International Journal of
Health Systems and Medical Sciences

ISSN: 2833-7433
Volume 2 | No 6 | june-2023

THE COURSE OF ARTICULAR SYNDROME IN OSTEOARTHRITIS IN PATIENTS WITH DIABETES MELLITUS

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Abstract In bone remodulation, a certain sequence of events is programmed. Under the influence of a complex of stimuli, a group of osteoplasts resorbs the bone. The space of the resorbed bone is covered with osteoblasts, which form a newly formed bone (osteoid) undergoing mineralization. [5, 6].The ratio between bone resorption and new tissue formation is regulated by calcium ions, parathyroid hormone, calcitonin, vitamin D metabolites, as well as sex and thyroid hormones, glucocorticoids, growth hormone and insulin, prostoglandins and the corresponding local action of cytokines. In adults, osteoblasts produce slightly less bone tissue than required. The consequence of this is a negative balance of bone mass.

Keywords: articular syndrome, osteoatrosis, diabetes mellitus

In bone remodulation, a certain sequence of events is programmed. Under the influence of a complex of stimuli, a group of osteoplasts resorbs the bone. The space of the resorbed bone is covered with osteoblasts, which form a newly formed bone (osteoid) undergoing mineralization. [5, 6].The ratio between bone resorption and new tissue formation is regulated by calcium ions, parathyroid hormone, calcitonin, vitamin D metabolites, as well as sex and thyroid hormones, glucocorticoids, growth hormone and insulin, prostoglandins and the corresponding local action of cytokines. In adults, osteoblasts produce slightly less bone tissue than required. The consequence of this is a negative balance of bone mass. This phenomenon is known as “age-related osteopenia” [7].On average, the peak of bone mass is formed by the age of 20, then there comes a period of relative equilibrium, and from 35-40 years, an increased physiological loss of bone mass begins at a rate of 0.3-0.5% per year. Bone tissue is a constantly updating dynamic system, where the processes of remodeling occur throughout life: the destruction of old bone - bone resorption and the formation of new bone − bone formation, or bone formation. At each moment of time, bone remodeling occurs in separate units of the bone structure− the basic multicellular unit, whose function is to maintain bone balance. There are contradictory data in the literature on the frequency of bone system damage in diabetes mellitus. Some authors give up to 78%, and others up to 93%. The defeat of the bone system manifests itself in the form of osteoporosis. Osteoporosis is a decrease in bone mass per unit volume and a violation of the microarchitectonics of bone tissue, leading to an increase in bone fragility and a high risk of their fractures. This definition was formulated at international conferences on osteoarthritis in Copenhagen and Hong Kong and is currently generally accepted [1-4].After menopause in women, bone loss accelerates to 2-5% per year, continuing at this rate until 60-70 years. It has been established that women lose up to 35% of cortical and about 50% of trabecular bone mass on average

throughout their lives. In men, bone losses account for 15-20% of cortical bone and 20-30% of trabecular bone tissue [5].There are several main stages in the pathogenesis of osteoporosis in diabetes mellitus:1. Absolute insulin deficiency reduces the production of osteoblasts collagen and alkaline phosphatase, which are necessary for the formation of the bone matrix and its mineralization, also reduces the stimulation of osteoblasts, mediated through insulin-like and other risk factors.

The direct effect of high glucose concentration due to the end products of glycolization can enhance bone resorption by osteoclasts.

Due to reduced insulin secretion, there may be a lack of active metabolites of vitamin D, which leads to a decrease in calcium absorption in the intestine and an increase in the secretion and activity of parathyroid hormone, which ultimately creates a negative balance of calcium in the body and increases bone resorption.

The role of complications of DM in the development of osteopenia:

a) microangiopathy of the vascular bed of patients may disrupt their blood supply;

b) hypogonadism, especially in patients suffering from diabetes from an early age, prevents the achievement of a normal peak of bone mass in both boys and girls;

c) in type 1 diabetes mellitus, there is a deficiency of STH, which is necessary for the formation of peak bone mass and skeletal development;

d) with type 1 diabetes, body weight deficits often occur, which is an important risk factor for low bone mass, which is possible indirectly through a decrease in the production of estrogens and their metabolism in adipose tissue. Along with these positions, such complications of DM as retinopathy, neuropathy, angiopathy lead to an increased risk of falls and, consequently, to bone fractures [8-11].

