ORIGINAL ARTICLE

CONTENTS

Interrelationship between lipid and bone metabolism in post-menopausal women

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ABSTRACT

Aim: To study the state and interrelationship of calcium-phosphorus metabolism, lipid profile, and bone mineral density in postmenopausal women.

Materials and Methods: A retrospective cohort study was conducted involving 42 women aged 51.9±2.95 years, with a postmenopausal period of 1.69±1.47 years. All patients underwent laboratory and instrumental examinations.

Results: The study found that most women were overweight or obese and had elevated levels of total cholesterol, low-density lipoprotein cholesterol, and atherogenicity index. The average level of 25(OH)D was 26.95±10.58 ng/ml, with more than half of the subjects diagnosed with deficiency or insufficiency. According to ultrasound densitometry, a significant proportion of women had varying degrees of BMD reduction, ranging from osteopenia to osteoporosis. Correlation analysis revealed statistically significant associations between lipid profile indicators, calcium-phosphorus metabolism, and bone tissue condition, in particular inverse correlations between HDL-C levels and phosphorus, parathyroid hormone, and alkaline phosphatase, as well as between the atherogenicity coefficient and vitamin D levels ($p<0.05$).

Conclusions: The data obtained confirm the feasibility of a comprehensive assessment of calcium-phosphorus metabolism, lipid profile, and ultrasound densitometry results. This approach allows for the timely identification of women at increased risk of developing osteodismetabolic disorders and monitoring of bone tissue condition in the postmenopausal period.

KEY WORDS: postmenopause, vitamin D, osteopenia, dyslipidemias, bone density, ultrasonography

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INTRODUCTION

According to the International Menopause Society (IMS), menopause is defined as the point in time 12 months after the last menstrual period, reflecting the loss of ovarian follicular function, which leads to a decrease in estrogen concentration and an increase in follicle-stimulating hormone (FSH) [1]. As a physiological process, menopause is an independent factor that increases the risk of low-trauma fractures due to decreased secretion of estrogen, a hormone that improves bone formation by increasing calcium absorption in the intestines and decreasing reabsorption in the kidneys [2]. Bone mineral density (BMD) is an important parameter for assessing the state of osteometabolism in postmenopausal women, indicating the prevalence of remodeling processes due to osteoclasts or osteoblasts through the activation of systemic inflammation involving the Receptor activator of nuclear factor κB ligand/Receptor activator of nuclear factor κB/Osteoprotegerin (RANKL/RANK/OPG) cascade [3,4]. According to population cohort studies and meta-analyses, bone mass loss increases sharply about a year before the onset of postmenopause and continues for the next five years, reaching about 5% per year [5, 6]. This process leads to the formation of osteopenic syndrome, with an emphasis on women aged 50 and older. The use of methods for screening for BMD disorders, in particular, using ultrasound densitometry, is advisable and allows measures to be taken to prevent the progression of osteoporosis in its early stages [7].

One of the key regulators of calcium-phosphorus homeostasis is vitamin D. Vitamin D comes in several forms, among which cholecalciferol (D_3) and ergocalciferol (D_2) are the main nutritional sources, while 25-hydroxyvitamin D (25(OH)D) and 1,25-dihydroxyvitamin D ($1,25(OH)_2D$) are key metabolites that regulate calcium-phosphorus homeostasis and maintain BMD [8]. As a fat-soluble prohormone, it affects the expression of more than 200 human genes and is used by the body for physiological development and maintenance of

the musculoskeletal system by stimulating calcium absorption from food, osteoid mineralization, bone tissue metabolism, and muscle function [9,10]. According to the recommendations of the Endocrine Society of the United States, a serum 25(OH)D level below 20 ng/mL is interpreted as a deficiency, a value between 20-30 ng/mL as insufficiency, and above 30 ng/mL as sufficient [11].

Postmenopausal women have low levels of vitamin D, which contributes to increased parathyroid hormone (PTH) secretion, reduces calcium absorption in the intestine and serum calcium levels, while increasing bone resorption, which increases the risk of low-trauma fractures [12,13].

In addition to endogenous influences on vitamin D synthesis, exogenous influences can also occur due to insufficient sun exposure and food consumption [14,15]. Thus, dietary strategies can help improve vitamin D status by consuming foods enriched with vitamin D in combination with calcium [16].

