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BIOCHEMICAL AND GENETIC CHARACTERISTICS OF SECONDARY OSTEOPOROSIS IN PATIENTS WITH COMBINED COURSE OF CHRONIC PANCREATITIS AND HYPERTENSIVE DISEASE

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Abstract

The purpose of the study is to assess the relationship between polymorphism of farnesyl diphosphate synthetase gene (FDPS c.IVS1 T-99G) and the content of biochemical markers of bone metabolism (total and ionized serum calcium, osteocalcin) in the risk of osteoporosis development in patients with chronic pancreatitis (CP) and hypertensive disease (HD). The study involved 110 patients with CP, in 70 of whom it developed secondary to HD and isolated CP in 40. The study showed that CP creates conditions for the formation of a negative calcium balance and its manifestations increase in combination with HD. The combined course of CP and HD in most cases is accompanied by changes in the content of osteocalcin, the level of which correlates with a violation of the mineral density of bone tissue. An increase in the number of individuals with C-allele of FDPS gene in both groups was noted, however, this polymorphism of the gene was not confirmed by the clinical course.

Keywords: chronic pancreatitis, hypertensive disease, FDPS receptor gene, markers of bone metabolism, osteoporosis.

Introduction. The development and functioning of the body as a whole and its individual systems occurs with the participation of macro- and microelements, and their quantitative composition is determined by the deficiency and their function in the body. That is, regulation of a specific biological system of nutritive homeostasis, which includes an extensive complex of macro- and microelements, is determined by the intake, metabolism, specific accumulation, excretion and influence of mineral substances in general on all living things [10,17].

The macronutrient composition is represented by 11 chemicals, among which calcium occupies a special place (1-1.5 kg of the total body weight). It participates in the functioning of the cardiovascular and nervous systems, in the processes of blood coagulation, in the production of hormones, enzymes and proteins, in the contraction and relaxation of muscles and, especially, in ensuring the strength of the bones [2,13]. Calcium increases the protective functions of the body, promotes the excretion of heavy metals, has an antioxidant and antihistamine effect. Together with phosphorus, it participates in the construction of bone tissue (98%), and with magnesium it ensures the normal functioning of the cardiovascular system, controls the heart rate, promotes iron metabolism, and transmission of nerve impulses [22].

The main regulators that maintain a constant level of calcium and phosphorus in the blood are calcitonin – a hormone of C-cells of the thyroid gland with hypocalcemic effect and parathyroid hormone (PTH) – a

hormone of the parathyroid glands that has a hypercalcemic effect [11,20]. About 150 diseases of the internal organs are associated with a deficiency of calcium in the body. Among them, the most common are osteoporosis (OP), diabetes mellitus, coronary heart disease and arterial hypertension. A special place among such disorders is also given to diseases of the digestive tract, since the intake of this macrocell is carried out only through the gastrointestinal tract.

In this case, the combined course of diseases of the digestive tract (for example, chronic pancreatitis (CP), as one of the socially significant and fairly common diseases) and the cardiovascular system (hypertensive disease, HD) can lead to calcium deficiency and, therefore, the development of complications.

The comorbidity of CP and HD is due to their significant prevalence and general risk factors, including chronic stress, smoking, increased body mass index, alcohol abuse and several others. Thus, it gives rise to conditions that act both through etiological and pathogenic structures (functional interaction of cytokines, lipid peroxidation system - antioxidant protection, lipid spectrum of the cell membrane, etc.). Disruption of calcium metabolism can be considered as one of the links in the combination of CP and HD, which allowed a number of researchers to attribute these disorders to calcium-dependent diseases [4,15,27].

