

Changes in bone mineral density in obese perimenopausal women during 5-year follow-up

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KEY WORDS

bone loss, bone mineral density, obesity, perimenopause

ABSTRACT

INTRODUCTION The beneficial effect of obesity on bone mineral density (BMD) has not been definitely established.

OBJECTIVES The aim of the study was to evaluate changes in BMD in obese perimenopausal women during a 5-year follow-up.

PATIENTS AND METHODS The study involved 54 obese women. The group was divided into 2 subgroups according to the menopausal status: postmenopausal women – M (n = 35) and premenopausal women – P (n = 19). Laboratory tests (parathyroid hormone, 25-hydroxyvitamin D₃, C-terminal telopeptide of type I collagen, osteocalcin, and osteoprotegerin), anthropometric measurements, and densitometry were performed twice during the 5-year follow-up. The control group consisted of 19 healthy women of the same age and with normal body weight.

RESULTS Obese postmenopausal women were characterized by lower BMD in the proximal femur and lumbar spine, higher fracture risk, and higher serum osteocalcin levels at baseline. During the 5-year follow-up, there was a 1.52% and 6.86% decrease in proximal femur BMD and 2.34% and 5.17% in lumbar spine BMD (in premenopausal and postmenopausal women, respectively). In postmenopausal controls, BMD reduction was 2.36% and 4.3%, respectively. In the combined analysis including all postmenopausal women, there was an inverse correlation between the initial body mass index and the changes in proximal femur BMD ($r = -0.25$; $P < 0.05$) and lumbar spine BMD ($r = -0.28$; $P = 0.08$) that occurred during the 5-year follow-up.

CONCLUSIONS Obesity appears not to protect against bone mineral loss in postmenopausal women.

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INTRODUCTION The relationship between estrogen deficiency and bone mass decline is well established.¹ In perimenopausal women, physiological bone loss is accelerated and estimated at 2% per year.² This process starts about 1.5 years prior to menopause, and during the next 8 years the rate of bone loss is up to 10.5%.³ Peripheral aromatization of adrenal androstendion, predominantly in the adipose and muscular tissues is the main source of estrogens (estron) in postmenopausal women.^{4,5} In obese women,

the increased production of adrenal androstendion and peripheral conversion,⁶ partially due to lower serum levels of sex hormone-binding globulin followed by high availability of precursors, is observed.⁷ Thus, in obese women, the level of free sex hormones is relatively higher than in nonobese women.^{8,9}

It has also been shown that high estrogen levels in mesenchymal bone marrow cells directly stimulate osteogenesis and inhibit differentiation of mesenchymal cells into adipocytes.¹⁰ The more

reduced levels of endogenous estrogen are related to increased amount of fat tissue, which may be one of those mechanisms that prevent sudden endogenous estrogen decrease in perimenopausal women.¹¹ This phenomenon, as well as the increased production of estrogens by adipose tissue, partially explains the protective effect of obesity on bone tissue and the lower prevalence of postmenopausal osteoporosis in women with simple obesity and without concomitant diseases.

It is well known that obese perimenopausal women have higher bone mineral density (BMD) compared with women with normal body weight. Evaluation of bone turnover markers suggests a slower rate of bone turnover in those patients.¹² The association between increased weight and bone mass was observed both in adults and children.¹³⁻¹⁵ Tomkinson et al.¹⁶ found that the body mass index (BMI) correlated positively with BMD in postmenopausal women, independently of estrogen levels. In line with this finding, the lower prevalence of osteoporosis in postmenopausal women with simple obesity and without comorbidities was shown. However, some authors did not confirm the protective effect of obesity on the development of osteoporosis.^{17,18} Moreover, obese subjects are characterized by more frequent disorders of calcium-phosphate homeostasis and bone metabolism.¹⁵ Thus, the protective effect of obesity on bone tissue has not been unequivocally demonstrated. There are few longitudinal studies evaluating changes in BMD and parameters of bone turnover in obese women. Studies published so far by Fogelholm et al.,¹⁹ Hinton et al.,²⁰ Compston et al.,²¹ Redman et al.,²² and Villareal et al.²³ were performed in obese but not perimenopausal women, and thus the protective effect of obesity during perimenopause could not be demonstrated. Recently, the results of a large observational, population-based study by Compston et al.²⁴ were published. The authors evaluated only BMD changes during follow-up and showed that obesity does not protect against fracture in postmenopausal women. However, they did not exclude concomitant disorders that might have affected bone tissue and bone metabolism (asthma, emphysema, and type 1 diabetes).

