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PRACA ORYGINALNA
ORIGINAL PAPER

Assessment of bone metabolism and fracture risk in obese men

Ocena metabolizmu kostnego i ryzyka złamań u otyłych mężczyzn

Małgorzata N. Grabarczyk[1], Marta J. Gorczyca[1], Paulina Kosińska[1], Katarzyna Klimek[1], Paweł Cieślik[2], Michał T. Holecki[2]

[1]Studenckie Koło Naukowe przy Katedrze i Klinice Chorób Wewnętrznych, Autoimmunologicznych i Metabolicznych, Wydział Nauk Medycznych w Katowicach, Śląski Uniwersytet Medyczny w Katowicach
[2]Katedra i Klinika Chorób Wewnętrznych, Autoimmunologicznych i Metabolicznych, Wydział Nauk Medycznych w Katowicach, Śląski Uniwersytet Medyczny w Katowicach

ABSTRACT

INTRODUCTION: Obesity and metabolic syndrome are increasingly common in the adult population. There is a well-known relationship between those two conditions and cardiovascular diseases; nonetheless, not much is known about how obesity and metabolic syndrome affect bone metabolism and fracture risk. The study aimed to assess the parameters of bone metabolism, as well as assess their relationship with the risk of fractures in obese men with central obesity and metabolic syndrome, and to compare the obtained results with those of healthy controls.

MATERIAL AND METHODS: The study involved 36 obese men (body mass index – BMI ≥ 30) with central obesity (waist circumference – WC ≥ 94) and 10 healthy men as controls, aged 54–77. The FRAX (Fracture Risk Assessment Tool) calculator was used to measure the 10-year fracture risk. The levels of bone metabolism markers osteoprotegerin (OPG), C-terminal telopeptide (CTX_1), and fibroblast growth factor 23 (FGF-23) were determined in the patients.

RESULTS: The FRAX parameter was significantly lower ($p < 0.001$) in the obese men when compared to the controls. A significant negative correlation between FRAX and BMI ($p < 0.001$) was observed in the obese men, but not in the healthy subjects. There was also a negative correlation between FRAX and WC ($p < 0.001$), again only among the obese subjects. A positive correlation ($p < 0.01$) between FGF-23 and FRAX was found in the non-obese group.

CONCLUSIONS: Obese men have a lower 10-years fracture risk compared to the healthy controls. Additionally, the increased BMI and waist circumference in the obese men were found to be associated with a reduced bone fracture risk, whereas no similar relationship in controls was observed. Moreover, higher FGF-23 levels in the healthy males was correlated with an increased 10-year fracture risk.

KEY WORDS

metabolic syndrome, obesity, OPG, osteoporosis, FRAX, bone fractures, FGF-23, male

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Address for correspondence: Małgorzata N. Grabarczyk, Studenckie Koło Naukowe przy Katedrze i Klinice Chorób Wewnętrznych, Autoimmunologicznych i Metabolicznych, Wydział Nauk Medycznych w Katowicach, Śląski Uniwersytet Medyczny w Katowicach, ul. Medyków 14, 40-752 Katowice, tel. +48 32 789 43 01, e-mail: mgrabarczyk@icloud.com

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STRESZCZENIE

WSTĘP: Otyłość oraz zespół metaboliczny coraz częściej występują w populacji osób dorosłych. Powszechnie znany jest związek wymienionych zaburzeń ze zwiększonym prawdopodobieństwem wystąpienia chorób układu sercowo-naczyniowego, jednakże mniej oczywisty jest ich wpływ na metabolizm kostny oraz ryzyko złamań. Celem badania była ocena parametrów metabolizmu kostnego, ich związku z ryzykiem złamań u otyłych mężczyzn z otyłością brzuszną oraz zespołem metabolicznym, a także porównanie uzyskanych wyników z wynikami osób zdrowych.

MATERIAŁ I METODY: W badaniu wzięło udział 36 otyłych mężczyzn (*body mass index* – BMI ≥ 30) ze współistniejącą otyłością trzewną (obwód talii – *waist circumference* – WC ≥ 94) oraz 10 zdrowych mężczyzn z grupy kontrolnej, w wieku 54–77 lat. Do oceny ryzyka złamań zastosowano kalkulator FRAX (Fracture Risk Assessment Tool). U pacjentów oznaczono stężenia markerów metabolizmu kostnego: osteoprotegeryny (OPG), C-końcowego usieciowanego telopeptydu łańcucha kolagenu typu I (CTX_1) oraz czynnika wzrostu fibroblastów 23 (*fibroblast growth factor 23* – FGF-23).