In addition to impaired calcium-phosphorus metabolism, the postmenopausal period is associated with the formation of an atherogenic lipid profile, one of the mechanisms of which is endothelial dysfunction [17,18], which increases the risk of developing cardiovascular diseases compared to women of reproductive age [19, 20].

That is why the study of calcium-phosphorus homeostasis, BMD, and lipid profile in the postmenopausal period will allow for a prospective assessment of the risks of developing osteometabolic disorders, such as osteopenia and osteoporosis, as well as cardiovascular risks in women of this age group.

AIM

The aim of this study was to investigate the state and interrelationship of calcium-phosphorus metabolism, lipid profile, and bone mineral density in postmenopausal women.

MATERIALS AND METHODS

This study is part of the initiative research work of the Department of Propaedeutics of Internal Medicine «Clinical and pathogenetic features of cardiovascular diseases in conditions of comorbidity, taking into account gender and age aspects and ways to correct their disorders,» state registration No. 0124U003397.

We conducted a retrospective cohort study. Forty-two postmenopausal women were included in the study.

The inclusion criteria were female gender and menopause lasting 1-5 years.

Exclusion criteria were: artificial early menopause, primary hyperparathyroidism or hypercalcemia, impaired kidney or liver function, malignant neoplasms, diabetes mellitus, thyroid dysfunction, connective tissue disease.

Patients were examined at the clinical base of the Department of Propaedeutics of Internal Medicine of Poltava State Medical University at the Municipal Enterprise «3rd City Clinical Hospital of Poltava City Council» in Poltava.

Before the study, all patients signed an informed voluntary consent form to participate in the study. The study was conducted in accordance with the requirements of the Helsinki Declaration and Order of the Ministry of Healthcare of Ukraine No. 690 of 23.09.2009 «On Approval of the Procedure for Conducting Clinical Trials of Medicinal Products and Examination of Clinical Trial Materials.»

The examination of patients included standard anthropometric, laboratory, and instrumental methods of investigation. Anthropometric indicators were interpreted according to the body mass index (BMI). The data were interpreted according to the criteria of the World Health Organization (WHO), with BMI > 25 kg/m^2 and <29.9 kg/m^2 considered indicators of excess body weight, and BMI was defined as BMI >30 kg/m^2 , with obesity classified as grade I: 30.0–34.9 kg/m^2, grade II: 35.0–39.9 kg/m^2, and grade III (morbid): ≥ 40.0 kg/m^2 [21, 22, 23].

Laboratory tests included assessment of calcium-phosphorus metabolism (ionized calcium, magnesium, phosphorus, alkaline phosphatase, 25OHD, parathyroid hormone (PTH) and lipid profile (total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C (HDL), low-density lipoprotein cholesterol (LDL), very low-density lipoprotein cholesterol (VLDL), atherogenicity coefficient, triglycerides (TG)) in the certified medical laboratory «Analytica».

The condition of BMD was assessed using a Sunlight MiniOmni ultrasonic densitometer (BeamMed, Israel), with measurements taken on the radius, which has a spongy structure. The obtained BMD values were evaluated according to the following criteria: normal condition (BMD change from the reference value as a result of measurement in young people within one standard deviation SD), osteopenia (BMD decrease by > 1.0 SD and < 2.5 SD from the reference value), grade I osteopenia (decrease in BMD > 1.0 SD and < 1.5 SD from the reference value), grade II osteopenia (decrease in BMD > 1.5 SD and < 2.0 SD from the reference value), Grade III osteopenia (decrease in BMD > 2.0 SD and < 2.5 SD from the reference value), osteoporosis (decrease in BMD > 2.5 SD from the reference value) [24].

Statistical calculations were performed using KyPlot 6.0 software and Microsoft Excel. The Shapiro-Wilk test

Table 1. Lipid profile indicators of the study cohort of women

Indicators, units of measurement	Reference range	Statistical indicator	Study cohort (n = 42)
TC, mmol/L	< 5.77	M±SD	6.21±1.13
LDL-C, mmol/L	< 3.24		4.15±1.09
HDL-C, mmol/L	< 0.78		0.70±0.57
HDL-C, mmol/L	1.0		1.62±0.41
TG, mmol/L	< 2.3		1.32±0.84
Atherogenicity coefficient, c.u.	< 2.39		3.0±1.18

Note: M – mean value, SD – standard deviation
Source: compiled by the authors of this study

