THE PREVALENCE OF PERI-IMPLANTITIS IN PATIENTS WITH OBESITY. A REVIEW OF LITERATURE

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Abstract:
Obesity is a chronic , complex, multifactorial disease, which develops as a result of the interaction between genotype and life style. Up to now has not been fully elucidated the mechanism by which it occurs and is developing obesity, but it has been noted that it involves both social factors, as well as those cultural, behavioural, physiological, metabolic, and genetic. The aim of this review is to evaluate how different sistemic diseases, beside type 2 diabetes,smoking , obesity may be risk factors in the onset of perimplantitis.
The association between periodontal disease and obesity has been shown in several studies to different age groups. Excessive adipose tissue produces a reduced inflammation which is a cause of oral amendments.
In obesity was noticed this inflammation which can be a point of contact between this and periodontal disease or periimplantitis disease.
Keywords: *periodontal disease, obesity, peri-implantitis.*

Introduction.

Currently, over 1.1 billion adults worldwide are overweight, and 312 million people are diagnosed with different degrees of obesity [1]. Becoming anxious is the fact that at least 155 million children worldwide are overweight or obese, according to the International Obesity Task Force. The World Health Organisation (WHO) has revised the definition of obesity, so to adjust for the different communities and ethnic groups, and this extended definition may reflect a greater prevalence- 1.7 billion people with categorized as overweight worldwide [1].

The obesity can be defined using body mass index (BMI) called the Quetelet Index. It is actually a ratio of body weight (expressed in kg) and height (measured in m ²) [2]. A BMI value between 25-29.9 Kg/m ² indicate a over-weight person, and a BMI equal to or > 30 kg/m ² defines obesity.

In the past 20 years, the obesity rate has tripled in countries in the developing world, one of the explanations of this phenomenon do to the fact that they have adopted a western lifestyle that involves decreased physical activity and overeating processed food, some with high caloric index. This lifestyle is both in adults and in children, and it has thus been observed the increasing prevalence of excess weight gain from 10 to 25% and the prevalence of obesity from 2 to 10% [1].

Obesity affects regular organismal level multisistemic favoring human conditions for the occurrence of certain diseases, such as hypertension, type 2 diabetes, atherosclerosis, cardiovascular diseases, cerebrovascular syndrome of sleep apnea, the disorder of gastroesophageal reflux, infertility, gout, osteoarthritis, respiratory disorders, metabolic syndrome, dyslipidemia and some types of cancer [4]. Characteristics of obesity are given three metabolic changes: hiperinsulinemia, hyperglycemia, hyperlipidemia. The changes observed in obesity are also associated with acute and chronic inflammation [5]. (Table 1)

Periodontal disease is known as a global issue with a large growth in the developed countries and in the developing world, it is de-fined as a chronic inflammation of periodontal tissue due to accumulation of plaque on the the dental surface. However, the destruction of the

periodontal structures can be caused by local factors such as dental biofilm or systemic factors such as diabetes, HIV/AIDS or other diseases that can depress immune response of the host organism [3]. The association between periodontal disease and obesity has been shown in several studies carried out in different stages of life, from childhood until adulthood. The relationship between obesity and periodontal disease was intuited in the year 1977 by Perlstein outlines & Bissada. They have noticed changes in histopathology obese hypertensive rats periodontal [6].

The association between periodontal disease and obesity has been shown in several studies to different age groups. Excessive adipose tissue produces a reduced inflammation which is a cause of oral amendments. In addition, it was observed an increased inflammatory cytokine production (interleukin IL-1 beta) with its important role in the development and progression of periodontal disease through a greater susceptibility to bacteria due to host immune response. Depending on the stage of life that occurs as obesity, periodontal disease, including degree or severity can be classified into several groups with a low risk, high or increased. At an early age and mature age, obesity fits into a group with high risk in periodontal disease. A low risk of developing periodontal disease we met people who have reached adulthood at normoponderability [7].