WYNIKI: U osób otyłych FRAX był istotnie niższy ($p < 0{,}001$) niż w grupie kontrolnej. Zaobserwowano ujemną korelację między FRAX i BMI ($p < 0{,}001$) u otyłych mężczyzn. U zdrowych osób taka korelacja nie wystąpiła. Jedynie u osób otyłych stwierdzono również ujemną korelację między FRAX i WC ($p < 0{,}001$). Obecnej w grupie osób zdrowych pozytywnej korelacji ($p < 0{,}01$) między FGF-23 i FRAX nie obserwowano u otyłych mężczyzn.

WNIOSKI: U otyłych mężczyzn stwierdzono mniejsze 10-letnie ryzyko złamań w porównaniu z osobami zdrowymi. Dodatkowo wykazano, że w grupie pacjentów otyłych większe BMI oraz obwód talii wiązały się z mniejszym ryzykiem złamań kości, natomiast u osób bez otyłości taka zależność nie występowała. Ponadto u zdrowych mężczyzn większe stężenie FGF-23 było skorelowane ze zwiększonym 10-letnim ryzykiem złamań.

KEY WORDS

zespół metaboliczny, otyłość, OPG, osteoporoza, FRAX, złamania, FGF-23, mężczyźni

INTRODUCTION

Obesity and osteoporosis are both associated with a sedentary lifestyle, low level of physical activity, improper diet, and aging [1,2]. The rising occurrence of both conditions has been a subject of interest among researchers for many years. However, the results of studies on the relationship between osteoporosis and obesity are still controversial.

According to the results of the WOBASZ II (Wieloośrodkowe Ogólnopolskie Badanie Stanu Zdrowia Ludności, edycja II) study, approximately 25% of adult women and 24.4% of adult men in Poland were obese [3]. World Health Organization (WHO) defined obesity as a body mass index (BMI) equal to 30 kg/m^2 or above [4]. Moreover, BMI is a simple, widely available indicator, which correlates with the percentage of body fat [5]. Nevertheless, this may not be fully accurate as it does not consider gender differences, nor does it distinguish between fat and muscle mass; additionally, it may be less sensitive in children and the elderly [6,7].

Metabolic syndrome (MS) is defined as a set of interconnected factors that directly increase the risk of atherosclerosis, type 2 diabetes (DMT2), and secondary cardiovascular complications, and the most important component of MS is central obesity [8]. According to the International Diabetes Federation, MS is present if the patient has central obesity (defined as abnormal, ethnicity-specific waist circumference (WC), which is ≥ 80 cm for European women and ≥ 94 cm for European men) plus two or more of the following criteria:

- triglycerides (TG) ≥ 150 mg/dL or specific treatment for this lipid abnormality,
- high density lipoprotein cholesterol (HDL-C) < 50 mg/dL in females and < 40 mg/dL in males or specific treatment for this lipid abnormality,
- blood pressure (BP) ≥ 130/≥ 85 mmHg or treatment of previously diagnosed hypertension,
- fasting glucose ≥ 100 mg/dL or previously diagnosed DMT2 [8].

Osteoporosis is a systemic skeletal disease characterized by reduced bone mineral density (BMD), leading to an increased risk of fractures. A person with osteoporosis may suffer a bone fracture after an injury that in healthy persons a fracture usually would not occur [9].

Nonetheless, the majority of fractures occur in patients who do not fulfill the WHO criteria of osteoporosis [10,11]. Thus, the densitometry is not sensitive enough to predict the risk of fracture. The FRAX (Fracture Risk Assessment Tool) algorithm is used to assess the 10--year probability of a major osteoporotic fracture. This tool merges the clinical risk factors and BMD at the femoral neck to identify the patients who ought to be treated [12,13].

