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A. O. Nykonenko[1], Ye. I. Haidarzhi[1], M. Bužga[2]

Bone metabolism changes after laparoscopic greater curvature plication. A one-year study

[1]Zaporizhzhia State Medical University, Ukraine,

[2]University of Ostrava, Czech Republic

***Key words**: Bariatric Surgery, Laparoscopic Plication, Bone Metabolism.*

In addition to the positive effect of weight loosing after bariatric surgery, the post-operative period is often associated with significant changes in bone metabolism.

Aim. The aim of this study was to examine changes in bone turnover markers and indicators of dual-energy X-ray absorptiometry (DXA) within 12 months after laparoscopic greater curvature plication (LGCP).

Materials and methods. We studied 54 patients from class III obesity who underwent LGCP. The average preoperative weight of the patients was 125.5±19.1 kg, and BMI of 43.0±4.9 kg/m^2. The gender distribution of the cohort was 35.2% man and 64.8% women. At three time points after the operation (at 3, 6 and 12 months) control examination was conducted that included DXA and markers of bone metabolism.

Results. After the surgical treatment we registered significant reduction of all the anthropometric indicators ($\rho<0.05$; EWL_{12} 45.8±18.8%; EBL_{12} 55.1±23.4%). According to the controlled studies on the 12th months, a significant increase in bone turnover markers was revealed ($\rho<0.05$; CTX, P1NP, OC, OPG, VTD) and reducing of PTH ($\rho<0.05$). The measure of DXA on the 12th month of observation showed no significant changes in bone metabolism. Strong positive correlation was uncovered between markers of bone metabolism: CTX / P1NP (r=0.790, p=0.000), CTX/OC, P1NP/OC (r=0.7, p=0.000), minor correlation between PTH/OC (r=0.249, p=0.027). We also report a minor and weak negative correlation between the markers of bone metabolism (CTX, P1NP, OC and VTD) and anthropometric data.

Conclusions. Twelve months post LGCP markers of bone turnover (CTX, P1NP, OC, OPG, VTD) were significantly increased. The increased level of VTD is associated with the loss of FAT. Also the change in DXA was detected.

Зміни кісткового метаболізму після лапароскопічної плікації великої кривизни шлунка. Річне дослідження

А. О. Никоненко, Є. І. Гайдаржі, М. Бужга

Незважаючи на позитивний ефект втрати ваги після баріатричних операцій, післяопераційний період часто пов’язаний зі значними змінами в метаболізмі кісткової тканини.

Мета роботи – вивчення динаміки маркерів ремоделювання кістки та показників її двоенергетичної рентгенівської абсорбціометрії протягом 12 місяців після лапароскопічної плікації великої кривизни шлунка.

Матеріали та методи. Досліджували 54 пацієнти з ожирінням III ступеня, які перенесли лапароскопічну плікацію великої кривизни шлунка. Середня передопераційна вага пацієнтів була 125,5±19,1 кг і ІМТ 43,0±4,9 кг/м^2. Поділ за статтю був такий: 35,2% чоловіків і 64,8% жінок. Контрольне обстеження здійснили на 3, 6 і 12 місяці після операції. Обстеження включало вимір показників двоенергетичної рентгенівської абсорбціометрії та визначення маркерів кісткового метаболізму.

Результати. Після хірургічного лікування зареєстрували значне зниження всіх антропометричних показників ($\rho<0{,}05$; EWL_{12} 45,8±18,8%; EBL_{12} 55,1±23,4%). Згідно з результатами досліджень через 12 місяців виявили значне збільшення маркерів ремоделювання кістки ($\rho<0{,}05$; CTX, P1NP, OC, OPG, VTD) та зниження PTH ($\rho<0{,}05$). Виміри показників двоенергетичної рентгенівської абсорбціометрії на 12 місяць спостереження не показали будь-яких істотних змін у метаболізмі кісткової тканини. Сильна позитивна кореляція виявлена між маркерами кісткового метаболізму: CTX/P1NP (r=0,790, p=0,000), CTX/OC, P1NP/OC (r=0,7; p=0,000), незначні кореляції – між PTH/OC (r=0,249, p=0,027). Також помічено незначну і слабку негативну кореляцію між маркерами кісткового метаболізму (CTX, P1NP, OC і VTD) та антропометричними даними.