The functioning of the pancreas occurs in the presence of calcium ions. Pancreatic juice contains up to 60 mg / l of calcium and its concentration depends mainly on the content in the extracellular fluid and the functional state of the pancreas [6]. An increase in the secretion of enzymes is always accompanied by an increase in the content of calcium ions in the juice. It is believed that the release of intracellular calcium promotes the secretion of enzymes, and extracellular calcium stimulates the maintenance of secretion [9]. Pancreatic lipase is essentially the only enzyme that breaks down dietary triglycerides. Lipase acts on the surface of fat droplets, so emulsification of fat with bile acids and their salts is of great importance for its digestion. This enzyme is specific in its activity: it hydrolyzes only triglycerides in an emulsified state. Unlike proteolytic enzymes, lipase does not damage acinocyte and other gland cells; its activity is increased by calcium, sodium, chlorine and bile salts [12].

In calcium deficiency, insulin secretion from pancreatic β-cells is inhibited, and insulin-dependent form of diabetes is exacerbated [1]. A change in the acidity of pancreatic juice is considered to be one of the reasons for the development of CP. So, consumption of cow's milk, which contains a large amount of calcium phosphate and an alkaline environment, contributes to the formation of stones in the ducts of the gland [16]. In addition, the development of CP leads to impaired absorption of a number of substances in the intestine, among which calcium is also given a certain value. Also, maldigestion syndrome is accompanied by a disruption of vitamin D absorption, which significantly slows down the process of calcium intake and the quantity of calcium becomes limited [18, 19]

One of the important effects of calcium-dependent receptors is the regulation of vascular tone and blood pressure (BP), which is carried out through the modulation of calcium homeostasis. Stimulation of these receptors was shown to cause the production of nitric oxide, which is a powerful vasodilator. Calcium-dependent receptors play an important role in extracellular calcium homeostasis, regulating the rate of secretion of parathyroid hormone (PTH) and the rate of calcium reabsorption by the kidneys. It was also found that calcium-dependent receptors of the vascular endothelium activate potassium channels, resulting in potassium-induced hyperpolarization of the SMC vessels. All this shows that calcium-dependent receptors can play a physiological role in modulating blood pressure [28].

Genetic defect at the level of angiotensin-converting enzyme gene is assumed to be the basis of calcium metabolism disorders in patients with hypertension [20]. Developing changes in the cell membrane are accompanied by the loss of potassium and accumulation of sodium and calcium inside the cell. Excess calcium leads to an increase in vascular tone and sensitivity of vessels to catecholamines. Hyper-reactivity of the vascular wall is accompanied by a change in the rheological properties of the blood, causing the development of hyperlipidemia. This mechanism provides a prolonged increase in blood pressure [7].

A number of studies have shown the importance of the expression of calcium-dependent receptors and their signaling mechanisms in understanding the physiology and pathophysiology of the cardiovascular system. Thus, increased expression in the heart of a protein containing calcium-dependent receptors was noted during myocardial ischemia and reperfusion [29]. In that manner, calcium-dependent receptors isolated from various types of vessels can participate in the regulation of blood pressure [3]. Such a dependence of pathogenic links on the calcium content in the body can lead to its competitive intake in chronic pancreatitis and hypertensive disease, thereby potentiating the exit from depot bone tissue. In other words, it can be assumed that the combined course of chronic pancreatitis and hypertensive disease will “provoke” the development of osteoporotic conditions. Osteoporosis is characterized by a decrease in bone mass and a violation of its quality, which in turn leads to brittle bones and an increased risk of fractures, both with minor injuries and often without them [5].

General and local disorders in the system of regulation of bone formation and resorption, which affect the severity of osteoporotic conditions, directly or indirectly depend on the structural usefulness of genes associated with mineral metabolism. One such gene is farnesyl diphosphate synthase receptor (FDPS) gene. Diphosphates are potential inhibitors of the activity of osteoclasts, cells that destroy bone tissue during its remodeling. Osteoclasts reduce the rate of bone metabolism, help reduce bone mass and its mineralization. Nitrogen-containing diphosphates inhibit enzyme farnesyl diphosphate synthetase, which plays a significant role in cholesterol synthesis. The described mechanism leads to a slowdown in sterol synthesis in osteoclasts and provokes their apoptosis [24].