The above facts prompted us to evaluate the changes in BMD in obese perimenopausal women without concomitant diseases during a 5-year follow-up.

PATIENTS AND METHODS The study involved 54 subjects selected from the group of 60 women with simple obesity, without concomitant diseases, and reexamined after 5 years. The group of 60 patients with simple obesity was chosen from more than 400 patients evaluated in an ambulatory clinic. They were precisely selected to avoid concomitant diseases that might have affected bone metabolism and obtained at least 10% weight loss during a 3-month therapy. The inclusion criteria were as follows: BMI ≥ 30 kg/m², normal bone mineralization (T score > -1.0),

normal lipid profile and serum glucose levels. Patients with arterial hypertension (blood pressure $> 140/90$ mmHg), chronic inflammatory diseases, smokers, and users of any medications potentially interfering with bone metabolism, including contraceptive drugs and hormone replacement therapy, were excluded. The study protocol was approved by the Bioethical Committee of the Medical University of Silesia (NN-013-64/03). Each participant was informed about the study design and signed the consent form.

Patients All subjects participated in a 3-month weight loss therapy that involved a recommended 1000–1200 kcal/day balanced diet (daily calcium consumption of 500 mg, carbohydrates – 60%, protein – 15%, fat – 25%), change of lifestyle, and regular physical exercise (60 minutes, 3 times a week). Each patient was examined every 2 weeks by a physician and a dietician and was asked to keep a food diary. Following the food diary was the *sine qua non* of the weight loss therapy. The result of a 10% weight loss was the proof that patients followed dietary advice.

Patients were divided according to their hormonal status into 2 subgroups: postmenopausal women – M (n = 35) and premenopausal women – P (n = 19). The menopausal status was determined based on the date of the last menses, which occurred at the mean age of 51 years in the postmenopausal group.

Blood analysis and anthropometric measurements (body weight, height, waist circumference) were made twice, after the 3-month weight loss therapy and 5 years after the therapy (± 3 months). Venous blood samples (20 ml) were withdrawn into plastic test tubes (Vacutiner system). Serum samples obtained after centrifugation (1000 G for 10 min) were stored at -70°C until the assessment of parathyroid hormone (PTH), 25-hydroxyvitamin D₃ (25(OH)D₃), C-terminal telopeptide of type I collagen (CTX₁), osteocalcin (OC), and osteoprotegerin levels. Serum calcium, inorganic phosphate, and creatinine levels were measured as part of routine work-up. Estimated glomerular filtration rate was calculated according to the Chronic Kidney Disease Epidemiology Collaboration formula.¹⁴ Dual energy X-ray absorptiometry (DXA) was performed twice, at baseline and at 5 years.

The control group consisted of 19 healthy, non-obese, postmenopausal women (BMI, 21.2 kg/m²). The same laboratory, anthropometric measurements, and DXA were performed.

Total absolute fracture risk was estimated using FRAX® – the World Health Organization Fracture Risk Assessment Tool (<http://www.shef.ac.uk/FRAX/tool.jsp>). Characteristics of the study group and controls are presented in TABLE 1.

