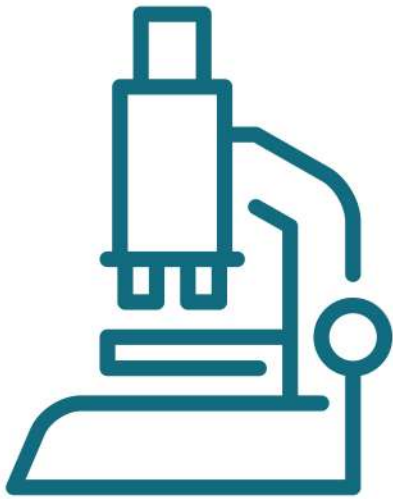


# KHARKIV NATIONAL MEDICAL UNIVERSITY

# ISIC-2020



**KHARKIV NATIONAL  
MEDICAL UNIVERSITY**



**BIOMEDICAL SCIENCES**





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## **DEVELOPMENT OF POSTMENOPAUSAL OSTEOPOROSIS: PATHOPHYSIOLOGY AND POSSIBLE THERAPUTICAL WAYS**

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Menopause or estrogen deficiency predisposes women to bone fractures resulting from reduced bone strength. Osteoporosis is a diffuse disease of the skeletal system which is characterized by a decline in bone microarchitecture, which causes fragility of the bone increasing risk of fracture. It became a major public health issue, with 200 million people who suffering from this disease in the world. Approximately 30% of all postmenopausal women would have developed osteoporosis. This fragility originates from several ways: firstly, increased bone resorption caused by activation and proliferation of osteoclasts; secondly, dissociation of bone formation with resorption ; thirdly, decline of bone quality ; fourthly, decreased calcium absorption from intestine and resorption from renal tubules. Biochemical mechanisms of the development of osteoporosis remain unclear but the main potential pathogenic role is oxidative stress (OS), especially in postmenopausal osteoporosis. Recent studies have shown that reactive oxygen species (ROS), which are pro-oxidant substances, can stimulate the resorption activity of osteoclasts and at the same time reduce a formation of osteoblasts. All these changes arise due to reducing of estrogen level in postmenopausal period. Serum uric acid, which is the most common non-enzymatic endogen antioxidant presented in blood, correlates positively with bone mineral density (BMD). From the other hand, estrogen deficiency leads to weight gain and to change in fat distribution, making the female body more like men. It can probably improves bone health, what may be mediated by the endocrine properties of adipocytes, physiological adaptation of the skeleton to increased physical stress and also by the action of adipokines. The other explanation of postmenopausal OP development is following: estrogen regulates remodeling of bones by controlling the production of growth factors and cytokines from bone cells and bone marrow. Among these factors the most important are: interleukin-1 a (IL-1a), IL-6, tumor necrosis factor (TNF-a), macrophage colony



stimulating factor (M-CSF), and granulocyte macrophage colony stimulating factor (GM-CSF). IL-1 and TNF play the most significant roles, they stimulate bone resorption, inhibit the formation of bones and stop osteoclast apoptosis. Several studies confirmed it, high concentrations of these factors lead to hypercalcemia and bone loss. To sum up, despite popular belief, treatment of postmenopausal osteoporosis could not have done with vitamin D or injections of calcium (considering the fact that abundance of calcium organism would fix with increasing its excretion by kidneys). While antioxidants, hormone replacement therapy and specific inhibitors of mentioned cytokines are able to treat and to stop the development of postmenopausal osteoporosis.

*Pylov Daniel*

**SIGNIFICANCE OF METABOLIC PARAMETERS IN PATIENTS WITH  
TYPE 2 DIABETES MELLITUS AND CHRONIC PANCREATITIS IN  
FORECASTING THE LEVEL OF VASPIN**

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**Introduction.** Adipokines are closely associated with the development of diseases and conditions such as type 2 diabetes mellitus (T2-DM), dyslipidemia, hypertension, atherosclerosis and inflammation, as they contribute to the development of insulin resistance, metabolic disorders, stimulate local inflammation and may affect atherogenesis process. Vaspin, a new serine protease inhibitor produced by visceral adipose tissue is an adipokine with insulin-sensitive and anti inflammatory properties and has angio- and cardioprotective effects.

**Aim.** Identify parameters that affect the level of vaspin in patients with diabetes mellitus type 2 and chronic pancreatitis (CP).

**Materials and methods.** 114 patients were involved in the study. Group I - patients with isolated T2-DM (n=31); Group II - patients with CP (n=23); Group III – patients with a combined T2-DM with CP (n=60). The mean age of the involved patients - 55.04±1.0 years. The control group (n=20) was representative by age and sex.



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