Osteoprotegerin (OPG) is a protein that inhibits the differentiation and maturation of osteoclasts, inhibits the activity of mature osteoclasts and their precursor cells by inducing their apoptosis. OPG suppresses the maturation of osteoclasts by its fusion with RANKL (receptor activator of nuclear factor κB ligand) and blocks interactions between RANKL and RANK [14]. OPG plays an osteoprotective role and its production is stimulated by estrogens [15]. Therefore, we included OPG to determine whether it can be a reliable

biomarker of low BMD and osteoporosis in men, as it can be for postmenopausal women [16].

Fibroblast growth factor 23 (FGF-23) is a hormone that regulates serum phosphate and $1,25(OH)_2D_3$ levels. FGF-23 reduces the concentration of phosphate by inhibiting reabsorption in proximal tubules and also inhibits the 1-alpha-hydroxylation of 25-OH-D_3 [17]. FGF-23 acts as an inhibitor of bone mineralization [17]. C-terminal telopeptide (CTX_1), produced by active osteoclasts, is one of the best indicators of bone resorption [18]. The intensification of bone turnover is associated with a higher serum level of CTX_1 [18]. Combining the measurement of BMD, FRAX, and bone turnover markers may improve the evaluation of fracture risk, especially in those treated with antiresorptive treatment [19].

Both MS and bone fracture are important problems in middle-aged and older adults, which cause a deterioration in the quality of life and lead to an increase in mortality. Obesity is both strongly associated with and has an impact on bone metabolism [20]. It was also demonstrated that MS can be an additional risk factor for osteoporotic fractures [21]. The authors found that when adjusted for BMI, the BMD in patients with MS was lower than in those without. Additionally, patients with MS, despite a higher number of observed falls, had a lower number of fractures. Studies of bone metabolism in obese men are rare, and little information is available about the effects of MS on bone metabolism in men.

The study aimed to:

- ascertain whether bone metabolism is affected in obese patients with central obesity in comparison to patients with additional MS risk factors in relation to healthy controls,
- evaluate the OPG, FGF-23, and CTX_1 concentrations in obese subjects with central obesity, MS, and healthy controls,
- assess the probability of serious osteoporotic fracture (FRAX) in obese men.

MATERIAL AND METHODS

The study group consisted of 36 obese men with coexisting central obesity (all obese group; N = 36, BMI = 33.80 ± 3.53 kg/m^2, aged 61.06 ± 5.62 years) from the Outpatient Weight Management Clinic.

The study group was divided into 2 subgroups: obese patients with central obesity (N = 18, BMI = 33.40 ± 3.76 kg/m^2, aged 60.39 ± 5.76 years) and obese patients with metabolic syndrome (N = 18, BMI = 34.19 ± 3.35 kg/m^2, aged 61.72 ± 5.56 years).

The inclusion criteria in the subgroup of obese patients with central obesity were:

- WC ≥ 94 cm,
- BMI ≥ 30 kg/m^2,
- stable body weight within 3 months prior to the study,
- normal lipid profile and glucose,
- BP < 130/85 mmHg,
- no history of chronic inflammatory diseases,
- no drugs affecting bone metabolism,
- informed consent to participate in the study.

The inclusion criteria in the subgroup of obese patients with MS were:

- BMI ≥ 30 kg/m^2,
- WC ≥ 94 cm,

and coexistence 2 of 4 of the following abnormalities:

- serum TG level > 150 mg/dL,
- serum HDL-C level < 40 mg/dL in men and < 50 mg/dL in women or specific treatment for this lipid abnormality,
- BP ≥ 130/≥ 85 mmHg or treatment of previously diagnosed hypertension,
- fasting glucose serum level ≥ 100 mg/dL or previously diagnosed DMT2.

The control group consisted of 10 age-matched, healthy, non-obese men with WC < 94 cm (N = 10, BMI = 24.32 ± 1.48 kg/m^2, aged 59.7 ± 5.93 years).

Measurements

The weight and height were measured in the fasting state by a medical electronic scale (RADWAG). BMI was calculated using the following formula: BMI = body weight (kg)/height (m^2). WC was measured midway between the lower costal margin and the superior iliac crest. Hip circumference (HC) was measured at the widest part of the hip at the level of the greater trochanter. Blood pressure measurements were made using a standard sphygmomanometer cuff (12 x 23 cm) on the left forearm after the patient spending 10 minutes in the sitting position.