Previous studies have shown that the activity of FDPS can affect the decrease in bone mass in women

through a change in the activity of osteoclasts. Genetic differences in FDPS gene, affecting its activity, can contribute to a decrease in bone density in women after 40 years of age [14]. These results were confirmed by Levy M.E. et al. (2007), who investigated the role of polymorphism (A> C) of FDPS gene in reducing peak bone tissue (PBT) in 283 women of the same age and body mass index in the postmenopausal period [23]. The minor C allele was found to be associated with a decrease in FDPS expression. Subsequently, it was found that FDPS enzyme plays a major role in the accumulation of bone tissue occurring in the puberty. At the same time, the presence of SS genotype leads to a higher activity of osteoclasts during bone mass growth and to a decrease in peak PBT values [14]. However, it cannot be ruled out that the main effect of FDPS on bone density is manifested precisely in the postmenopausal period [24].

Marini F et al. analyzed the relationship of A> C polymorphism of FDPS gene with osteoporosis in 234 menopausal women in response to treatment with amino-bisphosphonates for two years [25]. The results showed that gene polymorphism is not directly related to bone density. However, some markers of bone metabolism in the presence of SS genotype reach significantly lower values in response to therapy with amino-bisphosphonates compared with those with AS or AA genotypes [21, 26].

At the same time, the literature practically gives no data on the frequency of polymorphism of FDPS gene in patients with chronic pancreatitis and hypertensive disease, as well as its relationship with the development of osteoporotic conditions.

The purpose of the study: to determine the state of biochemical parameters of bone metabolism (serum calcium fractions, osteocalcin) and to establish the dependence of these indicators on polymorphism of farnesyl diphosphate synthetase receptor gene in patients with a combined course of chronic pancreatitis and hypertensive disease.

Materials and methods of the study. The study involved 110 patients with CP, 70 of whom were included in the main group with a combined course of CP and HD and 40 patients comprised a comparison group with isolated CP. The mean age in the groups was 33.2 ± 2.1 and 32.9 ± 3.1 years, respectively. The duration of follow-up on CP ranged from 2 to 15 years with an interquartile range (IR) of 4-7 years with a medial tendency of 5 years. Hypertensive disease was recorded in terms of 3 to 17 years with a similar IR range (4-8 years) and a medial tendency of 5 years. In 27 cases, HD preceded the formation of CP; in 19 patients CP was the first to develop. The remaining 24 patients were not able to determine the previous disease.

Control findings of biochemical and genetic studies were obtained in examination of 78 practically healthy individuals, representative of the groups by age and gender.

Written consent was obtained from each patient participating in the study, in accordance with the recommendations of the ethical committees for biomedical research, 2000 Helsinki Declaration, and European Community directives 86/609 on the participation of people in biomedical research.

HD was diagnosed using the recommendations of the European Society for Arterial Hypertension (ESH) (2009) and the recommendations of the working group on arterial hypertension of the European Association of Cardiologists for the Prevention and Treatment of Hypertension (2012), taking into account the classification of the degree and stage of hypertension, the risk of hypertension (risk stratification for assessment of hypertension prognosis). So, in patients of the main group, systolic blood pressure ranged from 159-170 mm Hg and the average for the group was 164 ± 6.3 mm Hg; diastolic blood pressure was 98.4 ± 3.1 mm Hg, which corresponded to stage II HD, 2nd degree.

The diagnosis of CP was verified by a comprehensive assessment of patient's history, past medical history, clinical, laboratory and instrumental findings. Excretory function of the pancreas was determined by a fecal test with elastase-1 (enzyme-linked immunosorbent assay, standard method by Freinstein K. and Janoff A.).

Patients with impaired endocrine gland function were not included in the study.

The direction of changes in bone metabolism was assessed by analyzing the state of calcium metabolism determining the specified ion in blood serum — the biochemical method (reagent kit PLIVA-Lachema, Czech Republic). Ionized calcium of blood serum was calculated according to the formula of D. I. Mitsura [8]. At the same time, the indicator of relative ionized calcium content (RICC) was calculated, which characterizes the ratio of total and ionized calcium in biological media: RICC = Ca (i) x100% / Ca, where Ca (i) is calcium ionized; Ca - total calcium.