Acute/chronic inflammation is implicated in the etiology of periodontal disease, and mucositis periimplantitis, respectively. In obesity was noticed this inflammation which can be a point of contact between this and periodontal disease or periimplantitis disease [8]. (table 1

Table 1

Design study	Type of the study	Metabolic Syndrome	No. Subjects	Results
Elangovan et al [22]	Cross-sectional study	Obesity	73	Modest positive correlation but statistically significant between the TOILET and the levels of IL-1 β in PISF ($P < 0.05$) between the plate and the index levels between IL-1 α in PISF
P. PAPI et al [23]	Systematic rewiev	Metabolic syndrome	19	12 studies on the correlations between diabetes, Hyperglycemia-periimplantitis 5 studies on the correlations between cardiovasculare diseases and peri-implantitis
Vohra, Alkhudhairy, Al-Kheraif, Akram, Javed [20]	Cross-sectional study	Obesity	84	Statistically significant differences periimplantitis and C-reactive protein in overweight persons
Hazem A. Bissada NF Demko C. Paes A Lang LA [24]	Cross-sectional study	Obesity	220	The study showed a small percentage of implant failure regardless of BMI patients. There was observed no association between obesity and implant failures. Obese patients have a higher risk of developing complications.

Depending on the stage of life where it appears obesity, periodontal disease has different degrees of severity. Childhood obesity persisting in maturity falls into the risk group in term of prevalence periodontal diseases. A low risk of occurrence of periodontal diseases we meet to maturity normal weight persons [7].

There are numerous studies that have analyzed the correlation between periodontal disease and diabetes. Thus, there are a considerable number of available evidence in the literature to describe the biological mechanisms associated with complications of diabetes, such as peripheral neuropathy, nephropathy, or arteriopathy [8].

Treatment of obesity should focus substantial weight loss for a prolonged period. Treatment options in the decision of overweight and obesity must be taken into account and the presence of comorbidities. Thus, there is evidence that shows the unanimously accepted a reduction of risk factors for diabetes and cardiovascular disease along with weight loss in overweight and obese individualsother hand,

improvements in physical activity. All these measures are aimed at preventing the progression of obesity and decrease moderate weith.

Losing weight has as consequence of reducing blood pressure, so people overweight and hypertensive for those non-hypertensive, reduce serum triglycerides and increase high-density lipoprotein (HDL cholesterol), produces a certain reduction of cholesterol and low density cholesterol (LDL) in serum. Also losing weight reduces blood glucose levels in people without diabetes, overweight and obese. Moreover, losing weight reduces blood glucose and hemoglobin values glicozilate (HbA1c) in some patients with type 2 diabetes [6].

There are some differences between the gingival flora of patient with diabetes that healthy, but most studies of culture have not showed major differences in this regard. Correlation of periodontal disease is supported by the immune response altered in patients with diabetes [8].

Adipose tissue secretes leptin. It has the role of activation of the immune system because it stimulates cytokine production by macrophages and phagocytes. Recently proved the role of leptin in bone formation and was reported to showcase her in blood and inflamed gingival edge. This observation led to the conclusion that leptin has a possible role in the development of periodontitis [9].

Techniques of oral rehabilitation included dental implant as a method of dental treatment with the current frequency indication to replace natural teeth lost in the dental arches. Peri-implantitis is an emerging disease like periodontal disease occurs mainly because of aggressive bacterial attack and a low immune response of the host with a spontaneous reaction progressive if left untreated [10]. The progression of disease rates peri-implant and periodontal disease differ in the case of peri-implantitis tissue destruction is faster due to lack of insertion of the collagen fibers around the implant which allows for faster expansion the inflammatory process at the level of the alveolar bone [11].

Peri-implant disease has two phases: mucositis, initial phase, which is reversible, in which pathological changes are represented only by soft tissue inflammation (mucous membrane) surrounding the implant. The second phase, the periimplantitis, is characterized by an inflammatory reaction that affects both soft tissue and bone causing bone loss over time and the appearance of puffiness around the implant accompanied by bleeding from survey and drainage [12].