Висновки. Через 12 місяців після лапароскопічної плікації великої кривизни шлунка значно збільшуються маркери ремоделювання кістки (CTX, P1NP, OC, OPG, VTD). Підвищений рівень вітаміну D пов’язаний із втратою жирової тканини. Визначаються зміни показників двоенергетичної рентгенівської абсорбціометрії.

***Ключові слова:** баріатрична хірургія, лапароскопічна плікація, кістковий метаболізм.*

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Динамика костного метаболизма после лапароскопической пликации большой кривизны желудка. Годовое исследование

А. А. Никоненко, Е. И. Гайдаржи, М. Бужга

Несмотря на положительный эффект потери веса после бариатрической операции, послеоперационный период часто связан со значительными изменениями в метаболизме костной ткани.

Цель работы – изучение динамики маркеров ремоделирования кости и показателей двухэнергетической рентгеновской абсорбциометрии в течение 12 месяцев после лапароскопической пликации большой кривизны желудка.

Материалы и методы. Исследовали 54 пациента с ожирением III степени, перенесших лапароскопическую пликацию большой кривизны желудка. Средняя предоперационная масса пациентов – 125,5±19,1 кг и ИМТ 43,0±4,9 кг/м^2. Распределение по полу было следующим: 35,2% мужчин и 64,8% женщин. Контрольное обследование проводилось на 3, 6 и 12 месяцы после операции. Обследование включало измерение показателей двухэнергетической рентгеновской абсорбциометрии и определение маркеров костного метаболизма.

Результаты. После хирургического лечения зарегистрировано значительное снижение всех антропометрических показателей (ρ<0,05; EWL_{12} 45,8±18,8%; EBL_{12} 55,1±23,4%). Согласно результатам исследований через 12 месяцев было обнаружено значительное увеличение маркеров ремоделирования кости (ρ<0,05; CTX, P1NP, OC, OPG, VTD) и снижение PTH (ρ<0,05). Измерения показателей двухэнергетической рентгеновской абсорбциометрии на 12 месяц наблюдения не показали каких-либо существенных изменений в метаболизме костной ткани. Сильная положительная корреляция обнаружена между маркерами костного метаболизма: CTX/P1NP (r=0,790, p=0,000), CTX/OC, P1NP/OC (r=0,7, p=0,000), незначительные корреляции между PTH/OC (r=0,249, p=0,027). Также замечена незначительная и слабая отрицательная корреляция между маркерами костного метаболизма (CTX, P1NP, OC и VTD) и антропометрическими данными.

Выводы. Через 12 месяцев после лапароскопической пликации большой кривизны желудка значительно увеличиваются маркеры ремоделирования кости (CTX, P1NP, OC, OPG, VTD). Повышенный уровень витамина D связан с потерей жировой ткани. Отмечаются изменения показателей двухэнергетической рентгеновской абсорбциометрии.

Ключевые слова: *бариатрическая хирургия, лапароскопическая пликация, костный метаболизм.*

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Despite the progress in the prevention and treatment obesity, in 2014 more than 1.9 billion adults (18 years and older) were overweight, 600 million of whom were obese [1]. According to the literature review, the incidence of bariatric surgery has plateaued at approximately 200,000 cases per year since 2002 [2].

Nowadays, the most common bariatric operations are laparoscopic adjustable gastric banding, laparoscopic Roux-en-Y gastric bypass, and laparoscopic sleeve gastrectomy. Such operations are quite effective at significantly reducing the body mass and are associated with low rates of surgical complications [3,4]. Laparoscopic greater curvature plication (LGCP) is a new method of surgical treatment for obesity and was proposed in 2007 by Talebpour et al. [5]. It belongs to the «safe organ preserving surgery», has a high efficiency rate and is commonly chosen for treating extreme cases of obesity. However, issues with changes in bone metabolism have recently been brought to light in association to bariatric surgery [6–9].

Multiple groups reported that after bariatric surgeries most patients achieved a 50% weight loss with a proportionately greater loss of bone mass [10,11]. The mechanisms of bone loss after bariatric surgery are currently unknown, but are likely multifactorial. Secondary hyperparathyroidism due to vitamin D deficiency and mechanical unloading due to weight loss are among the most commonly cited potential mechanisms underlying bone loss after bariatric surgery. Yet, evidence from the literature does not support these hypotheses. New hypotheses involving crosstalk between the skeleton and gastrointestinal, adipocytic and neurohormonal systems are now being explored [6]. In contrast, other authors attribute changes in bone metabolism to the effects of anatomical changes in the stomach, suggesting that anatomic changes in the gastrointestinal tract lead to malabsorption of nutrients, including calcium and vitamin D [12–14].