The content of osteocalcin in blood serum was determined by enzyme immunoassay using commercial IDS test systems manufactured by ELISA (USA) using an enzyme-linked immunosorbent analyzer Labline-90 (Austria).

Polymorphism of farnesyl diphosphate synthetase gene polymorphism (FDPS c. IVS1 T-99G) was determined using polymerase chain reaction (PCR) using Litech commercial kits (Russia) on a Rotor - Gene 6000 amplifier (Australia) in real time.

The data obtained were processed in the statistical environment STATISTICA 6.0. Conjugation tables were analyzed using the Pearson criterion χ-square (PCS), for the continuous scale indicators distributed according to the normal law, we used the two-sided t-test for unrelated samples (TT) and the non-parametric Mann-Whitney test (MWT) for distributions other than normal. To characterize the results, qualitative indicators were presented in the form of histograms, and continuous indicators were presented in the form of box plots.

Results of the study and their and discussion. Ultrasound examination performed in all cases made it possible to establish echo-signs of CP. So, fibrotic changes in the parenchyma corresponded to an increase in the echogenicity of the organ, pronounced unevenness of the echostructure, serration of the contour, and a decrease in the size of the gland (34 individuals). In

the predominance of the components of inflammation there was an increase in the echogenicity of organ tissue, blurring of the contour of the gland, an increase in the size of the pancreas or its part and expansion of the Wirsung duct (41 patients). In 35 cases, a combination of the above characteristics was established. Some patients (34.5%) underwent computed tomography at the previous stages of observation, which also confirmed the presence of CP.

All pancreatic patients had impaired pancreatic excretory function, taking into account the level of fecal pancreatic elastase-1. So, in the main group 27 patients (38.6%) had a mild and 43 (61.4%) a medium degree of excretory insufficiency. In the comparison group, this distribution corresponded to 15 (37.5%) and 25 (62.5%) individuals.

Determination of the content of free and protein-bound calcium in the blood serum of both groups of patients showed violations corresponding to hypocalcemia (Table 1).

Table 1.

Calcium metabolism in patients with chronic pancreatitis and hypertensive disease (mean sample, standard deviation, TT)

Indicators	Main group (o) (n=70)	Comparison group (c) (n=40)	Control group (n=20)	$p_{o,c}$	$p_{o,к}$, $p_{c,к}$
Total serum calcium, mmol/l	2.33±0.01	2.51±0.01	2.62±0.03	<0.01	<0.01 <0.01
Ionized blood calcium, mmol/l	1.16±0.01	1.18±0.01	1.23±0.01	<0.01	<0.01 <0.01
Calcium coefficient (CC), %	49.8±0.2	47.0±0.3	46.9±0.2	<0.01	<0.01 0.184

Thus, the study showed that in chronic pancreatitis, quantitative changes were observed in the content of both forms of calcium (free and protein-bound), but their ratio between the cell and the extracellular space remained practically unchanged, as indicated by the CC index. These changes in CP can be explained by the formation of maldigestion syndrome and, as a result, malabsorption of calcium and vitamin D in the small intestine. The addition of hypertensive disease, along with aggravated quantitative disorders, is accompanied by a redistribution of calcium fractions between biological fluids, leading to an increase in the calcium coefficient. That is, comorbidity of CP and HD is an unfavorable combination in the development of calcium-dependent complications, namely osteoporotic conditions.

Changes in indicators of calcium metabolism were not dependent on age, gender and duration of the disease.

To study the relationship between the levels of biochemical markers and the presence of fractures in patients of both groups, the indicators of osteocalcin (OC) were transformed into ordinal scales by comparison with reference values. The limits of reference values were considered the measurement intervals in patients of the control (C) group (78 people). So, the interval from 14.7 to 26.0 ng / ml was considered the normal range for osteocalcin. Later on, the ratio of the marker indicator with reference values on the scale of “N” - normal, “BN” - below normal, “AN” - above normal was determined for the marker of each of the patients, and the percentage composition of this gradation in each group was identified.

