ASSESSMENT OF CLINICAL PERIODONTAL MODIFICATIONS IN JUVENILE DIABETES

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ABSTRACT

Aim of the study The main goal of our study was to highlight the changes of preclinical and clinical periodontal status indices in a group of children and teen-agers with insulin-dependent diabetes (IDDM). Material and methods For objectifying our purpose 60 children, aged 7 to 18 years divided into three groups of 20 subjects each were examined: control group - children without diabetes, with good general condition, but with varying degrees of periodontal involvement, IDDM batch with good metabolic control (HbA1c <7,5%) and IDDM group with poorly metabolic control (HbA1c >7,5%). On all these subjects the following parameters were assessed: plaque index - Quigley-Hein (QHI), gingival index (GI), clinical attachment loss (CAL). The gingival crevicular fluid (GCF) volume from mesial sites of the incisors and first permanent molars was also evaluated. **Results** Our results show no significant differences in plaque index values between control and diabetic groups. Statistically significant differences were recorded when considering gingival index, higher values being recorded in children with IDDM and especially those with poorly controlled metabolic control, compared to control group. Clinical attachment loss registered augmented levels in children with IDDM compared to the control group, and in terms of measuring the GCF volume, values have been found significantly elevated in diabetics compared to control group, the largest being recorded in poorly metabolic controlled diabetic children and teen-agers. Conclusions Among the values of the evaluated indices, the highest increases were observed with parameters associated to significant inflammation, GI and GCF volume respectively. Our results are consistent with scientific literature in the field that claims inflammation as a major mechanism involved in the pathogenesis of both disorders: periodontal disease and diabetes.

Keywords: periodontal disease, juvenile diabetes, gingival crevicular fluid, inflammation

INTRODUCTION

The association between diabetes and periodontal disease is studied for over 50

years. Both diabetes mellitus (DM) as well as periodontal diseases are chronic inflammatory diseases with a major impact on the health of

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the population [1]. Diabetes affects about 20 milions of Americans, about 35 to 40% of whom have not received a diagnosis [2]. On the other hand 75% of the adult americans present gingivitis (inflammation of the gums around the tooth), of which approximately 35% have periodontitis (inflammation involving the tooth supporting structures, including the periodontal ligament and alveolar bone) [3].

Type 1 diabetes, wich represents about 5 to 10 percent of all cases in the United States, results from autoimmune distruction of insulin-producing □-cells of the pancreas, leading to total loss of insulin secretion [4]. A patient with type 1 diabetes must take exogenous insulin to remain alive − hence, he becomes one of the "insulin-dependent diabetes" individuals. Besides, this form of diabetes (insulin-dependent) is the predominant form that is found in children and adolescents. Type 1 diabetes has been associated to a five fold increased prevalence of periodontitis in teenagers [5].

An increased risk and earlier onset of periodontitis occur in both insulin dependent and non-insulin dependent diabetes mellitus [6], probably because impaired immune function. The literature points out that as many as 10% to 15% teenagers with insulin dependent diabetes mellitus have significant periodontal disease [7].

Evidences consistently reveal that diabetes is a risk factor for increased severity of gingivitis and periodontitis [8]. Conversely, periodontitis is a risk factor for worsening glycemic control in patients with diabetes, and may expand the risk for diabetic complications [8,9].

Numerous mechanisms have been elucidated to explain the impact of diabetes on the periodontium status. While inflammation plays an obvious role in periodontal impairment, evidence in the medical literature also supports the role of

inflammation as a major component in the pathogenesis of diabetes and diabetic complications. A number of researches suggest that as an infection process with prominent inflammatory component, periodontal disease can adversely affect the metabolic control of diabetes [10].

The aim of our study was to evaluate some parameters of periodontal status in a group of children and adolescents with insulin-dependent diabetes (IDDM) and compare their values with those of systemically healthy age-match children and teenagers.

The objectives of this study envisaged determination of the changes in these clinical parameters of periodontal damage in the context of systemic insulin dependent diabetes in children and adolescents, correlated to IDDM metabolic control and also inflammatory process.

MATERIAL AND METHODS

In order to achieve our goal, 60 children aged 7-18 years from urban and rural areas of Moldova were enrolled in the study. The young subjects have been subdivided into three groups (n=20 each), as follows: Group 1 - control - 20 children with good general status who addressed the dental clinic for various odontoperiodontal diseases; Group 2 -n=20 IDDM children with good metabolic control (HbA1c <7.5%) - registered and evaluated within St. Mary Children's Hospital Iași, department of nutrition and metabolic disorder; Group 3 - n=20 children with poorly controlled diabetes (HbA1c > 7.5%) also registered within the mentioned department and hospital.