Anthropometric measurements Weight and height were measured when fasting using electronic medical scales (RADWAG). The BMI was calculated according to the standard formula: BMI = weight

TABLE 1 Characteristics of obese women and controls at baseline

	Premenopausal	Postmenopausal		Statistical significance M vs. C <i>P</i>
	obese (n = 19) <i>P</i>	obese (n = 35) <i>M</i>	controls (n = 19) <i>C</i>	
age, y	45 ^a (44–46)	53 (52–55)	54 (51–56)	NS
body weight, kg	94.1 (87.5–100.7)	94.3 (90.6–98.0)	63.3 (60.6–66.1)	<0.001
fat content, %	47.5 (45.1–50.0)	49.5 (46.9–52.2)	34.3 (32.1–36.6)	<0.001
BMI, kg/m ²	35.9 (33.7–38.1)	36.7 (35.2–38.3)	24.0 (23.2–25.4)	<0.001
waist circumference, cm	99.5 (93.4–105.8)	106.0 (102.3–109.7)	82.8 (77.9–85.9)	<0.001
BMD L ₂ –L ₄ , g/cm ²	1.31 ^b (1.24–1.39)	1.22 (1.16–1.27)	1.06 (1.00–1.13)	<0.001
BMD of the proximal femur, g/cm ²	1.14 ^b (1.08–1.21)	1.05 (0.99–1.13)	0.86 (0.81–0.91)	<0.001
BMC of the proximal femur, g	5.8 (5.45–6.15)	5.41 (5.02–5.80)	4.36 (3.98–4.73)	<0.001
FRAX, %	1.11 ^a (0.97–1.25)	1.47 (1.29–1.66)	2.19 (1.78–2.59)	<0.001
total calcium, mmol/l	2.27 (2.24–2.32)	2.29 (2.27–2.32)	2.36 (2.29–2.44)	NS
phosphorus, mmol/l	1.14 (1.05–1.23)	1.17 (1.08–1.25)	1.34 (1.24–1.44)	<0.01
25(OH)D ₃ , ng/ml	24.8 (19.3–30.3)	28.5 (23.3–33.8)	40.2 (31.2–49.2)	<0.01
PTH, pg/ml	56.4 (48.2–64.5)	53.0 (46.1–60.0)	39.4 (27.1–46.1)	<0.01
osteocalcin, ng/ml	17.9 ^c (15.5–20.3)	24.0 (20.9–27.0)	26.9 (22.4–31.3)	NS
CTX ₁ , ng/ml	0.25 (0.19–0.30)	0.33 (0.27–0.39)	0.30 (0.24–0.37)	NS
osteoprotegerin, pmol/l	4.48 (3.80–5.17)	4.64 (4.12–5.16)	5.59 (5.33–5.84)	<0.01

Data are presented as mean ± 95% CI.

Statistical significance M vs. C

a *P* < 0.001

b *P* < 0.05

c *P* = 0.01

Abbreviations: BMC – bone mineral content, BMD – bone mineral density, BMI – body mass index, CI – confidence interval, CTX₁ – C-terminal telopeptide of type I collagen, FRAX – Fracture Risk Assessment Tool (10-year absolute fracture risk), NS – nonsignificant, PTH – parathyroid hormone

(kg) / height (m²). Waist circumference was measured between the lower costal arch and upper iliac crest. Body composition was analyzed using a bioimpedance method (Bodystat 1500, Bodystat Ltd., Great Britain).

Laboratory measurements Laboratory measurements were conducted in the Isotopic Laboratory of the Department of Nephrology, Endocrinology and Metabolic Diseases and in the Department of Pathophysiology, Medical University of Silesia, Katowice, Poland. The electrochemiluminescence immunoassay (Elecsys, Roche Diagnostics GmbH,

Germany) was used to measure PTH, OC, and CTX₁ levels. 25(OH)D₃ was measured using the radioimmunoassay technique (Bio Source-EUROPE S.A, Nivelles, Belgium). Osteoprotegerin was measured using an enzyme-linked immunosorbent assay (Biovendor, Modřice, Czech Republic). Total calcium and inorganic phosphate levels were measured using a spectrophotometer (Point Scientific Inc., Michigan, United States).