Thus, in the group of patients with CP, the osteocalcin index was higher than the norm in 57.5% of patients (23); in 37.5% of cases (15) the OC indicator was within normal limits and in 5% (2) it was lower than in the control group (Fig. 1).

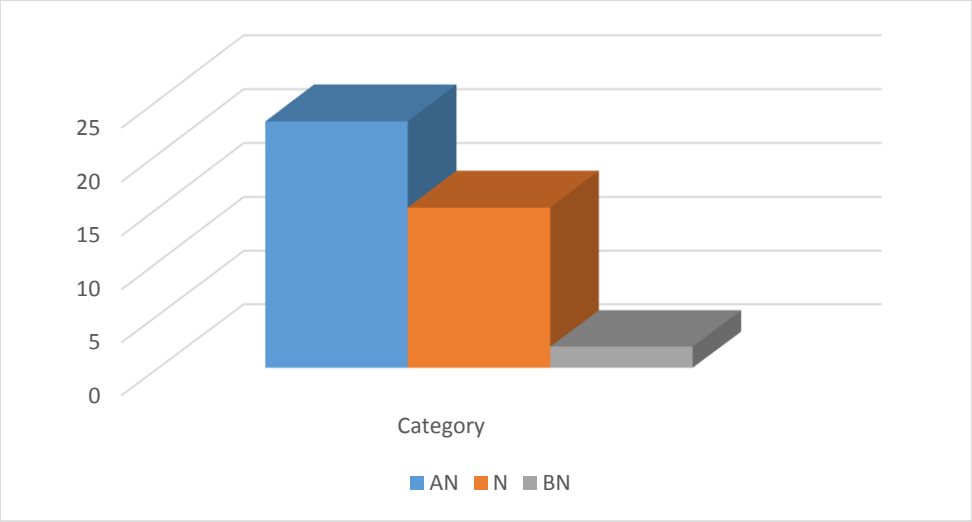

Fig. 1 Histogram of the distribution of osteocalcin in relation to the normal range in the CP group

Moreover, in the subgroup of patients with OC "AN", a history of fractures was observed in one patient (4%); in the subgroup "N" in one patient (7%) and in the subgroup "BN" also in one patient (50%). The distribution was statistically significant (Pearson Chi-square, $\chi 2 = 5.55$, df = 2, p = 0.052).

In the main group the content of osteocalcin in the blood serum corresponded to the following values: above the norm in 55.7% of patients (39), in 38.6% of cases (27) the indicator was within normal limits and in 5.7% (4) it was lower than the norm (Fig. 2).

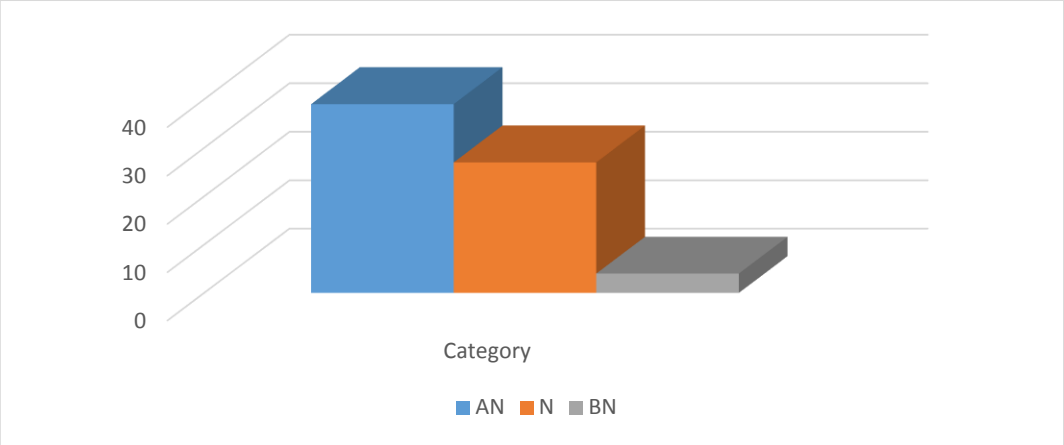

Fig. 2 Histogram of the distribution of osteocalcin in relation to the normal range in patients with CP and HD

In the subgroup "AN", 20 (51%) patients with a combined course of CP and HD indicated fractures of different localization. In the subgroup with normal values of osteocalcin, 14 patients (52%) had fractures in past history and one (25%) with a value of "BN".