Cong Liu et al. (2015) conducted a meta-analysis of bone changes following bariatric surgery, and identified significant decrease in serum calcium, increase in serum parathyroid hormone (PTH), but no difference in serum vitamin D 25(OH)D (VTD). In addition, they reported significant increase in serum or urinary N-telopeptide and in bone - specific alkaline phosphatase, accompanied by a decrease in bone mineral density (BMD) [8].

This time the most informative method of determining BMD is Dual-energy X-ray absorptiometry (DXA). The advantages of DXA are its high sensitivity to osteoporosis. In most studies dedicated to bone metabolism, investigations of BMD were made by DXA, and also by special markers of bone metabolism [7,8,15].

The aim of this study was to analyze possible postoperative changes in blood markers of bone metabolism (including vitamin D (25-OH-D), PTH) and DXA result during a 1-year period (3–6–12 month) after LGCP.

Material and Methods

We studied 54 patients from class III obesity [16] who underwent LGCP. We classified participants according to IFSO 2005 guidelines [17]. Thus, patients with a body mass index (BMI) > 40 kg/m^2 or BMI > 35 kg/m^2 with associated co-morbidities were included into the study. The study included patients, aged 20 to 58 years, referred for laparoscopic gastric plication to the old surgical ward Care Center digestive tract Vitkovice Hospital in Ostrava and the Center for Endoscopy Clinic of Internal Medicine University Hospital Ostrava. LGCP was performed according to the usual protocol – general anesthesia and a laparoscopic approach with five ports. The ports were placed in the abdomen according to a standard schema. The plication was standard with endoscopic observation. The percent of excess weight loss (EWL) was calculated using the formula described by Deitel and Greenstein [18].

At time points of 3, 6 and 12 months after the surgery, supervisory examinations were performed on all study participants. Control examination included: specialized surgical examination at the surgical clinic, DXA, bioimmunoassay (BIA) and blood sampling on basic and specialized examinations (C-terminal telopeptide of collagen I (CTX), parathyroid hormone (PTH), the serum receptor-activator of nuclear factor-kB ligand (sRANKL), N-terminal procollagen type 1 (P1NP), Osteocalcin (OC), Osteoprotegerin (OPG) and vitamin D (25-OH-D) (VTD) levels). Therefore, to assess bone metabolism, the entire cluster parameters of bone remodeling were evaluated. These parameters included both parameters of osteoresorption (bone resorption), CTX, PTH, and sRANKL and parameters of bone osteoformation (bone formation), P1NP, OC, and OPG. Also, the level of hormones Ghrelin, Leptin, and Adiponectin were studied before and after operation at 3, 6 and 12 months.

Method DXA was performed on Discovery apparatus (Hologic, Inc., USA) and a whole-body scan was done on body composition density and bone tissue in the femoral neck and vertebral segments L1-L5 within the sagittal projection in the laboratory of the Institute of Clinical Physiology, Medical Physiology OU. BMD in g/cm^2 and then the value of T and the Z score were assessed. The measured bone mineral density was automatically compared to the average value in young healthy individuals of the same sex (ie. T-score) and the average for the same age category (ie. Z-score). This comparison was expressed in standard deviations from the mean. Osteoporosis was defined as the condition where bone loss exceeds – 2.5 standard deviations (as determined by the World Health Organization). Decrease of -1 to - 2.5 standard deviations is considered osteopenia, which is not yet a disease, but signifies a greater risk of osteoporosis in the future. Also, using the DXA we have received the results of fat mass – Fat DXA in % and in kg and lean body mass (LBM) – it is a total lean mass – in kg.

Statistical Analysis

All analyses were performed using the Statistica program STATISTICA 7.0 for Windows (StatSoft.Inc., США). The distribution of analyzed data was checked by the Shapiro–Wilk test. Values were expressed as means ± standard deviation (SD) and for not normally distributed represented by Me (median) and lower/upper quartile (25–75%). Upon the comparison of the groups, before and after the treatment (3–6–12 months), we used parametric statistics, t-test for dependent variables, and non-parametric statistics, Wilcoxon singed-ranked test. Pearson test was performed to evaluate associations between variables. The estimated parameters were considered as significant at r<0.05.