Densitometry measurements DXA of the lumbar spine and proximal femur for BMD and bone mineral content (BMC) determinations was

TABLE 2 Changes in bone mineral density and body mass as well as fat tissue in post- and premenopausal women during 5-year follow-up

	Obese		Controls
	premenopausal n = 19	postmenopausal n = 35	postmenopausal n = 19
Δ body mass, %	0.1 (-4.6 to 4.7)	-1.5 (-4.1 to 1.0)	1.7 (-1.3 to 4.7)
Δ FAT content, %	2.7 (-0.8 to 6.1)	-0.4 (-2.6 to 6.9)	3.0 (-0.8 to 6.9)
Δ BMD L ₁ -L ₄ , %	-2.34 ^a (-4.24 to -0.44)	-5.17 ^{a,b} (-9.89 to -0.46)	-4.30 ^a (-11.41 to 2.81)
Δ BMD of the proximal femur, %	-1.52 ^a (-4.11 to -1.07)	-6.86 ^{a,d} (-10.11 to -3.61)	-2.36 ^a (-6.88 to 2.16)
Δ osteocalcin, %	3.8 (-10.6 to 18.2)	-5.2 (-17.4 to 7.0)	3.3 (-20.7 to 27.3)
Δ osteoprotegerin, %	-9.4 (-38.8 to 20.0)	-23.5 (-31.4 to -15.6)	-21.8 (-30.0 to -13.7)
Δ PTH, %	-3.3 (-25.4 to 18.7)	-3.4 (-16.5 to 9.7)	0.3 (-20.2 to 20.9)
Δ CTX ₁ , %	3.3 (-13.5 to 20.0)	-10.8 (-26.9 to 5.4)	13.8 (-34.8 to 62.5)
Δ 25(OH)D ₃ , %	25.8 (3.2-48.4)	29.6 (10.1 to 49.1)	-6.6 ^c (-28.4 to 15.1)

Data are presented as mean ± 95% CI.

Statistical significance vs. baseline: **a** $P < 0.01$

Statistical significance vs. postmenopausal controls: **b** $P < 0.05$; **c** $P < 0.01$

statistical significance postmenopausal vs. premenopausal: **d** $P < 0.01$

Abbreviations: see **TABLE 1**

performed using a Lunar Prodigy Advance scanner (GE Healthcare, Diegem, Belgium).

Statistical analysis All values were expressed as mean ± 95% confidence interval. All statistical analyses were performed using the STATISTICA 8.0 PL software. The distribution was analyzed using the Kolmogorov-Smirnoff test. Because of the size of the control group and the subgroups and the nonparametric distribution of some of the parameters, the Mann-Whitney test was used to compare the study groups. Parameter variation in time was evaluated using the analysis of variance. Correlation coefficients were calculated according to Spearman. $P < 0.05$ was considered statistically significant.

RESULTS Characteristics of the study groups Compared with nonobese women, obese postmenopausal subjects were characterized by higher BMD of the lumbar spine and femoral neck, lower 10-year absolute fracture risk, lower serum levels of osteoprotegerin, 25(OH)D₃, and phosphorus, and higher serum PTH levels (**TABLE 1**). Obese postmenopausal women were on average 8 years older (subgroup M) compared with obese premenopausal women (subgroup P). During the follow-up, none of the premenopausal women entered menopause. As expected, the M subgroup was characterized by lower BMD in both regions, higher fracture risk, and higher serum OC levels

(**TABLE 1**). During the follow-up, we did not observe any fractures either in obese or nonobese subjects.