On all these subjects the following parameters were evaluated at the incisors and first permanent molar: 1. Plaque index Quiqley-Hein (QHI) - performed with erythrosine tablet, each surface holding a score from 1 to 5 in agreement with the index described by Quiqley Hein; 2. Gingival index

(GI) Silness-Loe was achieved using manual periodontal samples each site holding a score from 1 to 3 according to Silness-Loe [11], levels of 2 and 3 denoting bleeding sites; 3. Clinical attachment loss was evaluated with periodontal probe through: a) probing depth, defined as the distance between the gingival margin and the bottom of the probeable pocket to the nearest whole milimeter, b) location of the gingival margin, the distance between cementoenamel junction and the gingival margin to the nearest whole milimeter. The two parameters above were used to compute clinical attachement level. Any loss of attachement represents destruction of periodontal support around teeth. 4. Gingival crevicular fluid (GCF) volume assessment: after isolating the tooth with cotton roll, GCF was collected by placing filter paper strips (Periopaper, Pro Flow, Amitywille, NY) into the mesial sulcus of the permanent incisor and premier molars until mild resistance was cented, and left in place for 30 seconds. The volume of the sample on the filter paper strips was measured using a calibration unit (Periotron 8000, ProFlow). 5. The degree of control of the diabetic disease - through analysis of the glycated hemoglobin - HbA1c values: levels of HbA1c <7.5% were considered for good metabolic control, HbA1c values> 7.5% indicating a poorly metabolic control of the diabetic disease.

RESULTS AND DISCUSSIONS

Values of the plaque index (QHI) have been displayed in Figure 1, where one can observe: QHI control group = 3.3, QHI of the good metabolic control IDDM subjects = 3.4 and poorly metabolic controlled IDDM QHI = 3.6. Therefore, considering this assessment tool used to evaluate the thickness of plaque at the gingival margin, no significant differences between the subjects of the three groups were recorded into our study. The

results are consistent with some of the literature [5], but in contradiction with others that reported a much greater plaque presence in children with IDDM compared to non-diabetics [12].

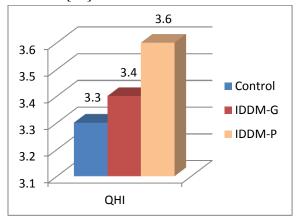


Figure 1. Levels of the plaque index (QHI) in Control group, good control of diabetes disease (IDDM -G) and poor metabolic control of the disease (IDDM-P)

On the other hand gingival index (GI) differences. registered significant minimal inflammation (GI = 0.9) in subjects of the control group, by contrast to subjects with IDDM in which inflammation and bleeding was very important, with GI = 1, 4in subjects with good metabolic control of IDDM and GI = 1.8 in IDDM subjects with poorly metabolic control (Figure 2). otherwise consistent with other results [13,14].

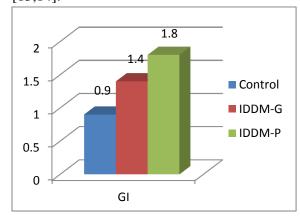


Figure 2. Levels of the gingival index (GI) in Control group, good control of diabetes disease (IDDM –G) and poor metabolic control of the disease (IDDM-P)

Clinical attachment loss (CAL) was detected with levels between 1-2 mm in three patients with good control of the diabetic disease, higher values (CAL > 2 mm) being recorded in only two subjects with poorly controlled IDDM, all these patients aging between 12-16. The literature data upon this parameter are quite contradictory. Poor glycemic control in patients with diabetes has been also asociated with an increased risk of progressive loss of periodontal attachment and alveolar bone over time [15,16,17]. However, other studies failed to identify any significant relationship between glycemic control and periodontal status [18, 19], reporting exclusively gingivitis in children with IDDM, clinical attachment loss being rather absent.

GCF volume measurement shows more elevated mean values in diabetic children versus control (Figure 3), the largest levels being recorded in children with poorly controlled metabolic disease (GCF volume = $1.02~\mu$ l), followed by $0.87~\mu$ l in the group of children with good metabolic control of diabetes and $0.46~\mu$ l in control group. According to Del Fabro et al (2001) and Griffiths (2003) the volume of GCF is directly proportional to the stage of periodontal inflammation, which underscores its importance as a valuable assessment tool [20,21].

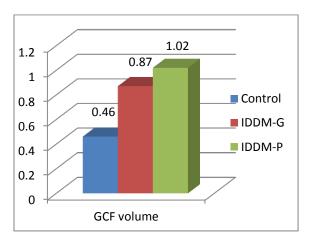


Figure 3. Levels of GCF volume (µl) in Control group, good control of diabetes disease (IDDM –G) and poor metabolic control of the disease (IDDM-P)

CONCLUSIONS

Our results indicate the existence of important relationship between the presence of metabolic impairment of diabetes nature in children and adolescents and changes in the marginal periodontium of these subjects, important correlations being established particularly with indicators of inflammation (gingival index and gingival crevicular volume). Clinical attachment loss recorded only in five of the diabetic children aged between 12-16 years, enrolled in the survey. The literature data that consider IDDM child and adolescent impairment of the periodontium are conflicting and scanty, our results being consistent with some of them but in disagreement with others.

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