Results

All 54 patients completed the 3–6–12 month follow-up examinations. There were 19 men (35.2%) and 35 women (64.8%). The average age of the patients was 41.3±7.9 years (men 46.5±8.7, women 38.5±5.9). The average preoperative weight of the patients was 125.5±19.1 kg, and BMI of 43.0±4.9 kg/m^2. This is consistent with the BMI of the III class obesity patient [17]. During the study period, patients didn't take any specific therapy to improve their bone metabolism.

Based on DXA analysis, initial average weight of the patients was 125.5±19.1 kg. Under dynamic observation the weight went down to – 16.7 kg over the period of 3 months, to 22.1 kg – over the 6th months and to 25.0 kg over the 12 months respectively. *Table 1* presents the dynamic of weight loss. This result implies that on the 3rd month, the average weight of the participants was within class II of obesity, and on the 6th and the 12th months – within class I. The final EWL was 45.8%, which signifies the effectiveness of surgical treatment based on the results of the 12-month period. As the «Fat DXA» and LBM assays indicated, there was a significant loss of the fat mass, nearly 19.4 kg and the muscle loss of 5.2 kg during the first three months, with a significant decrease during the following 12th months.

Parameters of bone osteoresorption and bone osteoformation are presented in *Table 2*. The initial figures of CTX were within reference values (N = 0.142–0.55 μg/l) and on the 3rd, 6th, 12th months there was a significant increase of CTX with the final figure of 0.38 μg/l after the 12th month of medical supervision. PTH level on the 3rd month after surgery decreased significantly to 3.8 pmmol/l. Changes of sRANKL (N=300–380 mmol/l) during the observation period were not authentic and after surgery ranged within 96.9 – 122.0 pmmol/l. Before the surgery sRANKL results were below normal values. It is noteworthy

Table 1

Anthropometric parameter changes at 3–6–12 months after surgery (n=54)

	Pre-operative examination	3 month after surgery	6 month after surgery	12 month after surgery
Weight [kg]	125,5±19,1	108,2±17,4*	103,4±18,0*	101,4±19,6*
BMI [kg/m^2]	43,0±4,9	37,1±4,9*	35,5±5,3*	34,7±6,11*
FAT DXA [kg]	56,1±9,9	45,4±9,9*	41,1±10,2*	38,7±11,9*
FAT DXA [%]	45,2±5,8	41,9±6,1*	39,7±6,6*	38,3±7,5*
LBM [kg]	62,8 (76,8-55)	58,3 (71,7-49,2)$^+$	56,9 (71,3-49,0)$^+$	58,5 (68,5-48,9)$^+$

Note: *– Comparison with pre-operative examination ρ<0.05 (*t-test*); $^+$ – Comparison with pre-operative examination ρ<0.05 (*Wilcoxon signed-rank test*).

Table 2

Parameters of bone metabolism before and after surgery (n=54)

	Pre-operative examination	3 month after surgery	6 month after surgery	12 month after surgery
CTX (μg/l)	0,25 (0,19-0,34)	0,41 (0,56-0,30)* p=0,0000	0,37 (0,63-0,27)* p=0,000004	0,38 (0,52-0,25)* p=0,000016
PTH (pmmol/l)	4,43 (5,9-2,7)	3,8 (5,1-2,5)* p=0,02	3,32 (4,43-2,53)* p=0,000034	3,8 (4,3-2,4)* p=0,0139
sRANKL (pmmol/l)	88,2 (223,8-49,4)	96,9 (256,4-63,1) p=0,12	119,7 (202,3-65,1) p=0,45	122,0 (184,8-53,1) p=0,84
P1NP (μg/l)	33,9 (39,8-26,4)	45,9 (59,6-32,0)* p=0,003	50,9±18,7* p=0,000017	47,9 (69,1-38,6)* p=0,00012
OC (μg/l)	13,6 (16,7-10,6)	17,4 (22,3-15,1)* p=0,00002	17,5 (23,8-14,6)* p=0,000055	19,2 (22,6-13,8)* p=0,00014
OPG (pmmol/l)	4,7 (6,4-4,1)	4,3 (5,0-3,6)* p=0,01	4,5 (5,7-3,8) p=0,716	5,2 (6,5-4,4)* p=0,015
Vit. D. (nmol/l)	14,6 (25,9-8,3)	19,6 (41,7-13,6)* p=0,000004	28,0 (51,1-12,9)* p=0,000001	30,5 (64,3-21,4)* p=0,000006