Laboratory tests

Laboratory tests were conducted at the Department of Pathophysiology, Medical University of Silesia in Katowice. The serum parathyroid hormone (PTH) concentration and C-terminal telopeptide of type I collagen (CTX_1) was determined by an electrochemiluminescence immunoassay (Elecsys, Roche Diagnostics GmbH, Germany). The serum concentration of 25-OH-D_3 was determined by the radioimmunoassay technique (RIA) (Bio Source--EUROPE SA, Nivelles, Belgium). The serum concentrations of total calcium, inorganic phosphate, glucose, and lipids were determined by spectrophotometry (Point Scientific Inc., Michigan, USA). The serum concentration of FGF-23 was ascertained by ELISA (Immuntopics, San Clemente, USA). The serum concentration of OPG was ascertained by ELISA (Biovendor, Modřice, Czech Republic).

FRAX assessment

The 10-year probability of a major osteoporotic fracture was estimated by the WHO recommendations based on the FRAX [13]. The University of Sheffield launched the FRAX tool in 2008 [12].

Statistical analysis

The values are presented as means and standard deviations. The comparison between the control and all obese group was made by the Mann-Whitney U test. On the other hand, the comparison between the control as well as the metabolic-obese and simple obese subgroups was made by the ANOVA Kruskal-Wallis test. Correlation coefficients R were obtained according to Spearman's rank correlation coefficient. $P < 0.05$ was considered statistically significant. All the statistical analyses were performed using STATISTICA 10 PL software.

RESULTS

The characteristics of the studied control and the all obese subgroups are presented in Table I.

The patients from the study group and control group were all the same age. The study group had higher anthropometric parameters (body weight, BMI, WC, HC), fasting blood glucose ($p < 0.001$), and creatinine concentration ($p < 0.05$) compared to the control group. The differences between the groups in the total cholesterol (TCh), low density lipoprotein cholesterol (LDL-C), HDL-C, TG were not statistically significant. Among the markers of bone metabolism, a lower serum OPG concentration ($p < 0.01$) and higher phosphate concentration ($p < 0.01$) were found in the all obese group compared to the control group. Moreover, there was a significant difference in the OPG concentration between the obese patients with MS, obese men with central obesity, and the controls. Nonetheless, there were no statistically significant differences between FGF-23, CTX_1, PTH, calcium, and 25-OH-D_3. FRAX was significantly ($p < 0.001$) lower in the all obese group (FRAX = 1.66 ± 0.32%) than in the healthy controls (FRAX = 2.08 ± 0.33%). Moreover, we noticed a strong positive correlation between FRAX and age ($R = 0.68$; $p < 0.001$) as well as a strong negative correlation between FRAX and BMI ($R = -0.67$; $p < 0.001$) in the group of all obese men. Among the healthy subjects, the correlation between FRAX and age was even stronger ($R = 0.87$; $p < 0.001$), but no relation was observed between FRAX and BMI.

Table I. Characteristics of studied groups
Tabela I. Charakterystyka grupy badanej