Genetic testing of patients in the control group revealed the following distribution of genotypes of the polymorphic FDPS gene: carriers of the CC genotype were represented by 2 individuals (2.6%); there were 24 carriers of the AS genotype (30.8%), and the AA genotype was found in 52 individuals (66.6%).

In the comparison group (patients with CP), the SS genotype was found in 22.5% of cases (9 individuals); AS in 37.5% (15) and AA in 40.0% (16).

The combined course of CP and HD was associated with the following FDPS gene polymorphism: 30.0% (21 patients), 22.9% (16) and 47.1% (33), respectively. The categorized histogram of the distribution of genotypes of polymorphic FDPS gene had the following graphic image (Fig. 3).

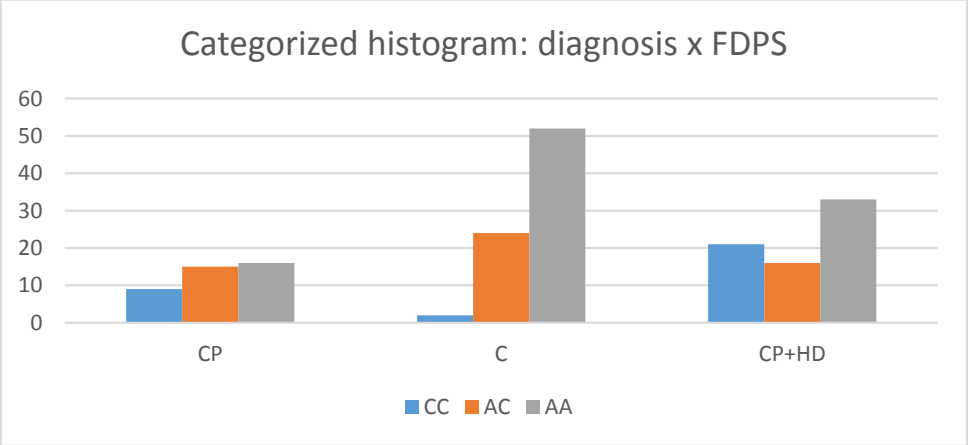

Fig. 3 Categorized histogram of the distribution of genotypes of polymorphic FDPS gene within study groups

This distribution was statistically significant (Pearson Chi-square, $\chi 2 = 23.58$, df = 2, p <0.00010).

Thus, when compared with the control in patients of both groups, there was an "intensification" of a statistically significant increase in the prevalence of CC genotype: 2.6 ± 1.8% in the control; 22.5 ± 6.6% and 30.0 ± 5.5% with CP and its combination with HD, respectively, secondary to a decrease in the prevalence of AA genotype (66.7 ± 5.3%, 40.0 ± 7.7%, 47.1 ± 6.0%, respectively).

In the analyzed sample, the dependence of the level of osteocalcin on the genotypes of polymorphic FDPS gene did not have a statistically significant character (MWT), which is probably due to insufficient sample size. However, as can be seen from the box plots of Figure 4, in the combined sample of patients with CP and CP + HD groups, there was a clear tendency to exceed the level of osteocalcin in patients with AA genotype (sample mean 36 ng / ml) in relation to patients with CC genotype (sample mean 28.5 ng / ml).