*Note:** – Comparison with pre-operative examination ρ<0.05 *(Wilcoxon signed-rank test).*

to mention a significant increase in P1NP on the 3rd, 6th and 12th post-surgery months, however the values stayed within the reference range (N=5–58.6 µg/l). Level of osteocalcin (OC) on the 12th month of supervision was 19.2 µg/l, which was a significant increase with respect to the preoperative period, and was within normal parameters (11–42.5 µg/l). There were no notable changes in the level of OPG, but from the 3rd month there was a significant decrease, and at the 12th month an increase, the final result was 5.2 pmmol/l at the reference value of 1.8–6.4 pmmol/l. VTD level has grown significantly by the end of the supervision period and reached 30.5 nmol/l.

All patients included in the study underwent a densitometry testing and DXA at the femoral neck and lumbar spine of the bone *(Table 3*). Based on the statistical analysis (Mann-Whitney U test and t-test) and the comparisons of DXA studies results, there were no clinically significant changes in DXA before and after surgical treatment of patients.

Thus, our study revealed significant changes in the following markers of bone metabolism: CTX, PTH, P1NP, OC, OPG and VTD, and also anthropometric indicators. To establish the dependence of changes we conducted a correlation between the markers of bone metabolism and anthropometry (*Table 4)*. Positive correlation was observed between the OC and CTX/PTH/P1NP, between CTX and P1NP as well as between the OS and BMI/FAT. In other cases, there was a negative correlation: VTD and PTH, BMI/FAT; between P1NP and FAT; between CTX and BMI/FAT. There was no correlation between other parameters.

Discussion

In the current study all LGCP patients attained effective weight loss – EWL on the 12th month after surgery was 45.8±18.8%, EBL – 55.1±23.4%. These figures confirm the effectiveness of the operation. There were no complications or mortalities after the surgery.

However, we found significant changes in bone metabolism according to the performed assays – serum bone turnover markers. Currently, serum-CTX and P1NP assays are recommended osteoporosis diagnostics, and to the level of bone turnover determination [19,20]. CTX and P1NP assays are specific markers of bone metabolism and their increasing levels can denote osteoporosis or osteopenia. In our study, we found a significant increase of CTX and P1NP levels from the 3rd postoperative month, but it was within the reference values.

Table 3

Parameters of DXA: before and after surgery (n=54)

		Pre-operative examination	3 month after surgery	6 month after surgery	12 month after surgery
Spine	BMC (g)	68,9±13,5	72,7±14,6* p=0,001	73,0±13,6* p=0,0016	73,2±14,9 p=0,32
	BMD [g/cm]	1,13±0,13	1,15±0,13 p=0,313	1,14±0,13 p=0,604	1,14±0,13 p=0,776
	T-score	0,72±1,22	0,81±1,23 p=0,36	0,78±1,22 p=0,67	0,73±1,16 p=0,73
	Z-score	0,97±1,3	1,07±1,3 p=0,34	1,04±1,31 p=0,55	1,05±1,24 p=0,91
Femur neck	BMC (g)	5,1±1,02	5,1±1,02 p=0,79	5,03±1,03 p=0,27	5,0±1,19 p=0,41
	BMD [g/cm]	0,96±0,14	0,95±0,12 p=0,19	0,94±0,13 p=0,07	0,93±0,13 p=0,06
	T-score	0,78±1,19	0,67±1,06 p=0,12	0,53±1,06 p=0,05	0,49±1,02 p=0,08
	Z-score	1,16±1,19	1,08±1,07 p=0,17	0,96±1,07 p=0,09	0,94±1,0 p=0,11
Femur total	BMC (g)	42,2 (50,6-36,8)	41,8 (51,0-36,8) p=0,96	42,0 (52,1-36,7) p=0,84	42,9 (53,0-37,9) p=0,96
	BMD [g/cm]	1,14±0,13	1,12±0,14* p=0,015	1,11±0,13* p=0,004	1,12±0,13 p=0,219
	T-score	1,34±1,03	1,18±1,08* p=0,009	1,09±1,03* p=0,002	1,12±0,89 p=0,228
	Z-score	1,55±1,03	1,39±1,09* p=0,01	1,3±1,03* p=0,003	1,36±0,87 p=0,269

Note: *– Comparison with pre-operative examination ρ<0.05 (t-test): + – Comparison with pre-operative examination ρ<0.05 (*Wilcoxon signed-rank test*).

