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The relationship between insulin resistance and periodontitis is not affected by Mediterranean diet in a Spanish population

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ABSTRACT

Objetive: To examine the insulin resistance measured by surrogate indices in subjects with and without periodontitis and to find out any correlation among dietary intake with insulin resistance.
Design: Fifty-five patients were recruited to participate in this cross-sectional study. Insulin resistance measured by the homoeostasis model assessment (HOMA-IR) and the quantitative insulin sensitivity check index moreover glycaemia, creatinine, uric acid, high density lipoproteins, low density lipoproteins, very low density lipoproteins and triglycerides among others. True periodontal disease was elucidated through the examination of probing pocket depth, clinical attachment level, recession of the gingival margin and gingival bleeding. The statistical analyses used were the student's T-test for independent variables, Kolmogorov-Smirnov if variations were homogeneous; if not, the Mann-Whitney U Test was applied instead. Correlations between variables were assessed using Pearson's correlation coefficients. True periodontal disease was confirmed through the greater values of probing pocket depth, clinical attachment level, gingival margin and gingival bleeding in the periodontitis group in comparison with non-periodontitis group.
Results: Insulin resistance was evidenced by the greater values of HOMA-IR as well as by the lower quantitative insulin sensitivity check index values in the periodontitis group. Fasting insulin, glucose, uric acid, creatinine, low density lipoproteins, triglycerides and very low density lipoprotein levels were significant higher in periodontitis group. Pearson's correlations did not show any association among diet data and insulin resistance parameters in periodontitis patients.
Conclusion: A putative systemic relationship between insulin resistance and periodontitis exists but it does not seem conceivable any effect of diet over such relationship.

1. Introduction

Periodontitis affects both the tooth supporting tissues and the host inflammatory immune response. This condition is due fundamentally to an ecological imbalance between the normal microbial biofilm on teeth and the host tissues (Bullon et al., 2009). However, there is increasing evidence linking periodontitis to other systemic conditions, such as diabetes (Llambes, Arias-Herrera, & Caffesse, 2015; Wu, Xiao, & Graves, 2015), cardiovascular disease (Bullon et al., 2011; Bullon et al., 2012; Kholy, Genco, & Van Dyke, 2015; Saffi et al., 2015) or rheumatoid arthritis(Leech & Bartold, 2015). There are many existing papers on the relationship between diabetes or metabolic syndrome and periodontal disease (Li et al., 2015; Shimoe et al., 2011; Watanabe & Cho, 2014), and evidence of the relationship between a major marker of diabetes, glycated hemoglobin (HbA1c), C- reactive protein and periodontal parameters (Altamash, Klinge, & Engstrom, 2015; Bullon et al., 2009; Islam, Seo, Lee, & Moon, 2015; Lopez et al., 2012) exists. However, there is controversial reports about the relationship

Abbreviations: HOMA-IR, insulin resistance measured by the homoeostasis model assessment; QUICKI, quantitative insulin sensitivity check index; PPD, probing pocket depth; CAL, clinical attachment level; GM, gingival margin.

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between insulin resistance measured by surrogate indices and periodontitis (Gurav, 2012; Lim et al., 2014; Timonen et al., 2013).

Insulin resistance is a condition in which normal amounts of insulin are inadequate to elicit a normal insulin response from fat, muscle and liver cells (Bullon et al., 2009). This condition leads to an eventual hyperglycaemia which has a systemic deleterious effect, mainly acting over the vasculature (Rhee & Kim, 2015; Thiruvoipati, Kielhorn, & Armstrong, 2015). The euglycaemic hyperinsulinaemic clamp technique is the gold-standard method of measuring insulin sensitivity (DeFronzo, Tobin, & Andres, 1979). However, this method is expensive, time-consuming and only used in research. Hence, more simple methods for measuring or estimating insulin sensitivity have been developed. The homoeostasis model assessment (HOMA-IR) (Lim et al., 2014; Timonen et al., 2013) and the quantitative insulin sensitivity check index (QUICKI) (Katz et al., 2000) have become the most widely used surrogate indices. Both are based on fasting plasma glucose and insulin values, and correlate well with the glycaemic hyperinsulinaemic clamp.

It is widely accepted the role of nutrition on insulin resistance (Drehmer et al., 2015; Turner, Keogh, & Clifton, 2015), and several studies have reported an important relationship among diet and nutritional status and periodontitis (Jenzsch, Eick, Rassoul, Purschwitz, & Jentsch, 2009; Zare Javid, Seal, Heasman, & Moynihan, 2014), nevertheless, there is no evidence regarding the effects of diet on insulin resistance once the periodontal disease is settled.

Moreover, all these studies conclude that future studies should be performed to support this hypothesis and to confirm the role of periodontitis in the deterioration of insulin resistance.