Changes of bone mineral density during 5-year follow-up At 5 years, body weight and fat mass in obese postmenopausal women were lower than at baseline, by 1.5% and 0.4% on average, respectively. At the same time, body weight and fat mass in obese premenopausal women increased by 0.1% and 2.7%, respectively (**TABLE 2**).

At 5 years, lower BMD in the lumbar spine and proximal femur and lower BMC in the proximal femur were observed in both obese subgroups and controls. Of note, higher but statistically nonsignificant loss of BMD ($P = 0.07$) was observed in obese women (**FIGURES 1** and **2**). During the 5-year follow-up, proximal femur BMD decreased by 6.86% and lumbar spine BMD by 5.17% in obese postmenopausal women.

In obese premenopausal women, the decrease was much smaller: 1.52% for proximal femur BMD and 2.34% for lumbar spine BMD (**FIGURES 3** and **4**). In postmenopausal controls, the decrease was 2.36% for proximal femur BMD and 4.30% for lumbar spine BMD (**TABLE 2**). Significant changes in BMD (at least 3%) in the proximal femur and lumbar spine were observed in 9 and 9 obese premenopausal women, 30 and 20 obese postmenopausal women, and 13 and 11 controls, respectively.

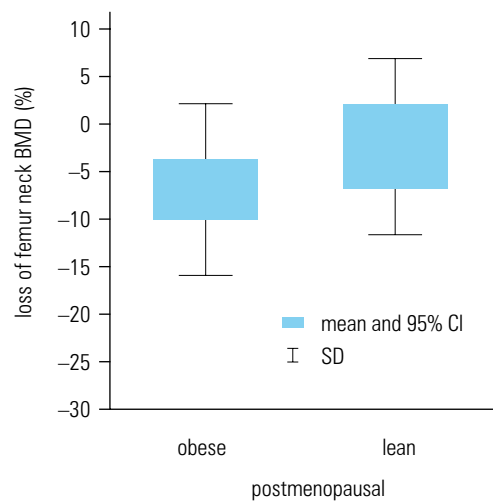


FIGURE 1 Loss of proximal femur bone mass density in obese postmenopausal women and controls during 5-year follow-up
Abbreviations: SD – standard deviation, others – see [TABLE 1](#)

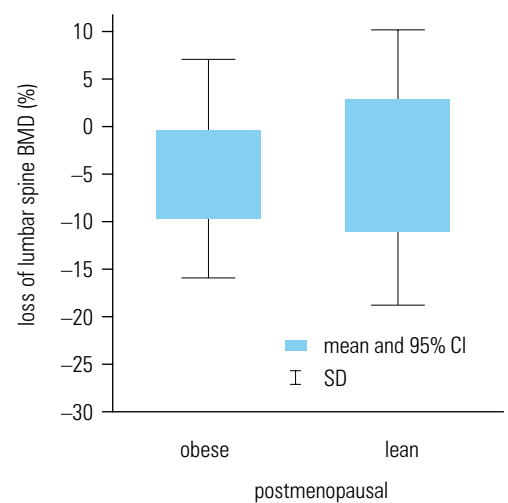


FIGURE 2 Loss of lumbar spine bone mineral density in obese postmenopausal women and controls during 5-year follow-up
Abbreviations: see [TABLE 1](#) and [FIGURE 1](#)

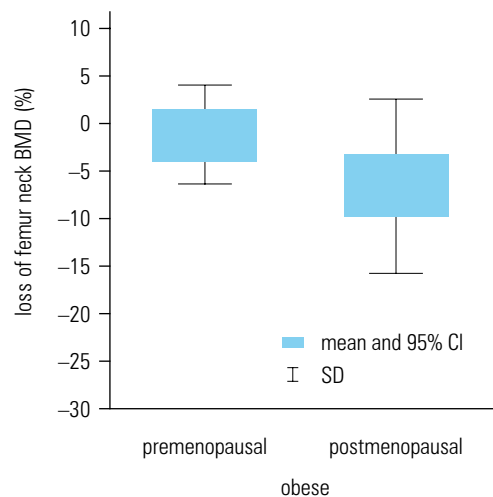


FIGURE 3 Loss of proximal femur bone mass density in obese pre- and postmenopausal women during 5-year follow-up
Abbreviations: see [TABLE 1](#) and [FIGURE 1](#)