Parameters	Control group (N = 10)	All obese group (N = 36)	U	Obese men with central obesity (N = 18)	Obese men with MS (N = 18)	KW
Anthropometric parameters						
Age (y)	59.70 ± 5.93	61.06 ± 5.62	*ns*	60.39 ± 5.76	61.72 ± 5.56	*ns*
BMI (kg/m^2)	24.32 ± 1.48	33.80 ± 3.53	***	33.40 ± 3.76	34.19 ± 3.35	***
Body mass (kg)	75.00 ± 8.92	100.24 ± 13.45	***	100.19 ± 15.92	100.28 ± 10.90	***
WC (cm)	86.10 ± 4.82	114.44 ± 13.39	***	114.56 ± 13.13	114.33 ± 14.03	***
HC (cm)	88.50 ± 7.50	112.86 ± 14.25	***	115.44 ± 16.42	110.28 ± 11.59	***
Biochemical factors						
TCh (mg%)	164.70 ± 45.89	200.64 ± 44.38	*ns*	202.83 ± 49.66	198.44 ± 39.74	*ns*
HDL-C (mg%)	40.00 ± 22.38	43.22 ± 6.95	*ns*	41.44 ± 5.98	45.00 ± 7.54	*ns*
LDL-C (mg%)	106.60 ± 36.92	125.97 ± 34.51	*ns*	128.00 ± 37.54	123.94 ± 32.15	*ns*
TG (mg%)	124.10 ± 41.77	151.11 ± 65.68	*ns*	150.67 ± 52.28	151.56 ± 78.41	*ns*
Glucose (mg%)	84.20 ± 14.50	114.53 ± 41.23	***	95.11 ± 13.55	133.94 ± 50.18	***
Creatinine (mg%)	0.83 ± 0.23	1.03 ± 0.20	*	0.96 ± 0.11	1.11 ± 0.25	*
Bone metabolism markers						
OPG (pmol/l)	9.31 ± 4.55	5.25 ± 4.03	**	5.24 ± 4.19	5.25 ± 3.98	*
CTX_1 (ng/ml)	0.39 ± 0.17	0.42 ± 0.20	*ns*	0.42 ± 0.16	0.42 ± 0.24	*ns*
PTH (pg/ml)	32.51 ± 14.10	38.78 ± 19.41	*ns*	35.62 ± 19.90	41.93 ± 18.93	*ns*
25-OH-D_3 (ng/ml)	30.66 ± 10.22	27.00 ± 20.16	*ns*	28.61 ± 20.58	25.39 ± 20.19	*ns*
Calcium (mg%)	2.19 ± 0.30	2.23 ± 0.39	*ns*	2.28 ± 0.19	2.19 ± 0.52	*ns*
Phosphates (mg%)	1.02 ± 0.34	1.79 ± 0.78	**	1.71 ± 0.95	1.87 ± 0.57	**
FGF-23 (ng/ml)	(N = 8) 19.04 ± 15.61	(N = 26) 24.81 ± 23.71	*ns*	(N = 15) 25.12 ± 24.95	(N = 11) 24.38 ± 23.10	*ns*
FRAX (%)	2.08 ± 0.33	1.66 ± 0.32	***	1.66 ± 0.25	1.67 ± 0.38	***

The comparison between the control and the all obese group was made by the Mann-Whitney U test (U). On the other hand, the comparison between the control group and both obese subgroups was made by the ANOVA Kruskal-Wallis test (KW); * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$, ns – non significant, N – number of subjects, BMI – body mass index, WC – waist circumference, HC – hip circumference, TCh – total cholesterol, HDL-C – high density lipoprotein cholesterol, LDL-C – low density lipoprotein cholesterol, TG – triglycerides, OPG – osteoprotegerin, CTX_1 – C-terminal telopeptide, PTH – parathyroid hormone, FGF-23 – fibroblast growth factor 23, FRAX – Fracture Risk Assessment Tool.

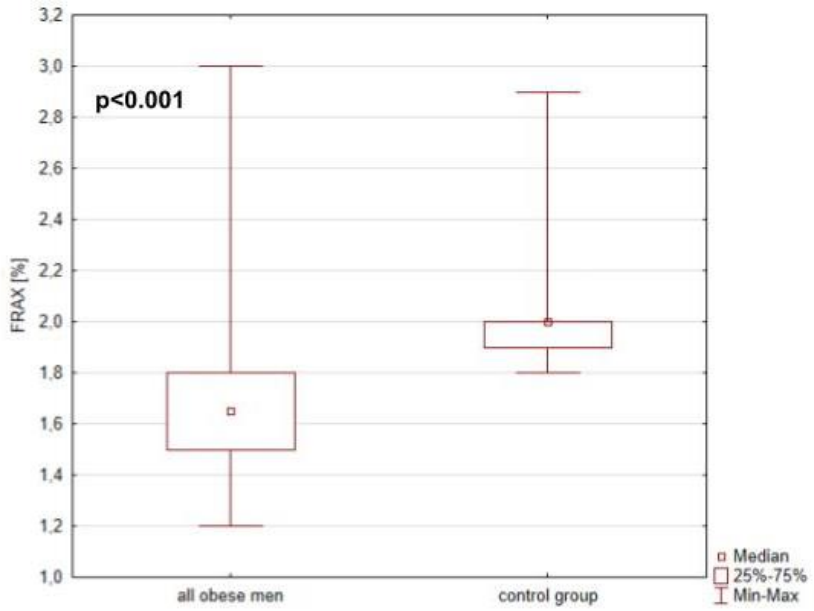

Fig. 1. FRAX in all obese men with central obesity and control group.
Ryc. 1. FRAX u otyłych mężczyzn z otyłością centralną i w grupie kontrolnej.