Table 4

Correlation between markers of bone metabolism and anthropometry

	CTX, (r/p)	PTH (r/p)	P1NP, (r/p)	OC, (r/p)	OPG (r/p)	BMI, (r/p)	FAT,(r/p)	LBM, (r/p)
CTX	na	NC	0,790/0,000	0,738/0,000	NC	-0,338/0,003	-0,459/0,000	NC
PTH	-	na	NC	0,249/0,027	NC	NC	NC	NC
P1NP	-	-	na	0,772/0,000	NC	NC	-0,330/0,004	NC
OC	-	-	-	na	NC	-0,398/0,000	-0,420/0,000	NC
OPG	-	-	-	-	na	NC	NC	NC
VTD	NC	-0,314/0,005	NC	NC	NC	-0,272/0,012	-0,281/0,010	NC

Note: NC – no correlation, na – not applicable, r – correlation, p – significant.

[dark grey] – negative correlation [light grey] – positive correlation

According to the analysis of correlation a strong positive dependence of these parameters on each other was revealed (r=0.790, p=0.000). The increase of P1NP and CTX levels strongly correlated with increased content of OC in serum. CTX and P1NP showed slightly negative correlation with BMI and FAT. This proved that the decrease in body weight was accompanied by increase in blood levels of CTX and P1NP. This pattern was more significant between CTX and FAT – during the loss of fat mass an increase of CTX occurred. This might indicate a potential development and progression of osteoporosis after 12 months post-surgery.

Most studies have reported that increased levels of OPG and sRANKL are characteristic of osteoporosis [21–23]. In observing the patients within 12th month, we have found increased level of OPG (p_{12}=0.015) and decreased sRANKL p_{12}=0.84. Such changes are typical for osteoresorption. Taken into consideration a significant change in OPG, we didn't find any dependence in its growth and any of the analyzed parameters (no correlation).

OC is synthesized by osteoblast cells in the late maturation stage, and when it enters the blood, OC behaves as a hormone that has effect on various cells including fat cells. OC levels are related to increase in bone turnover [24]. Significant OC levels increase were seen at the the 3rd month of the post-operative observation. According to the result for the correlation analysis, this increase was related to decrease in BMI and FAT (weak negative correlation).

According to the literature, the level of PTH in the blood after bariatric surgery can change in the direction of its decrease or increase [25–27]. In our study, we note a significant decrease in PTH levels starting from the 3rd month of observation, which is in weak negative correlation with VTD, BMI and FAT. The changes of VTD were significant from the first control month till the end of the observation period. The growth of VTD was remarked. Also, this hypothesis is supported by the works of various authors [28], suggesting that the increase of the VTD is associated with a significant decrease in the volume of adipose tissue in patients, because adipose tissue is a depot for VTD [29]. In our study, we observed slight correlation with the loss of fat mass and with an increase of VTD level, which is confirmed by the increase in VTD that accompanies weight loss.

Thus, the above-revealed changes in serum bone turnover markers are likely to be associated with a significant and clinically apparent loss of total body mass (p_{12}=0.0004; EWL_{12}% 45.8±18.8; EBL_{12}% 55.1±23.4). As the result, there is a reduction of unloading on a skeleton, which leads to a significant, but not clinically important, increase of CTX and P1NP levels. Since the changes in these indicators were not clinically significant in our sample, this has not led to an increase of sRANKL and OPG, and to the changes of DXA results.

Conclusions. Twelve months post LGCP markers of bone turnover (CTX, P1NP, OC, OPG, VTD) were significantly increased. The increased level of VTD is associated with the loss of FAT. Also the change in DXA was detected.

Conflicts of Interest: authors have no conflict of interests.

