DOI

MOLECULAR MECHANISMS OF PERIODONTITIS DEVELOPMENT IN PATIENTS WITH TYPE 2 DIABETES MELLITUS AND MEASURES FOR THEIR TREATMENT

Komilova Adiba,
Hadzhimetov Abdugafur,
Usmanova Shoira
Tashkent State Dental Institute
Tashkent, Uzbekistan

Target: The analysis of modern data on the molecular mechanisms of the development and progression of insulin-dependent diabetes is presented. The leading role of HSP72 reduction in pancreatic cells and target organs for insulin in disorders of the structure of insulin molecules, its receptor perception, and mitochondrial functioning in DM2 is substantiated.
Materials and methods. In this regard, the aim of the study was to determine the features of the microbial composition of the biofilm of the dentoalveolar furrow in chronic periodontitis and the combination of chronic periodontitis with type II diabetes mellitus based on the use of modern molecular and metagenomic technologies. Patients and methods We conducted a comprehensive dental examination of 84 patients who applied to the dentist for treatment or prophylactic purposes, who met the criteria for inclusion in the study and filled out an informed consent. Group 1 consisted of 22 patients (11 men and 11 women aged 47-63 years), in whom type II diabetes was combined with the phenomena of chronic periodontitis. They were diagnosed with periodontitis of moderate severity, confirmed by a detailed description of the dental status (Table. 1) and the results of PCR diagnostics for the detection of genetic markers of periodontopathogen bacteria of the 1st and 2nd order. Group 2 included 30 patients (13 men and 17 women aged 42-60 years) who did not complain, and inflammatory periodontal diseases were detected in them when they applied for the treatment of caries and its complications. Based on clinical data, indicators of periodontal indices and the results of PCR diagnostics, they were diagnosed with "periodontitis of moderate severity", confirmed by a detailed description of the dental status and the results of PCR diagnostics. Control group 3 was formed by 22 individuals (9 men and 13 women aged 25-28 years) with a healthy (intact) periodontal disease and the absence of type II diabetes. This group includes only subjects with negative results of PCR diagnostics for the detection of genetic markers of periodontopathogen bacteria of the 1st and 2nd order. The hygienic condition of patients in comparison groups 1 and 2 was satisfactory and comparable. According to the anamnesis, the following comorbid diseases were detected in patients of these groups: vegetative-vascular dystonia, symptomatic arterial hypertension, arterial hypertension. The prescription of periodontitis disease was 5-10 years. The previous periodontological treatment of patients of both groups included removal of dental deposits, local anti–inflammatory therapy and, according to indications, curettage of periodontal pockets. In the subjects of the control group 3 (without chronic periodontitis and type II diabetes), the hygienic condition of the gums was good, and the periodontal indices corresponded to normal values. The observation period for all subjects was 1.5 years. The material was taken from 4 sites in the area of the dentoalveolar furrow using sterile paper endodontic pins (No. 30), which were placed in a test tube with 0.2 ml of saline solution.
Results. As is known, many proteins cannot independently acquire their functionally active forms, as a result of which they tend to form distorted and inactive aggregates. The ability to transform and form aggregates has been proven for insulin molecules [5]. In cells, either special enzymes (foldases) or a group of auxiliary specialized proteins called chaperones

https://wordlyknowledge.uz

contribute to maintaining the functionally correct structure of proteins. As it turned out, patients with DM2 had a reduced expression of chaperone HSP72, which clearly correlated with reduced insulin sensitivity [6]. It can be assumed that it is the deficiency of this chaperone in β-cells that does not allow the insulin molecules synthesized in the human pancreas to acquire the functionally necessary tertiary structure for interaction with the receptor, which is the most important cause of insulin resistance. 71 It was further found that not only in the pancreas, but also in the cells of peripheral organs consuming glucose (in skeletal muscles, liver, etc.), there is also a reduced expression of the HSP72 protein. It is important to note that a decrease in the formation of HSP72 was combined with increased phosphorylation of JNK – inflammatory cytokine, i.e., its activation. It is known that inflammation initiates the secretion of a number of inflammatory cytokines, such as tumor necrosis factor α (TNF-α) from macrophages and/or adipocytes, which leads to the activation of serine threonine kinases: c-jipamine-terminal kinase (JNK) and kinase inhibitor kB (IKK) in insulin-sensitive tissues. Under these conditions, the conformation of specific phosphatases is disrupted, which usually inhibit the activity of both JNK itself and the activity of proteins associated with JNK activation. [7]. Activated JNK and IKK are able to phosphorylate IRS-1(IRS-1) by Ser-307, which makes this protein a poor substrate for the activated insulin receptor, leading to disruption of the activity of various signaling pathways, including those responsible for glucose uptake by the tissue. The importance of JNK and IKK in insulin resistance is emphasized by the fact that the genetic disruption of these pathways in mice provides protection against insulin resistance induced by obesity.

Conclusion. Diabetes mellitus and periodontal disease are among the most prevalent human disorders. Frequently these two medical problems are present concurrently in many people. For years, attempts have been made to relate these two processes. In recent decades, many studies have reported that the presence of diabetes mellitus increases the incidence and severity of periodontal disease. There appears to be a relationship between the two processes, whereby the consequences of diabetes mellitus serve as modifiers of the expression of periodontal pathology. There are many aspects of this relationship that remain unclear. In this context, it has not been clarified whether good metabolic control influences the success of periodontal treatment or vice versa. Likewise, it remains to be determined whether the mechanisms involved are the same in both type 1 and type 2 diabetes mellitus. For proper treatment of diabetic patients with periodontitis, the medical and dental professions should work together The signs, symptoms, and clinical presentation of periodontitis need to be recognized by physicians so that diabetic patients are promptly referred to dentists for treatment and similarly dentists should understand the parameters of glycemia that are used to establish a diagnosis of diabetes and the methods used in diabetic care, thus potentially preventing further complications.

https://wordlyknowledge.uz