Table 2. Calcium-phosphorus metabolism indicators in the studied cohort of women

Indicators, units of measurement	Reference range	Statistical indicator	Study cohort (n = 42)
Total calcium, mmol/l	2.25	M±SD	2.21±0.45
Ionized calcium, mmol/l	1.13		1.15±0.25
Parathyroid hormone, pg/ml	11		42.91±20.39
Magnesium, mmol/l	0.7		0.87±0.09
Phosphorus, mmol/l	0.8 – 1.45		1.18±0.15
Alkaline phosphatase, <U/l	<105	Me (Q1; Q3)	81 (73.63; 102.18)

Note: M – mean value, SD – standard deviation, Me – median, Q – quartile
Source: compiled by the authors of this study

Table 3. Correlations between bone metabolism biomarkers

	Mg, mmol/L	Ca^2+, mmol/L	P, mmol/L	ALP, U/l	25(OH)D, ng/ml	PTH, pg/ml
Magnesium, mmol/L	–	r=0.210; p=0.182	r=0.277; p=0.076	r=-0.121; p=0.446;	r=0.194; p=0.218	r=0.060; p=0.708
Ionized calcium, mmol/L	r=0.210; p=0.182	–	r=0.283; p=0.069	r=0.045; p=0.779	r=0.092; p=0.564	r=0.226; p=0.150
Phosphorus, mol/L	r=0.277; p=0.076	r=0.283; p=0.069	–	r=0.127; p=0.424	r=-0.041; p=0.795	r=0.079; p=0.617
Alkaline phosphatase, U/l	r=-0.121; p=0.446	r=0.045; p=0.779	r=0.127; p=0.424	–	r=-0.269; p=0.085	r=0.215; p=0.172
25(OH)D, нг/мл	r=0.194; p=0.218	r=0.092; p=0.564	r=-0.041; p=0.795	r=-0.269; p=0.085	–	r=-0.275; p=0.078
PTH, pg/ml	r=0.060; p=0.708	r=0.226; p=0.150	r=0.079; p=0.617	r=0.215; p=0.172	r=-0.275; p=0.078	–

Note: r – correlation coefficient, p – significance level
Source: compiled by the authors of this study

was used to assess the normality of distribution. To assess the relationships between indicators, we used the correlation analysis method with the calculation of Pearson's correlation coefficients for normal distribution and Spearman's for other types of distribution. The data were presented as M±SD, where M is the mean value and SD is the standard deviation. For distributions that differed from normal, the results were presented as the median (Me) and interquartile range (IQR), (Q1; Q3), where Q1 and Q3 are the first and third quartiles, respectively. The criterion for statistical significance was $p<0.05$.

RESULTS

The mean age of the patients in the study cohort was 51.9±2.95 years, and the duration of menopause was 1.69±1.47 years. Fifty percent (21 individuals) of women were overweight, 23.8% (10 individuals) had grade I obesity, 4.8% (2 individuals) had grade II obesity, and the rest of the patients had a normal BMI.

The study group of women showed higher levels of total cholesterol, LDL cholesterol, and atherogenicity coefficient than the reference values (Table 1).

The mean level of 25(OH)D in the entire study group was 26.95±10.58 ng/ml. In 30.9% (13 people) of women,