References

1. World Health Organization. Overweight and Obesity. Retrieved from http://www.who.int/mediacentre/factsheets/fs311/en/.
2. Livingston, E. H. (2010) The incidence of bariatric surgery has plateaued in the U.S. *Am. J. Surg.,* 200(3), 378–385. doi: 10.1016/j.amjsurg.2009.11.007.
3. Peterli, R., Borbély, Y., Kern, B., Gass, M., Peters, T., Thurnheer, M., et al. (2013) Early results of the Swiss Multicentre Bypass or Sleeve Study (SM-BOSS): a prospective randomized trial comparing laparoscopic sleeve gastrectomy and Roux-en-Y gastric bypass. *Ann Surg.,* 258(5), 690–694. doi: 10.1097/SLA.0b013e3182a67426.
4. Sjöström, L. (2013) Review of the key results from the Swedish Obese Subjects (SOS) trial a prospective controlled intervention study of bariatric surgery. *J Intern Med.,* 273(3), 219–234. doi: 10.1111/joim.12012.
5. Talebpour, M., & Amoli, B. S. (2007) Laparoscopic total gastric vertical plication in morbid obesity. *J. Laparoendosc. Adv. Surg. Tech. A.*, 17(6), 793–798. doi: 10.1089/lap.2006.0128.
6. Yu, E. W. (2014) Bone metabolism after bariatric surgery. *J. Bone Miner. Res.,* 29(7), 1507–1518. doi: 10.1002/jbmr.2226.
7. Elias, E., Casselbrant, A., Werling, M., Abegg, K., Vincent, R. P., Alaghband-Zadeh, J., et al. (2014) Bone mineral density and expression of vitamin D receptor-dependent calcium uptake mechanisms in the Proximal small intestine after bariatric surgery. *Br. J. Surg.,* 101(12), 1566–1575. doi: 10.1002/bjs.9626.
8. Liu, C., Wu, D., Zhang, J. F., Xu, D., Xu, W. F., Chen, Y., et al. (2016) Changes in Bone Metabolism in Morbidly Obese Patients After Bariatric Surgery: A Meta-Analysis. *Obes. Surg.,* 26(1), 91–97. doi: 10.1007/s11695-015-1724-5.
9. Stein, E. M., & Silverberg, S. J. (2014) Bone loss after bariatric surgery: causes, consequences, and management. *Lancet Diabetes Endocrinol.,* 2(2), 165–174. doi: 10.1016/S2213-8587(13)70183-9.
10. Buchwald, H., Avidor, Y., Braunwald, E., Jensen, M. D., Pories, W., Fahrbach, K., & Schoelles, K. (2004) Bariatric surgery: a systematic review and meta-analysis. *JAMA.,* 292(14), 1724–1737.
11. Schneider, B. E., & Mun, E. C. (2005) Surgical management of morbid obesity. *Diabetes Care.,* 28(2), 475–480. doi: 10.2337/diacare.28.2.475.
12. Collazo-Clavell, M. L., Jimenez, A., Hodgson, S. F., & Sarr, M. G. (2004) Osteomalacia after Roux-en-Y gastric bypass. *Endocr. Pract.,* 10(3), 195–198.
13. von Mach, M. A., Stoeckli, R., Bilz, S., Kraenzlin, M., Langer, I., & Keller, U. (2004) Changes in bone mineral content after surgical treatment of morbid obesity. *Metabolism.,* 53(7), 918–921. doi: 10.1016/j.metabol.2004.01.015.
14. Slater, G. H., Ren, C. J., Siegel, N. et al. (2004) Serum fat-soluble vitamin deficiency and abnormal calcium metabolism after malabsorptive bariatric surgery. *J. Gastrointest. Surg.,* 8(1), 48–55.
15. Costa, T. L., Paganotto, M., Radominski, R. B., Kulak, C. M., & Borba, V. C. (2015) Calcium metabolism, vitamin D and bone mineral density after bariatric surgery. *Osteoporos Int.*, 26(2), 757–764. doi: 10.1007/s00198-014-2962-4.
16. *World Health Organ Tech Rep Ser.* (2000) Obesity: preventing and managing the global epidemic. Report of a WHO consultation., 894, i-xii, 1–253.
17. Fried, M., Hainer, V., Basdevant, A., Buchwald, H., Deitel, M., Finer, N., et al. (2008) Interdisciplinary European guidelines on surgery of severe obesity. *Obes Facts.*, 1(1), 52–59. doi: 10.1159/000113937.
18. Deitel, M., & Greenstein, R. J. (2003) Recommendations for reporting weight loss. *Obes Surg.*, 13(2), 159–160.
19. Vasikaran, S., Eastell, R., Bruyere, O., Foldes, A. J., Garnero, P., Griesmacher, A. et al. (2011) Markers of bone turnover for the