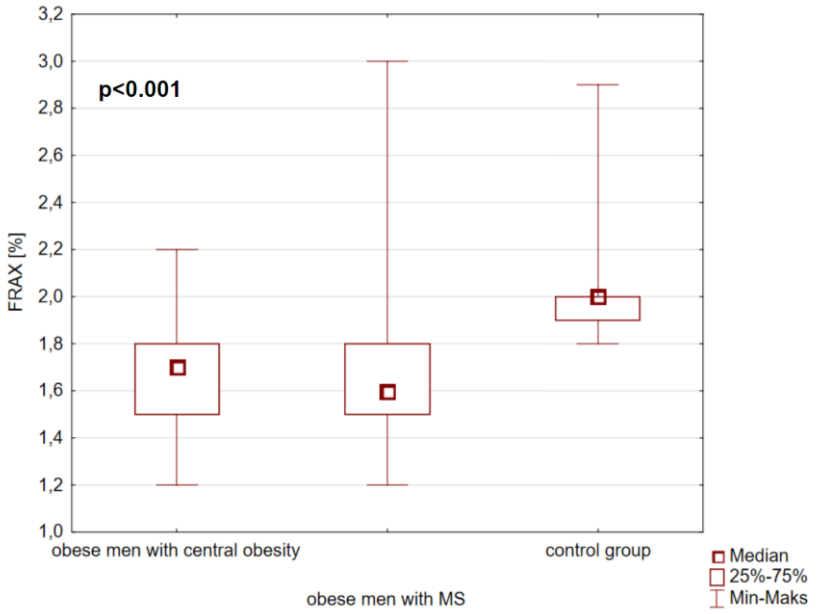

Fig. 2. FRAX in obese men with central obesity, obese men with MS, and control group.
Ryc. 2. FRAX u otyłych mężczyzn z otyłością centralną, otyłych mężczyzn z zespołem metabolicznym oraz w grupie kontrolnej.

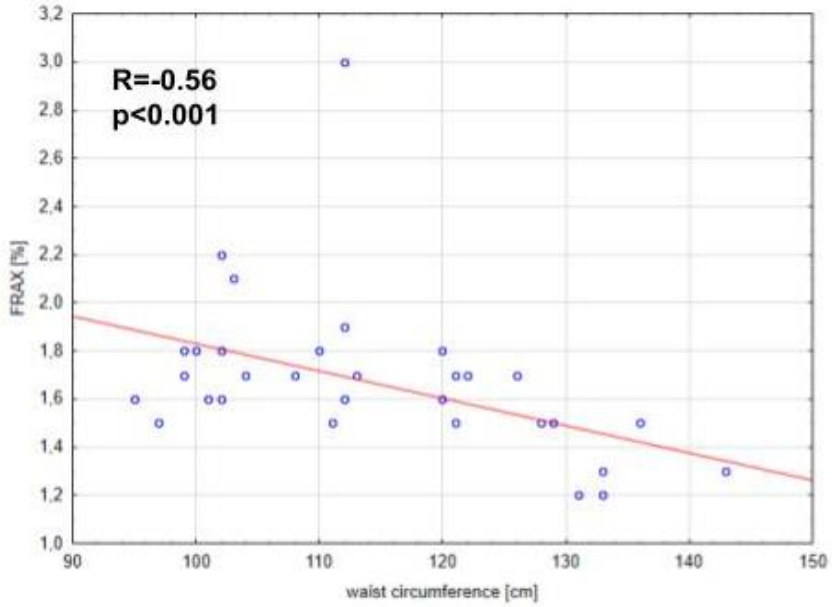

Fig. 3. Correlation between waist circumference and FRAX in all obese men.
Ryc. 3. Korelacja między obwodem talii i wskaźnikiem FRAX w grupie wszystkich otyłych.