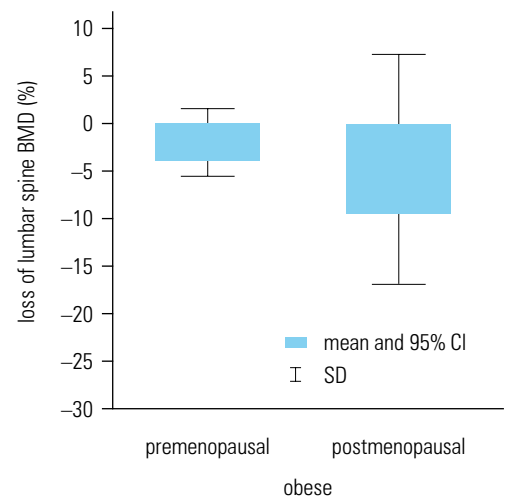


FIGURE 4 Loss of lumbar spine bone mineral density in obese pre- and postmenopausal women during 5-year follow-up
Abbreviations: see [TABLE 1](#) and [FIGURE 1](#)

Effect of changes in body mass on bone mass density

In the combined analysis including all postmenopausal women, there was an inverse correlation between the initial BMI and the changes in proximal femur BMD ($r = -0.25$, $P < 0.05$) and lumbar spine BMD ($r = -0.28$, $P = 0.08$) that occurred over the 5-year follow-up.

DISCUSSION Our study supplements the current knowledge with the results on the changes in BMD during premenopausal and early postmenopausal periods. We showed that obesity does not protect against bone mineral loss in the early postmenopausal period. During the 5-year follow-up, BMD loss in obese postmenopausal women was more than twice as high as in premenopausal women

in the lumbar spine (5.17% vs. 2.34%). The difference in BMD loss in the proximal femur was even greater (6.86% vs. 1.52%). Moreover, in obese patients BMD loss in the proximal femur was significantly higher than in nonobese women in the early postmenopausal period (6.86% vs. 2.36%). Recently, the results of a large multinational, prospective, observational, population-based study by Compston et al.²⁴ were published, showing that obesity does not protect against fracture in postmenopausal women. The risk of incident ankle and upper leg fracture was significantly higher in obese women compared with nonobese women, while the risk of wrist fracture was significantly lower. Obese women with fracture were more likely to have experienced early menopause and to

report 2 or more falls in the past year. The above study evaluated changes in BMD during a follow-up. However, the authors did not exclude patients with concomitant diseases that might have affected BMD.

Because obesity does not protect against BMD loss in the postmenopausal period and does not slow down the process of bone mineral loss, lower incidence of osteoporosis in obese postmenopausal women seems to be mainly related to higher peak bone mass. Thus, the increased risk of fractures will occur in obese women with delay.

In our study, women with simple obesity were characterized by higher BMD in the lumbar spine and proximal femur, but lower 10-year absolute fracture risk. Serum OC, osteoprotegerin, 25(OH)D₃, and phosphorous levels were lower and serum PTH levels were higher compared with controls. The both subgroups of postmenopausal women were characterized by significantly higher OC levels compared with obese premenopausal women. We did not observe any significant changes in bone turnover markers during the follow-up (data not shown). Higher OC levels in both postmenopausal groups may suggest an increased osteogenesis in the early postmenopausal period in obese women rather than inhibited osteogenesis as previously reported.^{25,26}

