s11695-018-3450-2.

4. Gasmi A, Bjørklund G, Mujawdiya PK, et al. Micronutrient deficiencies in patients after bariatric surgery. Eur J Nutr. 2022;61(1):55-67. https://doi:10.1007/s00394-021-02619-8.

5. Gjessing H, Nielsen H, Mellgren G, et al. Energy intake, nutritional status and weight reduction in patients one year after laparoscopic sleeve gastrectomy. Springerplus. 2013;2(1):352. https://doi:10.1186/2193-1801-2-352.

6. Mussolino ME, Looker AC, Madans JH, Langlois JA, Orwoll ES. Risk factors for hip fracture in white men: the NHANES I Epidemiologic Follow-up Study. J Bone Miner Res. 1998;13(6):918-924. https://doi:10.1359/jbmr.1998.13.6.918.
7. Ensrud KE, Fullman RL, Barrett-Connor E, et al. Voluntary weight reduction in older men increases hip bone loss: the osteoporotic fractures in men study. J Clin Endocrinol Metab. 2005;90(4):1998-2004. https://doi:10.1210/jc.2004-1805.
8. Hewitt S, Søvik TT, Aasheim ET, et al. Secondary hyperparathyroidism, vitamin D deficiency, and serum calcium 5 years after gastric bypass and duodenal switch. Obes Surg. 2013;23(3):384-390. https://doi:10.1007/s11695-012-0772-3.
9. Chow A, Switzer NJ, Gill RS et al. Roux-en-Y gastric bypass in the elderly: a systematic review. Obes Surg 2016; 26: 626–630. https://doi:10.1007/s11695-015-2007-x
10. Yu EW, Bouxsein ML, Putman MS et al. Two-year changes in bone density after Roux-en-Y gastric bypass surgery. J Clin Endocrinol Metab 2015; 100: 1452–1459. https://doi:10.1210/jc.2014-4341
11. Carlin AM, Rao DS, Yager KM, Genaw JA, Parikh NJ, Szymanski W. Effect of gastric bypass surgery on vitamin D nutritional status. Surg Obes Relat Dis. 2006;2(6):638–42. https://doi:10.1016/j.soard.2006.09.003
12. Johnson JM, Maher JW, Samuel I, Heitshusen D, Doherty C, Downs RW. Effects of gastric bypass procedures on bone mineral density, calcium, parathyroid hormone, and vitamin D. J Gastrointest Surg 2005; 9: 1106–1110; discussion 1110-1. https://doi:10.1016/j.gassur.2005.07.012
13. Shapses SA, Sukumar D. Bone metabolism in obesity and weight loss. Annu Rev Nutr 2012; 32: 287–309. https://doi:10.1146/annurev.nutr.012809.104655
14. Liu C, Wu D, Zhang JF et al. Changes in bone metabolism in morbidly obese patients after bariatric surgery: a meta-analysis. Obes Surg 2016; 26: 91–97. https://doi:10.1007/s11695-015-1724-5
15. Riedt CS, Brolin RE, Sherrell RM, et al. True fractional calcium absorption is decreased after Roux-en-Y gastric bypass surgery. Obesity 2006;14:1940. https://doi:10.1038/oby.2006.226
16. Goldner WS, Stoner JA, Lyden E, Thompson J, Taylor K, Larson L, et al. Finding the optimal dose of vitamin D following Roux-en-Y gastric bypass: a prospective, randomized pilot clinical trial. Obes Surg. 2009;19(2):173–9. https://doi:10.1007/s11695-008-9680-y
17. Heber D, Greenway FL, Kaplan LM, Livingston E, Salvador J, Still C, et al. Endocrine and nutritional management of the post-bariatric surgery patient: an Endocrine Society Clinical Practice Guideline. J Clin Endocrinol Metab. 2010;95(11):4823–43. https://doi:10.1210/jc.2009-2128