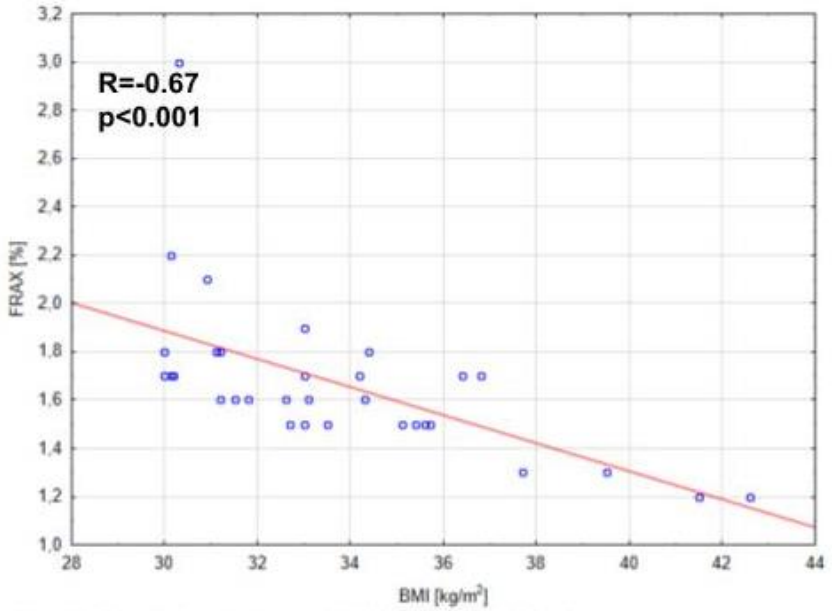

Fig. 4 Correlation between BMI and FRAX in all obese men.
Ryc. 4. Korelacja między BMI i wskaźnikiem FRAX w grupie wszystkich otyłych.

We observed a negative correlation between FRAX and WC in the all obese group ($R = -0.56$; $p < 0.001$), in the obese men with central obesity ($R = -0.69$; $p < 0.01$), and also in the obese subjects with MS ($R = -0.55$; $p < 0.05$). Furthermore, we observed a negative correlation between FRAX and HC in the all obese group ($R = -0.39$; $p < 0.05$) and in the obese subjects with MS ($R = -0.60$; $p < 0.01$). OPG was positively correlated with WC ($R = 0.50$; $p < 0.05$) in the obese subjects with MS. A positive correlation was also noticed between FRAX and FGF-23 ($R = 0.84$; $p < 0.01$) in the healthy subjects from the control group, but it was not observed in the obese patients.

In the healthy subjects, we observed a negative correlation between PTH and body mass ($R = -0.70$; $p < 0.05$) as well as between PTH and BMI ($R = -0.64$;

$p < 0.05$), and these correlations were not observed among the obese.

DISCUSSION

Assessing bone metabolism in men with MS is complicated because each simple element of MS may have a different and sometimes contrary impact on bone metabolism [20]. It was demonstrated that MS can be an additional risk factor for osteoporotic fractures [21]. Moreover, the incidence of osteoporotic non-vertebral fractures was higher in the participants with MS. Szulc et al. [20] reported the existence of an association between BMD and abdominal obesity in patients with MS; nevertheless, no correlation of BMD with the other components of MS was noticed. A lower risk of fracture was related to hypertriglyceridemia and it was the most predictive of reduced fracture risk among all the MS components. Additionally, the patients with MS, despite an observed higher number of falls, had a lower number of fractures.

In general, our study demonstrated that obese men have a lower 10-year fracture risk that is correlated with age. The increase in fracture risk in elderly subjects can be explained by bone loss associated with the hormonal consequences of aging such as lower serum estrogen and testosterone, both of which regulate bone renewal [22]. Progressively decreasing BMD with age results in a higher FRAX parameter [23]. An elevated BMI in obese patients increases bone load and consequently has a protective impact on BMD. Several ways have been proposed. As a response to mechanical load, a larger weight can stimulate bone formation and increase bone mass [24]. Obesity is associated with a decreased sex hormone-binding globuline (SHBG) concentration, lower total testosterone, and increasing levels of circulating estradiol [25,26]. Also, testosterone conversion by aromatase to E2 (estradiol) in adipose tissue increases the estrogen fraction, which may have a positive impact on bone metabolism [22,25]. Obesity-induced insulin resistance and hyperinsulinemia increase the IGF-1 concentration, which stimulates osteoblastic proliferation [27].