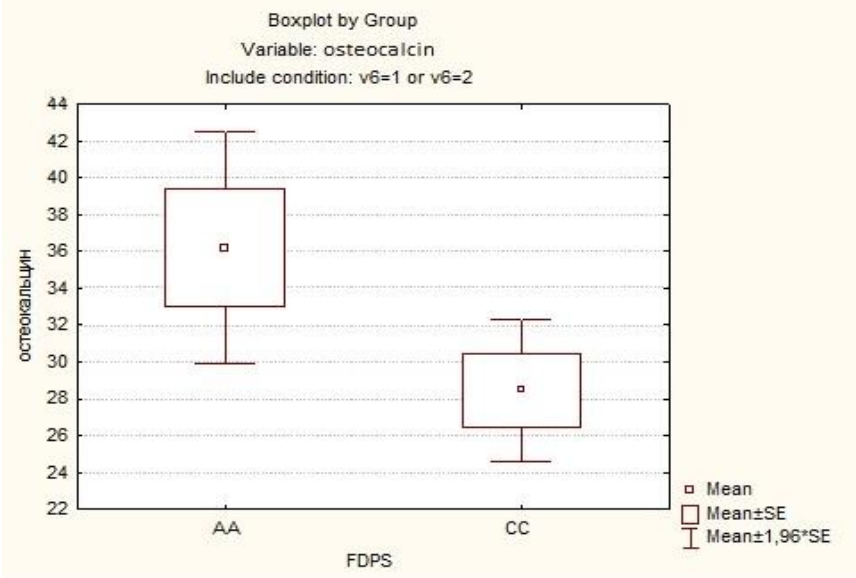

Fig. 4 Box plots of confidence intervals for the mean values of osteocalcin in patients of the combined sample - CP and CP + HD groups

Conclusions. Chronic recurrent nature of pancreatitis contributes to the conditions for the formation of a negative calcium balance and its manifestations increase with the onset of hypertensive disease.

The combined course of chronic pancreatitis and hypertensive disease in most cases is accompanied by changes in the content of osteocalcin, the level of which correlates with an impairment of the mineral density of bone tissue.

Patients with chronic pancreatitis and its combination with hypertensive disease were found to have an increase in prevalence of C-allele of FDPS gene; however, his polymorphism of the gene was not confirmed by the clinical course.

Changes in the indicators of calcium metabolism and osteocalcin content in patients with chronic pancreatitis and in combination with hypertensive disease can indicate an impairment of the mineral density of bone tissue with the development of osteoporotic conditions. Metabolic changes in bone tissue in patients with chronic pancreatitis and hypertensive disease can be considered as an unfavorable background for the chronic course and progression of both disorders, as well as the formation of complications.

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ON THE COMPLIANCE OF DENTAL PATIENTS IN SCHOOL AGE

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Abstract

The article considered the problem of compliance of dental patients in school age, as well as its relationship with the state of dental health. A study conducted by the authors demonstrated the high efficiency of health education as a way to normalize the hygienic state of the oral cavity in schoolchildren.

Keywords: compliance, school age, oral hygiene, health education.

Compliance, adherence to treatment - the degree of correspondence between the patient's behavior and the recommendations received from the doctor [3]. With the development of medicine in general and dentistry in particular, the formation and maintenance of patient habits that contribute to the prevention of diseases, which focuses on personal (home) oral hygiene and control of plaque, is of particular importance.

Microbiology made a great contribution to the study of dental plaque, changing our views on personal oral hygiene, the main purpose of which was to prevent the development of the most common dental disease in children and adolescents - caries. When treating a child, the dentist must not only carry out the necessary manipulations in the oral cavity, but also teach him how to brush his teeth, tell about hygiene items and hygiene, clearly demonstrating the basic techniques. Controlled brushing is important, allowing you to evaluate how well the patient has learned the doctor's recommendations, as well as staining of plaque (to identify areas that are not cleaned well enough).

In other words, perhaps the main stage in the activities of a pediatric dentist is health education [1]. Moreover, in each age group, the doctor must use various psychological and pedagogical techniques, interacting not only with the young patient, but also with his parents. A lot depends on the patient: how much he is trained, how conscientious he is in fulfilling the recommendations of his doctor [2].

Younger and middle school age (from 6 to 12). The child's sense of responsibility is increasing. By the middle of this period, most children are able to independently perform basic hygiene procedures. Parents can only help the child clean hard-to-reach places or intervene when the child does not want to perform hygiene measures. During this period, the use of funds for