In recent years, it is increasingly clear that some risk factors-biological complications but also some incorrect techniques are responsible for periimplantitis disease occurrence. The latest theories bring the following explanations: lack of preoperative tomography analysis of the jaw, a incorrect prostethic rehabilitation , poor oral hygiene , cement around the implant after stage of prosthetic fixation. Also, patients with chronic or aggresive periodontal have increased risk of developing the condition. These patients require before using dental implant prosthesis to follow a treatment of periodontal disease. However, there is no common diagnostic criteria and therefore is still difficult to determine the exact cause of occurrence of peri-implantititis [13].

The prevalence of peri-implantitis remains controversial and relatively unknown to most ,depending on the method and study population: Derks and Tomasi reported in a survey conducted on the Swedish population percentage of 43% and 22% of mucositis peri-implantitis after a period of 9 years [13].

Implants affected by periimplantitis-considering the disease in a high percentage depending on the degree of inflammation and the amount of bone lost keeps its stability to maxillary bone as a result considering the periodisease can progress asymptomatic and patients arrive at medical dentist with large loss of bone around the implant. It is therefore important that monitoring of each patient affected by means of dental implants to be made with riguare [12].

Practitioners are finding it even more difficult to understand the factors that lead to disease they come into the equation this system diseases, they tried even associations between severity of peri-implantitis and risk indicators. Along the years only a few studies have focused attention on co-existing medical conditions and peri-implantitis. (tabel1)

Van Vezen *et al.* (2015) reported a success rate of dental implant of 91.6% and only 7% of cases of periimplantitis after 10 years [14].

A systematic review, recent meta-analysis reported a higher prevalence of peri-implantitis ,21.7% of authors saying that this percentage is an understatement. Widely accepted a loss of 1.5 mm crestala material non-pathological bone around the dental implant in the first year of the insert and a further loss of up to 0.2 mm [11].

Studies on the occurrence factors favoring local peri-implantitis showed a 2.2 times higher in people with periodontal disease, 3.6 times higher in the case of cemented crowns versus screwed crowns, 16.1 times higher in whole rehabilitation compared to those unidentare [15].

Statistical analysis on multiple levels identified risk factors, location of the dental implant in the jaw and male gender [16].

A review has demonstrated that type 2 diabetes mellitus has been regarded as an absolute contraindication regarding dental implant insertion. From these patients failure rate percentage was between 0%-14.3% in 17 studies analyzed, but the majority of investigations have not reported the presence of HbA1c [17,18]. Can`nt be omitted smoking effect on the immune system and decrease of damage over osseointegration dental implant [19].

Another study was aimed at comparing inflammatory parameters and radiological clinic periimplantitis in patients with different levels of obesity and the correlation between these parameters with CRP levels. The result of this study has been positive in terms of the groups of patients with a large degree of obesity, concluding that peri-inflammatory parameters distribution parks clinics and radiological and serum CRP-s values were significantly higher in patients with a severe form of obesity [20].

Were searched for correlations between inflammatory parameters peri-distribution parks clinics and radiological profile of cytokines and inflammatory whole salivary destructive to women obese and non-obese. Inflammatory parameters peri-distribution parks were less represented, and IL-6 and IL-1 β salivary integral were higher in obese than in those subjects of normal weight. Thus, obese patients present a greater risk of periimplantitis than non-obese healthy subjects. [21].

Obese patients with osseointegrate implants must follow a strict program of oral hygiene to prevent inflammation and to maintain optimal health of peri-periimplant tissue [21].

Obesity has reached epidemic proportions worldwide. In a population with a stable adult maintenance therapy to periodontal health ,Amarjothi Elangovan, *et all*. for the first time showed a modest but statistically significant correlation between the abdominal circumference, a well-known parameter for evaluation of obesity and the expression of IL-1 β from peri-implant sulcular fluid [22].

A considerable number of studies [23-35] have watched this or not to interrelating periodontal disease and systemic disease, some of them having results that help us to a better menagement of patients with such diseases. (Table 1)

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