Increased OC levels were previously reported in postmenopausal women. Iki et al.²⁷ observed 71% higher OC levels in postmenopausal vs. premenopausal Japanese women. Similar differences were observed by Garnero et al.²⁸ in Caucasian population. In other studies by Iki et al.,^{29,30} the authors suggested that elevated OC levels may predict bone loss in perimenopausal women independently of age and body size. These results are consistent with our findings, because obese postmenopausal women in our study were characterized by lower BMD and higher serum OC levels than premenopausal women. Lofman et al.³¹ reported a 40% increase in OC levels within the first year of menopause, followed by a slow continuous increase up to 75 years of age. However, other investigators showed either a decrease³² or no change with age.³³ This might be explained by the fact that OC is more abundant in cortical (which is less metabolically active) than in trabecular bone,³⁴ and the cortical bone may release OC later in life when the compartment of compact bone is affected to a greater extent.³⁵ Additionally, the glomerular filtration rate, which decreases with age, may be responsible for lower OC clearance and, in consequence, for an increase in its levels. Another possible explanation is that the posttranslational carboxylation of OC is dependent on vitamin K and that vitamin K deficiency is more common in older subjects. Thus, higher OC levels may be caused by an increase in undercarboxylated fraction exclusively in postmenopausal (older) women.³⁶ An inverse relationship between serum OC levels and BMD depletion supports the hypothesis that OC may act as a negative stimulator of bone formation

without affecting bone resorption.³⁷ However, bone resorption driven by lack of estrogen predominates in postmenopausal women.

In 2010, we published an analysis involving a large proportion of the current group of patients, but focused on bone metabolism only in obese women after a short-term weight loss therapy during a 5-year follow-up, without dividing patients according to their menopausal status.³⁸ In this smaller group, we observed a similar decrease in BMD in the lumbar spine and femoral neck, and a comparable decrease in the serum levels of CTX₁ and OC in both obese and nonobese subjects during the 5-year follow-up. Changes in serum PTH levels were statistically nonsignificant. In obese women, a nonsignificant increase in the serum level of 25(OH)D₃ was observed as early as after a 3-month weight loss therapy and the change was maintained during follow-up. In controls, serum 25(OH)D₃ levels tended to decrease. During follow-up, the number of obese patients with disturbances in vitamin D metabolism decreased from 78.7% to 53.2% ($P = 0.01$), which constitutes the most important finding of the above study. Such disturbances were observed in 35.3% of the controls. We also observed a positive correlation between the change in body mass and BMD in the proximal femur in obese patients ($r = 0.279$, $P = 0.04$).

The other above-mentioned studies evaluating BMD and parameters of bone turnover in obese women were not performed during the perimenopausal period. Fogelholm et al.¹⁹ assessed changes in BMD in 74 obese premenopausal women (aged 30–45 years) after a 3-month weight loss therapy and weight regain during a 36-month follow-up. They reported a slight decrease in BMD at 3 months that was partially abolished after weight regain. In a recent study, Hinton et al.²⁰ evaluated the effect of a 12-week weight reduction program (1200 kcal/24 h) with a 24-week follow-up on bone metabolism in 113 obese patients in their forties (49 men and 64 women). Apart from reduced serum PTH levels, they showed no significant effect of weight loss on either BMD or total BMC. Compston et al.,²¹ in a small group of obese women undergoing a 10-week weight loss therapy ($n = 8$), observed that BMD returned to baseline after 10 months, during which women regained their weight. In another study, Redman et al.²² reported no significant effect of 10% weight reduction on BMD during a 6-month follow-up of 64 obese individuals. However, Hinton et al.³⁹ in 37 obese patients (aged 50 ± 9.8 years; 13 men and 24 women) and Villareal et al.²³ in 27 senile obese patients (aged 70 ± 5.0 years) reported significant reductions in BMD and increased levels of bone turnover markers during a 24-week and 12-week weight loss program with 6- and 9-month follow-up, respectively.

However, the above studies did not involve obese perimenopausal women and thus the pro-

tective effect of obesity in this particular period could not be demonstrated or denied.