Fracture risk was assessed by the FRAX index, which is specific to osteoporotic fractures. It means we do not take into account traumatic fractures, where lower risk could interlink a sedentary lifestyle, a low level of physical activity and a lower tendency to perform extreme sports or the protective effect of fatty tissue in the obese. The research focused on bone metabolism changes, the relationship between them and increased body weight. It is worth mentioning that in both subgroups (metabolic and simple obesity) the FRAX parameter was less than 5%, which means a low 10-year probability of major osteoporosis-related fractures.

A higher WC is correlated with a lower FRAX parameter in all the obese men, but not in the healthy subjects. A negative relationship between osteoporosis and central obesity was proved by a study conducted by Chang et al. [28], but only in women. In contrast, a meta-analysis conducted by Sadeghi et al. [29] shows that central obesity is positively associated with a risk of hip fracture in both men and women. In the study by Meyer et al. [30], the researchers found that central obesity was associated with an increased risk of hip fracture in women, but not in men. On the other hand, in the study by Ornstrup et al. [31], it was shown that among obese men with MS, those with higher visceral fat accumulation frequently had osteopenia at the hip. The groups did not differ in terms of WC, but rather visceral fat accumulation, which was assessed quantitatively by MRI in cm^3. Due to the inconclusive results of different studies, our findings should be verified in larger epidemiological studies in the future.

The current study reported a lower OPG concentration in obese men than in the healthy controls. The obese subjects generally are more insulin-resistant, resulting in a lower OPG, which is inversely correlated with fasting glucose, insulin, and insulin sensitivity (HOMA-IR) [32]. Surprisingly, the concentration of OPG in the subgroup with MS was positively correlated with WC. Nonetheless, as we mentioned before, the reason for this may be the fact that visceral fat accumulation increases aromatase activity and leads to higher estrogen levels [25], which stimulates OPG production [33].

Previously, researchers proved that obese people have significantly higher serum FGF-23 levels compared to the non-obese population [34]. This association was particularly important in subjects with central obesity. In our study, we also observed this relation, but without statistical significance, probably because of the small size of our group. In many studies, the dependence between fracture risk and FGF-23 is not unequivocal. We found that in the healthy subjects, FGF-23 was positively correlated with FRAX. Similar results were presented in research involving elderly men, where a higher level of FGF-23 was associated with an increased fracture risk. That relation was even stronger when the FGF-23 level was above the cut-off point of 55.7 pg/ml [35]. Also, among elderly men with chronic kidney disease (CKD), it was observed that the FGF-23 level was independently associated with vertebral fracture probability [36]. Contrary to these outcomes, Isakova et al. [37] reported that a higher level of FGF-23 was not associated with a higher risk of fracture in older subjects with low CKD prevalence. Jovanovich et al. [38] reported that in elderly men, a higher level of FGF-23 was associated with a higher BMD, but was not associated with a higher risk of hip fracture in men and women, and the results were not different in patients with CKD. Taking into

consideration obese men, our study did not prove associations between FGF-23 and FRAX. The reason for this fact may be the occurrence of obesity and metabolic disorders in this population, which influences the FGF-23 serum level or in the obese population that association does not exist. There is a limited number of studies on the links between fracture risk in obese men with central obesity and the FGF-23 level. The conflicting results of different studies indicate the need to conduct more studies involving different populations of patients to prove the unequivocal influence of FGF-23 on the risk of fractures.

Our study has some limitations such as the small size of the groups, lack of information on comorbidities, history of past fractures, and BMD. Moreover, the research included only Caucasian men aged ≥ 54 years. Therefore, the obtained results cannot be applied to women, other ethnic groups, or the younger population.

CONCLUSIONS

1. Obesity is associated with decreased 10-year fracture risk, which is correlated with age.
2. Obese patients on average have lower levels of OPG than non-obese subjects.
3. Healthy subjects with a higher FGF-23 level have an increased 10-year fracture risk.

Author's contribution

Study design – M.N. Grabarczyk, M.T. Holecki
Data collection – M.T. Holecki
Data interpretation – M.N. Grabarczyk, M.J. Gorczyca
Statistical analysis – M.J. Gorczyca, P. Cieślik
Manuscript preparation – M.N. Grabarczyk, M.J. Gorczyca, K. Klimek, P. Kosińska, P. Cieślik, M.T. Holecki
Literature research – M.N. Grabarczyk, M.J. Gorczyca, K. Klimek, P. Kosińska

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