It was found that in 90% of cases the metatarsal and tarsal bones are affected, in 10% of cases the ankle joints and the joints of the thumbs of the feet are affected. Radiologically, this manifests itself in the form of narrowing of the articular gap, marginal exophytes, joint defiguration and decontaminating arthrosis. In practice, osteoporosis is asymptomatic and manifests itself in the presence of fractures. The latter occurs with minimal physical exertion in the form of ordinary walking and the patient cannot name the time, place when the fracture occurred. (Yu. Franke, G. Runge, 1995; L.Ya. Rozhinskaya, 1996; S.S. Radio Nova and co.., 1998) [4, 12, 13]. Currently, the following research methods are used (A.Y. Tokmakova et al., 2002) [3]:

a) X-ray morphometry; b) nuclear magnetic resonance; c) iliac wing biopsy and histological examination. OP and evaluate its severity on the basis of such an objective symptom as depletion of the cortical layer, which leads to a decrease in the bone index − the ratio between the diameter of the bone and the thickness of its cortical part. Despite its simplicity, this method determines only the pronounced stages of OP and is not suitable for assessing the dynamics of changes in bone tissue. Unfortunately, radiologically, the diagnosis of OP can be made when 20-30% of the bone mass is already lost, to a large extent, the diagnosis of OP depends on the qualification of the radiologist

Currently, various methods of quantitative bone densitometry are used for the early diagnosis of OP (osteoporosis), which makes it possible to identify already with the loss of 2-5% of bone mass, to assess the dynamics of changes in bone density during the development of the disease and during treatment (O.V. Udovichenko, 2001) [10].

Modern densitometers represent a great achievement of science and technology and make it possible to significantly improve the diagnosis of OP, especially in the early stages. However, they are highly specialized devices that allow indirectly monitoring the saturation of bone tissue with mineral salts that determine the mechanical strength of bones. In addition, to clarify the state of the bone system, it is necessary to conduct biochemical studies. This is the determination of total and ionized calcium, phosphorus in the blood, as well as their daily excretion. Changes in the level of parathyroid hormone,

calcitonin and active metabolites of vitamin D [15, 16]. A.N. Afanasyev et al. [17] studying patients with PREOP (diabetic purulent osteoarthropathy) believe that the muscular system also changes. Swelling, loosening and lumpy disintegration of muscle fibers were revealed, their cytoplasm was homogenized, sometimes encrusted with lime. There is moderate edema and focal dehydration of the intervertebral space, the nuclei of muscle fibers are pyknotically hyperchromic, there is wrinkling and lumpy decay of the nuclei of Sh.A. Yerzhanova [18] based on radioisotope, morphological, and biochemical studies, the development of DGOAP was divided into five stages, which take into account the state of soft tissues (thickness), the thickness of the cortical layer of bone and the time of complete fluid absorption.

Ya.I. Kozhukhova, I.V. Guryeva [19] identified three stages in development DGOAP by examination of local skin temperature; R-graphy of the foot bones. stage − bone destruction and local hyperthermia difference 2.50S;

Stage 1 subacute – moderate hyperthermia (1.5 to 2.50 C);

Stage 2 is chronic − sclerosis with functional restructuring of the structure and normalization of temperature to 1.50C.

A.N. Afanasyev (1998) [17] proposed a classification of DOAP (diabetic osteoarthritis) with GNPS (purulent necrotic lesions of the foot) consisting of several stages.

M.B. Antsiferov, G.R. Galstyan et al. (2001) identified the stages of development of "Charcot's foot":

stage 1 − The presence of edema of the foot, hyperthermia and hyperemia.

2 − Formation of foot deformity. Bone changes in the form of osteoporosis, bone destruction, fragmentation of bone structures are determined by rentgenology.

3 − Pronounced deformation of the feet, the presence of spontaneous fractures and dislocations.

4. Formation of ulcerative defects during infection, rapid development of the disease is possible.

The presence of neuropathy in the patient may cause spontaneous fractures of the bones of the foot,

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