Bone demineralization may be also affected by microinflammation, independently of the impaired production of estradiol by the ovaries. Higher levels of proinflammatory cytokines have osteoclastic effect, as shown previously.⁴⁰ Both animal (rodent) and human studies have reported that proinflammatory cytokines produced by adipose tissue, such as interleukin 6 (IL-6) or tumor necrosis factor α , stimulate osteoclastogenesis.^{41,42} Adipose tissue is responsible for the production of one-third of the amount of IL-6. Higher IL-6 levels were observed in obese compared with non-obese subjects.⁴³ Thus, inflammation may participate in bone loss in obese patients, which is in line with the study by Hsu et al.⁴⁴ who reported even higher risk of osteopenia, osteoporosis, and nonvertebral fractures in patients with elevated fat mass, regardless of body weight.

There are numerous other factors, beside menopausal status and microinflammation, that may affect bone mass, for example lifestyle (physical activity), which stimulates bone mineralization and at the same time reduces fat mass. These other factors were not examined in our study, which constitutes its main limitation. Another limitation is the lack of measurement of daily phosphorus consumption that may affect bone metabolism. Nevertheless, our study demonstrated that estrogen deficiency has a crucial role in BMD loss in the perimenopausal period.

In conclusion, it seems that obesity does not protect against bone mineral loss in postmenopausal women.

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Zmiany gęstości mineralnej kości u otyłych kobiet w wieku okołomenopauzalnym w obserwacji 5-letniej

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SŁOWA KLUCZOWE

gęstość mineralna kości, otyłość, okres okołomenopauzalny, ubytek masy kostnej

STRESZCZENIE

WPROWADZENIE Dotychczas nie udało się jednoznacznie dowieść, że otyłość ma korzystny wpływ na gęstość mineralną kości (*bone mineral density* – BMD).

CELE Celem badania była ocena zmian BMD u otyłych kobiet w wieku okołomenopauzalnym w 5-letniej obserwacji.

PACJENCI I METODY W badaniu wzięły udział 54 otyłe kobiety. Badaną grupę podzielono na dwie podgrupy w zależności od statusu menopauzalnego: kobiety po menopauzie – M (n = 35) i przed menopauzą – P (n = 19). W trakcie 5-letniej obserwacji 2-krotnie wykonano badania laboratoryjne (parathormon, 25-hydroksywitamina D₃, C-końcowy usieciowany telopeptyd kolagenu typu 1, osteokalcyna oraz osteoprotegeryna), pomiary antropometryczne i badanie densytometryczne. Grupę kontrolną stanowiło 19 zdrowych kobiet w tym samym wieku, z prawidłową masą ciała.

WYNIKI U otyłych kobiet po menopauzie stwierdzono mniejszą BMD bliższego końca kości udowej oraz odcinka lędźwiowego kręgosłupa, większe ryzyko złamania i większe stężenie osteokalcyny w surowicy na początku obserwacji. W 5-letniej obserwacji stwierdzono zmniejszenie BMD bliższego końca kości udowej o 1,52% i 6,86% oraz zmniejszenie BMD odcinka lędźwiowego kręgosłupa o 2,34% i 5,17% (odpowiednio u kobiet przed menopauzą i po menopauzie). W grupie kontrolnej u kobiet po menopauzie zmniejszenie BMD wynosiło 2,36% i 4,3%. W analizie obejmującej wszystkie kobiety po menopauzie stwierdzono ujemną korelację pomiędzy wskaźnikiem masy ciała a zmianami BMD bliższego końca kości udowej (R = -0,25; p < 0,05) i odcinka lędźwiowego kręgosłupa (R = -0,28; p = 0,08), które nastąpiły w trakcie 5-letniej obserwacji.

WNIOSKI Jak się wydaje, otyłość nie chroni przed ubytkiem masy kostnej u kobiet po menopauzie.

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