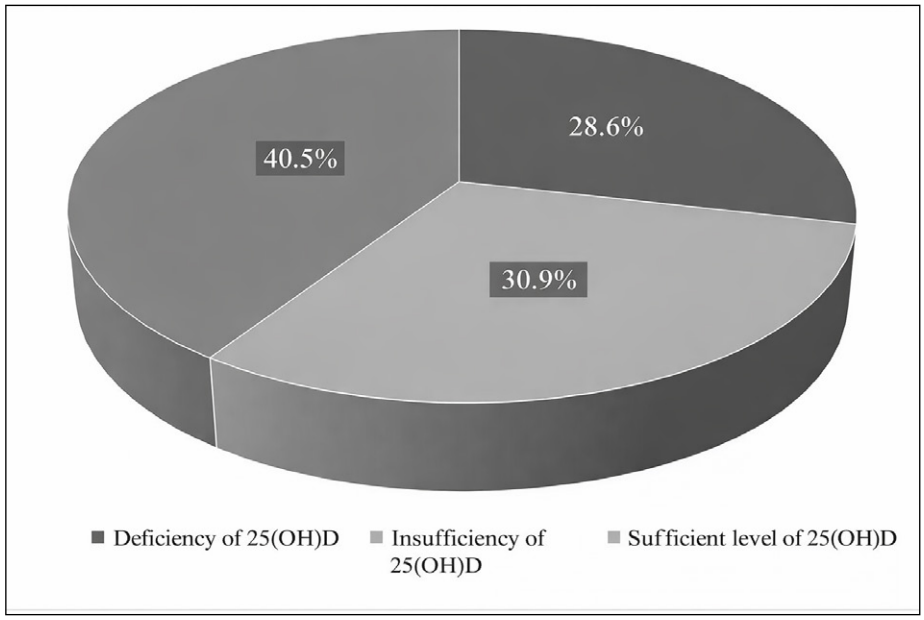

Fig. 1. Distribution of patients by serum 25(OH)D level
Source: compiled by the authors of this study

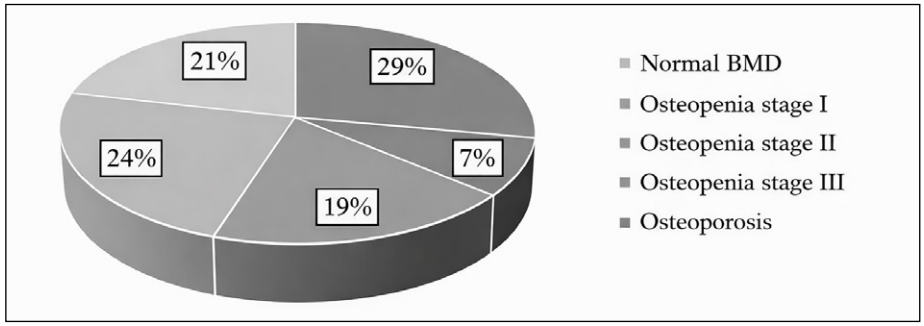

Fig. 2. Distribution of patients according to bone mineral density indicators
Source: compiled by the authors of this study

an insufficient level of 25(OH)D in the blood was determined, in 28.6% (12 people) – a deficiency, and in 40.5% (17 people) – a sufficient level (Fig. 1).

The levels of total and ionized calcium, magnesium, phosphorus, and parathyroid hormone in blood serum were within the reference range in all subjects studied (Table 2). An increase in alkaline phosphatase levels was recorded in 21.4% of women (9 individuals).

Ultrasound densitometry results verified a decrease in BMD, which meets the criteria for grade I–III osteopenia and osteoporosis. In 7.1% (3 individuals) of the women examined, grade I osteopenia was diagnosed, 19.1% (8 people) had grade II osteopenia, 23.8% (10 people) had grade III osteopenia, 21.4% (9 people) had osteoporosis, and the rest of the patients had preserved BMD (Fig. 2).

Correlation analysis between mineral metabolism indicators did not reveal any statistically significant patterns ($p>0.05$) (Table 3).

When conducting a correlation analysis between bone remodeling biomarkers and bone mineral density, a significant negative correlation was found between alkaline phosphatase levels and T- and Z-scores ($p<0.05$), as well as between Z-scores and BMI ($p<0.05$) (Table 4).

When studying lipid profile indicators, calcium-phosphorus metabolism, and BMD, a statistically significant ($p<0.05$) inverse correlation was found between HDL-C and phosphorus levels ($r=-0.3045$, $p=0.0499$), between HDL-C and PTH ($r=-0.3639$, $p=0.0178$), and between HDL-C and alkaline phosphatase levels ($r=-0.3100$, $p=0.0457$). In addition, a significant direct correlation was found between TG and BMI levels, as well as between the atherogenicity coefficient and phosphorus levels ($p<0.05$). At the same time, the atherogenicity coefficient had a significant inverse correlation with vitamin D levels ($p<0.05$) (Table 5).

DISCUSSION

Our results demonstrate the presence of dyslipidemia in women in the study group due to increased levels of cholesterol, LDL cholesterol, and atherogenicity coefficient. This confirms that one of the leading mechanisms in the development of lipid metabolism disorders is estrogen deficiency, which accompanies postmenopause [25,26]. Estrogens exert an antiatherogenic effect by indirectly increasing the expression of LDL receptors in the liver, enhancing LDL clearance from the blood, and thus ensuring their stable levels [27]. In addition, with their antioxidant properties, estrogens promote the synthesis of nitric oxide, which has an anti-inflammatory effect, reducing endothelial cell apoptosis and local inflammation in the vessel wall [28].