Hence, the aims of this study were to find out some correlation among dietary intake with insulin resistance in middle-aged Spanish patients with periodontitis, and to demonstrate some relationship between insulin resistance, measured by HOMA-IR and QUICKI indices, and the periodontal status in the same study population.

2. Methods and materials

2.1. Study population and clinical examination

During 18 months a total of 55 subjects attending in our University Dental School for several dental complaints were recruited for this study. All of them must have a minimum of 15 remaining teeth, excluding third molars. Exclusion criteria were: any periodontal treatment during the past 6 months; any use of anti-inflammatory and/or antimicrobial drugs during the past 6 months; and a medical history for immunodeficiency or pregnancy. The study protocol was approved by the Committee of Ethics and Research of University of Seville's Odontology Faculty. Written informed consent was obtained from all participants before examination. The periodontal examination included the assessment of: probing pocket depth (PPD), measured from the gingival margin to the most apical penetration of the probe; clinical attachment level (CAL), measured from the cement-enamel junction to the most apical penetration of the probe; the recession of the gingival margin (GM); and gingival bleeding on probing, evaluated as presence or absence of bleeding after the most apical penetration of the probe. Parameters were measured at six sites on every tooth by means of a calibrated periodontal probe (Hu-Friedy®, Chicago, IL, USA), 15 mm in length and 0.35 mm in diameter. Established periodontitis was diagnosed according to criteria proposed by Machtei et al., 1992 (Machtei et al., 1992), i.e. the presence of CAL ≥ 6 mm in 2 or more teeth and one or more sites with PPD ≥ 5 mm. Those participants not fulfilling these criteria were assigned to control group. Finally, periodontitis group consisted of 30 subjects, and control group 25 subjects.

2.2. Dietary assessment

The habitual diet of the subjects was daily checked with 24 h dietary recalls using food records of measured and weighed food intake and all recipes of homemade dishes during one week. In particular, three recall days were registered at the day of recruitment by a dietician. Another four days (including one weekend day) were registered by the patient, starting on the first day after recruitment, with further supervision by the dietician. The content of macronutrients and selected micronutrients in the diet was calculated using the computer program ALIMENTACION Y SALUD 0698.046 (BitASDE General Medica Farmaceutica, Valencia, Spain).

2.3. Biochemical parameters

Blood samples from participants were collected by venipuncture in the antecubital fossa and were centrifuged within 2 h since sampling. Aliquots were prepared and stored at −80 °C until analysis. Plasma glucose, creatinine, uric acid, urea, alanine aminotransferase (ALT), aspartate aminotransferase (AST), total cholesterol, high density lipoproteins (HDL), LDL, VLDL and triglycerides were analyzed using an automatic analyzer (Roche-Hitachi Modular PyD Autoanalyzer, Roche Laboratory Systems, Mannheim, Germany). Plasma insulin was analyzed by radioimmunoassay using an automatic analyzer for 20 microparticles (Axsym, Abbott Laboratories, Chicago, IL, USA).

Insulin resistance and tissue insulin sensitivity were calculated by using the HOMA-IR index, as defined by the equation HOMA-IR = fasting glucose (mmol/L) x fasting insulin (μUI/mL)/22.5 (Matthews et al., 1985), and the QUICKI index, as defined by the equation QUICKI = 1/[log fasting insulin (μUI/mL) + log fasting glucose (mg/dL)] (Katz, Chaushu, & Sgan-Cohen, 2000).

2.4. Statistics

Data were expressed as means ± standard error of the mean (SEM). Comparisons between periodontitis and control groups were analyzed by the student's T-test for independent variables. When the variations were homogeneous they were analyzed by Kolmogorov-Smirnov; if not, the Mann-Whitney U Test was applied instead. Correlations between variables were assessed using Pearson's correlation coefficients. Analyses were performed using the software program SPSS/PC 15.0 (Chicago, IL, USA). P values less than 0.05 were considered significant.

3. Results

Fifty-five patients met the inclusion criteria and accepted to participate in the study; 30 were diagnosed as chronic adult periodontitis. The clinical results of the study population (control group and periodontitis group) are listed in Table 1. No significant differences between both groups for any of the parameters shown in Table 1 were found. The assessment of true periodontal disease is a key factor in this study and it was confirmed through the measurement of PPD, CAL, GM and gingival bleeding in the whole study population. We observed statistically significant greater values of PPD, CAL, GM and gingival bleeding in the periodontitis group (3.02 ± 0.09; 3.84 ± 0.2; -0.75 ± 0.12 and 63.6 ± 4.4 respectively; $P < 0.01$) while lower values were observed for the non-periodontitis group (2.34 ± 0.09; 2.49 ± 0.1; -0.15 ± 0.04 and 47.93 ± 4.0 respectively; $P < 0.01$) (Table 2).