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FEATURES OF THE COURSE OF DIABETIC NEUROSTEOARTHROPATHY IN PATIENTS WITH TYPE 2 DIABETES MELLITUS

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Abstract: Diabetic neuroosteoarthropathy (DNOA, Charcot foot) is a relatively rare complication of diabetes mellitus (DM), which can lead not only to impaired support function of the lower limb in such patients, but also to high amputation. DNOAP is characterized by persistent aseptic inflammation of the bone structures of the foot, which creates significant difficulties in planning treatment measures[1,2,3,4]. In the medical literature, there is data demonstrating the role of individual cytokines and neurohumoral factors in the prolongation of the inflammatory process in diabetes, however, studies identifying significant markers Aseptic inflammation with DNOAP is currently extremely rare[5,6,7].

Keywords: Perception, Practice, Pap smear test, Human papilloma virus, Cervical cancer.

INTRODUCTION

The connection between peripheral neuropathy and ankle injury was first established at the end of the 19th century. neurologist J.-M. Charkot when monitoring patients with late stages of syphilis[8,9,10]. Later, similar deformities were described in leprosy, spinal cord injuries, alcoholism, and in 1976, WR Jordan published a clinical case of arthropathy in diabetes mellitus (DM) [11]. Today, this endocrinological disease is the most common cause of neuroosteoarthropathy. The prevalence of DNOAP, according to the Diabetes Association of Patients with Diabetes Mellitus 2021-2022, is 17.9% for type 1 diabetes mellitus (T1DM) and 7.4% for type 2 diabetes mellitus (T2DM); the acute stage of DNOAP is diagnosed in 0 ,15–2.5% of cases [12,13,14]. Most often, the pathological process is localized in the bones and joints of the feet, mainly the tarsometatarsal and tarsal joints[15]. Cases of DNOAP affecting the knee and even elbow joints are extremely rare. In the available literature, a description of 25 cases of damage to the knee joint was found [16]. We present the result of a long-term clinical observation of a patient with early development of complications of T2DM, diabetic

dermatopathy, widespread DNOAP with damage to the joints of the feet, ankles, knees and elbows[26,27,28]. Diabetic dermatopathy is a common but poorly studied complication of diabetes[23,24,25]. According to the literature, it is observed in 30–50% of patients with diabetes [17,18,19]. To date, about 30 types of skin diseases associated with carbohydrate metabolism disorders have been described. It is noted that dermatopathies can both appear against the background of already manifested diabetes and be its predictors [20,21,22]. Diabetic bullosis (diabetic bullosis, bullosis diabetorum, diabetic bullae) is a dermatosis that occurs in patients with diabetes and is characterized by a rash of subepidermal blisters, most often localized on the anterior surfaces of the legs [29,30]. In modern literature, descriptions of combinations of dermatopathies with other complications of diabetes are extremely rare, and we have not come across any mention of a combination of common DNOAP and diabetic bullosis[31,32,33,34,35].

TARGET. To study the influence of neurohumoral factors and advanced glycation end products on the activity of the aseptic inflammatory process in the bone structures of the foot in people with type 2 diabetes mellitus (T2DM) and DNOAP.

MATERIALS AND METHODS. The study included 88 patients with T2DM (45 men, 43 women). Group 1 consisted of patients with type 2 diabetes (T2DM) and inactive stage of DNOAP (n=43), group 2 (n=45) - persons with T2DM and distal diabetic neuropathy without osteoarticular pathology. The diagnosis of diabetic neuropathy was based on an analysis of the clinical picture and indicators of peripheral sensitivity. Diagnosis of DNOAP and determination of its stage were based on clinical data, results of infrared thermometry and radiation methods for studying the bone structures of the foot. The patients underwent general clinical examinations, radiography of the foot, MRI of the foot, determination of C-reactive protein (CRP), calprotectin, copeptin, glutathione peroxidase 1 (GP1).

RESULTS. Based on the results of examination and palpation of the feet, as well as analysis of the temperature gradient of the skin of the affected and contralateral extremities (infrared thermometry), DNOAP was detected and the status was determined. The diagnosis of this complication. The diagnosis of the chronic stage of DNOAP was confirmed by the results of MRI and clinical picture (no difference in skin temperature on symmetrical areas of the feet). According to the results of laboratory analysis, statistically significant differences in copeptin values were revealed: in group 1 - 0.232 ng/ml [0.147; 0.342], in group 2 - 0.115 [0.065; 0.203] ng/ml ($p < 0.05$) and CRP: in group 1 - 7.113 mg/l [2.453; 16.505], in group 2 - 2.187 mg/l [1.131; 5.567] ($p < 0.05$), leukocyte counts in the groups did not differ significantly: group 1 - 7.86 [6.40; 9.00] 10^9 , group 2 - 7.00 [6.00; 8.15] 10^9 ($p > 0.05$). There was a trend toward increased levels of calprotectin and GP1 in the DNOAP group, but the differences were not significant. Thus, calprotectin in group 1 was 1.948 [1.229; 2.969] $\mu\text{g/ml}$, in group 2 - 1.692 [1.16; 2.514] $\mu\text{g/ml}$ and GP1 in group 1 - 24.72 ng/ml [20.1; 31.82], in group 2 - 22.98 [18.94; 31.2] ng/ml.

CONCLUSION. The study found statistically significant differences in copeptin levels and CRP: in patients with DNOAP, their values were significantly higher, which indicates the persistence of the aseptic inflammatory process in the bone tissue of patients even in the chronic stage of the complication. This data may provide assistance in making a decision on the use of one or another method of unloading the affected joints, which will affect the clinical prognosis. The study of neurohumoral markers of arthropathy in the blood serum of patients with T2DM is carried out for the first time, and therefore comparison with the results of other authors is difficult. It can be assumed that copeptin and CRP are significant markers of persistent inflammation of the osteoarticular structures of the foot in DNOAP.

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