Table 4. Correlations between bone metabolism biomarkers and BMD, BMI, and age

	Mg, mmol/L	Ca^{2+}, mmol/L	P, mmol/L	ALP, U/l	25(OH)D, ng/mL	PTH, pg/ml	T-score radius, SD	Z-score radius, SD
T-score radius, SD	-0.134 p=0.397	0.020 p=0.901	0.112 p=0.478	-0.305* p=0.049	0.141 p=0.374	-0.009 p=0.954	-	-
Z-score radius, SD	0.001 p=0.995	-0.040 p=0.806	0.264 p=0.100	-0.354* p=0.025	0.213 p=0.188	-0.062 p=0.702	-	-
SOS, m/s	-0.177 p=0.263	-0.001 p=0.994	-0.017 p=0.916	-0.262 p=0.093	0.234 p=0.136	-0.002 p=0.990	-	-
BMI, kg/m2	-0.045 p=0.779	0.067 p=0.673	-0.028 p=0.859	0.107 p=0.500	-0.005 p=0.976	-0.039 p=0.807	-0.252 p=0.107	-0.318* p=0.045
Age, years	0.074 p=0.643	-0.232 p=0.139	0.233 p=0.137	0.153 p=0.332)	-0.001 p=0.993	-0.100 p=0.529	-0.079 p=0.617	0.052 p=0.752

Note: r – correlation coefficient; * – statistically significant correlation ($p<0.05$)
Source: compiled by the authors of this study

Table 5. Correlations between lipid profile indicators, BMD, BMI, and age

	Mg, mmol/L	Ca^{2+}, mmol/L	P, mmol/L	ALP, U/l	25(OH)D, ng/mL	PTH, pg/ml	T-score radius, SD	Z-score radius, SD	BMI, kg/m2	Age, years
TC, mmol/L	0,05997 p=0,7060	-0,1328 p=0,4019	-0,02454 0,8774	-0,1143 0,4711	-0,2729 0,0803	-0,2632 0,0922	-0,1427 0,3674	-0,1782 0,2587	0,1143 0,4710	-0,09713 0,5406
LDL-C,mmol/L	-0,08487 0,5931	-0,2423 0,1221	-0,1704 0,2807	0,08440 0,5951	-0,2336 0,1365	-0,1635 0,3009	-0,2996 0,0539	-0,3030 0,0511	0,1451 0,3592	-0,03001 0,8504
VLDL-C, mmol/L	0,06655 0,6754	-0,04146 0,7943	0,1535 0,3318	-0,01223 0,9387	-0,2735 0,0797	0,04385 0,7828	-0,02144 0,8928	-0,07549 0,6347	0,2466 0,1153	0,05601 0,7246
HDL-C, mmol/L	0,03452 0,8282	0,02912 0,8547	-0,3045 0,0499*	-0,3100 0,0457*	0,09742 0,5394	-0,3639 0,0178*	-0,08176 0,6067	-0,1412 0,3724	-0,2407 0,1247	-0,2302 0,1425
TG, mmol/L	0,1216 0,4432	0,02920 0,8543	0,1558 0,3244	0,09163 0,5639	-0,2815 0,0710	0,1202 0,4483	-0,08838 0,5778	-0,09835 0,5355	0,3092 0,0463*	0,1364 0,3891
Atherogenicity coefficient, c.u	0,02471 0,8880	-0,08032 0,6465	0,3471 0,0411*	0,2435 0,1587	-0,3992 0,0175*	-0,0006273 0,9971	-0,1142 0,5135	-0,1056 0,5461	0,2845 0,0976	0,1171 0,5030

Note: r – correlation coefficient; * – statistically significant correlation ($p<0.05$)
Source: compiled by the authors of this study

That is why women before menopause are more protected from atherosclerotic heart disease than men, with approximately half the risk of cardiovascular disease [29,30].

At the same time, in the study group of women, 59.5% of the subjects had reduced levels of 25(OH)D, which meets the criteria for deficiency and insufficiency (Fig. 1). It is known that about 64% of women worldwide in the postmenopausal period have serum 25(OH)D levels below 30 ng/ml [31]. Vitamin D deficiency, despite its high prevalence worldwide, is still not adequately corrected. According to researchers, an increase in age by 10 years leads to a 13% decrease in vitamin D synthesis, which is due to a decrease in the formation of 7-dehydroxycholesterol in the epidermis [32]. A decrease in estrogen levels leads to a disruption in the regulation of enzymes involved in vitamin D metabolism, in particular 1α-hydroxylase, reducing the formation of the active form of 1,25-dihydroxyvitamin D ($1,25(OH)_2D$), which is involved in the regulation of calcium-phosphorus metabolism and bone mineralization. Also, in postmenopause, the proportion of adipose tissue in which vitamin D is deposited increases, which reduces its bioavailability in the blood [33, 34].