prediction of fracture risk and monitoring of osteoporosis treatment: a need for international reference standards. *Osteoporos Int.*, 22(2), 391–420. doi: 10.1007/s00198-010-1501-1.
20. (2012) Measurement of C-terminal telopeptide of type I collagen (CTX) in serum. *Clin Biochem.,* 45(12), 928–935.
21. Hofbauer, L. C., & Heufelder, A. E. (2000) The role of receptor activator of nuclear factor-kB ligand and osteoprotegerin in the pathogenesis and treatment of metabolic bone diseases. *J Clin Endocrinol Metab.,* 85, 2355–2363.
22. Simonet, W. S., Lacey, D. L., Dunstan, C. R., Kelley, M., Chang, M. S., Luthy, R., et al. (1997) Osteoprotegerin: A novel secreted protein involved in the regulation of bone density. *Cell*, 89, 309–319. doi: 10.1016/S0092-8674(00)80209-3.
23. Li, J., Sarosi, I., Yan, X. Q., Morony, S., Capparelli, C., Tan, H. L., et al. (2000) RANK is the intrinsic hematopoietic cell surface receptor that controls osteoclastogenesis and regulation of bone mass and calcium metabolism. *Proc Natl Acad Sci USA,* 97, 1566–1571.
24. Delmas, P. D., Eastell, R., Garnero, P., Seibel, M. J., & Stepan, J. (2000) The use of biochemical markers of bone turnover in osteoporosis. Committee of Scientific Advisors of the International Osteoporosis Foundation. *Osteoporos Int*., 11(6), 2–17.
25. Cundy, T., Evans, M. C., Kay, R. G., Dowman, M., Wattie, D., & Reid, I.R. (1996) Effects of vertical-banded gastroplasty on bone and mineral metabolism in obese patients. *Br J Surg.,* 83(10), 1468–1472. doi: 10.1002/bjs.1800831046.
26. Moreiro, J., Ruiz, O., Perez, G., Salinas, R., Urgeles, J. R., Riesco, M., & García-Sanz, M. (2007) Parathyroid hormone and bone marker levels in patients with morbid obesity before and after biliopancreatic diversion. *Obes. Surg.,* 17(3), 348–354. doi: 10.1007/s11695-007-9063-9.
27. Ruiz-Tovar, J., Oller, I., Tomas, A., Llavero, C., Arroyo, A., Calero, A., et al. (2012) Mid-term effects of sleeve gastrectomy on calcium metabolism parameters, vitamin D and parathormone (PTH) in morbid obese women. *Obes. Surg*., 22(5), 797–801. doi: 10.1007/s11695-011-0582-z.
28. Martínez, P., Moreno, I., De Miguel, F., Vila, V., Esbrit, P., & Martínez, M. E. (2001) Changes in osteocalcin response to 1,25-dihydroxyvitamin D(3) stimulation and basal vitamin D receptor expression in human osteoblastic cells according to donor age and skeletal origin. *Bone.*, 29(1), 35–41. doi: 10.1016/S8756-3282(01)00479-3.
29. Holick, M. F. (2007) Vitamin D deficiency. *N. Engl. J. Med.,* 357, 266–281.

Information about authors:
Nykonenko A. O., MD, PhD, DSci., Professor, Department of Hospital Surgery, Zaporizhzhia State Medical University,
E-mail: nikonandra@gmail.com.
Haidarzhi Ye. I., MD, PhD, Associate Professor, Department of Hospital Surgery, Zaporizhzhia State Medical University.
Marek Bužga, MD, PhD, Head of Department of Physiology and Pathophysiology, Faculty of Medicine, University of Ostrava, CZ 70103, Ostrava, Czech Republic. E-mail: marekbuzga@osu.cz.
Відомості про авторів:
Никоненко А. О., д-р мед. наук, професор каф. госпітальної хірургії, Запорізький державний медичний університет,
E-mail: nikonandra@gmail.com.
Гайдаржі Є. І., канд. мед. наук, доцент каф. госпітальної хірургії, Запорізький державний медичний університет.
Бужга Марек, канд. мед. наук, зав. каф. фізіології та патофізіології, факультет медицини, Університет Острава. Острава, CZ 70103, Чеська Республіка. E-mail: marekbuzga@osu.cz.
Сведения об авторах:
Никоненко А. А., д-р мед. наук, профессор каф. госпитальной хирургии, Запорожский государственный медицинский университет,
E-mail: nikonandra@gmail.com.
Гайдаржи Е. И., канд. мед. наук, доцент каф. госпитальной хирургии, Запорожский государственный медицинский университет.
Бужга Марек, канд. мед. наук, зав. каф. физиологии и патофизиологии, факультет медицины, Университет Остравы. Острава, CZ 70103, Чешская Республика. E-mail: marekbuzga@osu.cz.

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