The vast majority of patients in the study group showed a decrease in BMD, which meets the criteria for grade I-III osteopenia and osteoporosis, consistent with scientific understanding of the effect of hypoestrogenism on bone tissue (Fig. 2). The ratio between osteoblasts, which are involved in the synthesis of new bone mass, and osteoclasts, which ensure bone resorption processes, plays an important role in the remodeling of bone tissue. Estrogens suppress osteoclast activity, promoting osteoblast differentiation by increasing OPG formation and decreasing RANKL, which provides osteoprotection [35, 36]. In the postmenopausal period, estrogen deficiency leads to an increase in the

RANKL/OPG ratio due to increased RANKL expression and decreased OPG synthesis, which causes excessive osteoclast activation and enhanced bone resorption through stimulation of signaling pathways involving NF-κB. It should be noted that estrogen deficiency is also accompanied by an increase in pro-inflammatory cytokines, in particular interleukin (IL) 1, IL-6, and tumor necrosis factor α (TNF-α), which stimulate RANKL expression and further suppress OPG, thereby enhancing osteoclastogenesis [37, 38].

An increase in alkaline phosphatase levels in 21.4% of the subjects may indicate a compensatory response of osteoblasts to increased bone resorption, i.e., it is an indicator of enhanced bone remodeling, where resorption predominates, but some bone formation activity is still present [39]. Our study found that higher alkaline phosphatase levels are associated with a decrease in BMD, as confirmed by a negative correlation with T- and Z-scores (Table 4). Thus, alkaline phosphatase levels can be considered a biochemical marker of bone remodeling in this cohort of women [40].

In 78.6% of women in the study group, an increased BMI was found, which has a negative correlation with the Z-score, consistent with current data on the negative effect of visceral obesity on bone metabolism through mechanisms of chronic low-intensity inflammation [41, 42].

The results of the correlation analysis indicate the existence of relationships between the lipid profile and calcium-phosphorus metabolism. In particular, the negative correlation between HDL-C levels and phosphorus, PTH, and alkaline phosphatase does not indicate a causal relationship but reflects the interrelationship of metabolic processes caused by the postmenopausal period. An increase in HDL levels may occur compensatorily in conditions of metabolic disorders and changes in the inflammatory background due to a decrease in the effect of parathyroid hormone on bone tissue and osteoclast activity, which is simultaneously manifested in a decrease in alkaline phosphatase levels. Thus, high HDL may be an indirect marker of bone mass preservation, at least through a decrease in parathyroid hormone-mediated resorption [43].

The direct correlation between TG and BMI levels confirms that an increase in body weight is associated with increased triglyceride synthesis in the liver, leading to dyslipidemia. That is why an increase in BMI is a risk factor for the development of metabolic syndrome, including dyslipidemia [44].

The established inverse relationship between the atherogenicity coefficient and vitamin D levels in the women studied demonstrates risk factors for both calcium-phosphorus metabolism imbalance and lipid profile. This result confirms that low vitamin D levels are associated with an atherogenic lipid profile, accompanied by increased systemic inflammatory activity [45].

CONCLUSIONS

The vast majority of women in early postmenopause have dyslipidemia with elevated total cholesterol and low-density lipoprotein cholesterol levels, low blood vitamin D levels, and varying degrees of bone mineral density disorders, ranging from osteopenia to osteoporosis.

It has been found that high-density lipoprotein cholesterol levels may indirectly reflect the condition of bone tissue.

The results of the study demonstrate the importance of a comprehensive approach to assessing bone metabolism, taking into account not only traditional markers of bone remodeling, but also the lipid profile, which is especially relevant for postmenopausal women who are at increased risk of developing osteopenia and osteoporosis.

Determining the state of calcium-phosphorus metabolism and lipid profile in combination with ultrasound densitometry allows identifying groups at increased risk of developing osteodystrophic syndrome, as well as monitoring bone tissue condition in registered cases of osteopenia or osteoporosis.

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