Experimental gingivitis in overweight subjects
Clinical and Microbiological study

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ABSTRACT

Background: Obesity is a complex multifactorial chronic disease that affecting the host immunity which may stimulate a hyperinflammatory response in periodontal disease. The aim of the study is to determine and compare the microbiological findings between Obese, pre-obese, and normal weight. Along with estimating the effect of overweight on healing process of experimental gingivitis, also to determine and compare the microbiological findings between Obese, pre-obese, and normal weight.

Materials and Methods: Our study dealing with experimental gingivitis using the clinical parameters (plaque index and gingival index), existent bacterial flora, oral hygiene improvement on normal weight, pre-obese, and obese subjects showing healthy systemic condition, using the body mass index (BMI) and inter parametric comparison on 30 individuals, male with no previous medical history, age ranged from 20-30 years old, 10 of them are obese, 10 are within overweight (pre-obese) and the other 10 are within normal weight. Dental plaque samples were taken from each subject when the gingiva reaches inflammation (Mean GI3).

Results: The G.I parameter showed a significant higher score of inflammation on the pre-obese and obese samples during the initiation of the disease (P≤0.002), and also showed a prolonged improvement response after the recovery of the oral hygiene control (P≤0.001). The bacterial findings showed an equal percentage of streptococci and staphylococci but with predominance on other species in normal weight subjects. The pre-obese showed an increase percent of klebsiella. The obese subjects showed insignificant differences and almost equal percent of streptococci, staphylococci and pseudomonades with undetectable number of klebsiella.

Conclusion: This study found that Obesity is an active risk factor for gingival and periodontal disease, and it’s playing a role in elevating body response to dental plaque with increasing the healing period.

Key words: Obesity, experimental gingivitis, microbiology, healing period. (J Bagh Coll Dentistry 2011;23(3): 88-91).

INTRODUCTION

Periodontal disease is a chronic disease of the oral cavity comprising a group of inflammatory conditions affecting the supporting structures of the dentition (1).

The impact of dental plaque biofilms on the etiology of periodontal diseases has been studied in detail. However, it is the paradoxical impact of the susceptible host's inflammatory response to the microbial challenge that ultimately leads to the destruction of periodontal tissues and subsequent tooth loss (2).

Periodontal diseases represent chronic inflammatory responses to a bacterial challenge. Although bacterial biofilms have been shown to be necessary in the initiation of gingival inflammation and subsequent destruction of periodontal tissues (3), its presence alone explains a relatively small proportion (i.e. 20–30%) of the variance in disease expression (4). Based on an established model of pathogenesis (5), the bacterial biofilm alone is insufficient to explain disease initiation and progression. Evidence suggests that periodontal tissue destruction is mainly due to the host’s inflammatory response to the bacterial challenge (5).

Obesity is the fastest growing health-related problem in the world (6).

A common metabolic and nutritional disorder, obesity is a complex multifactorial chronic disease that develops from an interaction of genotype and the environment (7).

The impact of obesity on health status has the same outcome as twenty years of aging, and has been indicated to exceed the impact of smoking or alcohol abuse (8).

The first paper on the relationship between obesity and periodontal disease was appeared in 1977 on rats, which are more likely to have periodontal disease deteriorations (9).

In humans, firstly on Japanese adult in 1998, (10) a higher BMI was related to a greater prevalence of periodontal disease in 241 apparently healthy adult age 20-59 years, using BMI and body fat to assess obesity and the community periodontal index (CPI). Subjects with BMI >30 compared to subjects with a BMI <20 body fat was analyzed using dual – energy X-ray absorptionometry found a 5% increase in body fat correspond to a 30% increased risk of periodontal disease in patients having normal level fasting blood glucose and cholesterol (11/12).

It has been suggested that obesity contributes to an overall systemic inflammatory state through its effect on metabolic and immune parameters,
thereby increasing susceptibility to periodontal disease (13,14).

In recent years, the evidence linking obesity to increased incidence and severity of periodontal disease has grown (15).

In general, data indicate that increased body mass index, waist circumference (abdominal obesity), serum lipid levels and percentage of subcutaneous body fat are associated with increased risk for periodontitis. After adjusting for confounding factors such as smoking, age and systemic conditions, the risk association appears to be linear. For instance, more bleeding on probing, deeper periodontal pockets and more bone loss were noticed in individuals with higher indicators of obesity (16).

The most recent study provides what is perhaps the most compelling evidence to date for a significant association between obesity and increased prevalence, severity and extent of periodontal disease (17). Therefore, it was decided to conduct this study.

MATERIAL AND METHODS

Our study dealing with experimental gingivitis uses the clinical parameters (plaque index and gingival index), existent bacterial flora, oral hygiene improvement on normal weight, pre-obese, and obese subjects showing healthy systemic condition, using the body mass index (BMI) and inter-parametric comparison.

The sample consist of 30 individuals, male with no previous medical history, no diabetes, non-smoker, with age ranged from 20-30 years old.

10 of them are obese, 10 are within overweight (pre-obese) and the other 10 are within normal weight.

The gingival parameters were indexed on the case sheet form which was filled for each subject of the three groups, dental plaque samples were taken from each subject when the gingiva reaches inflammation (Mean GI ≥ 1).

RESULTS

Statistical analysis comparing between the GI of the three groups (normal weight, pre-obese, and obese) in the 1st week of experimental gingivitis using fisher test revealed a significant difference (P ≤ 0.000).

The same statistical analysis comparing between the GI of the three groups in the 2nd week of experimental gingivitis using fisher test revealed a significant difference (P ≤ 0.002).

Also one week after oral hygiene re-establishment a statistical analysis comparing between the GI of the three groups using fisher test revealed a significant difference (P ≤ 0.000)(Table-2).

DISCUSSION

Our study dealing with experimental gingivitis using the clinical parameters (plaque index and gingival index), existent bacterial flora, oral hygiene improvement on normal weight, pre-obese, and obese subjects showing healthy systemic condition, using the body mass index (BMI) and inter-parametric comparison. We found a significant difference obtained between normal weight subjects (BMI 21.08) and those of pre-obese (BMI 27.085) and obese subjects (BMI 32.384) of their plaque index in the 1st week of the experiment which signifies that pre and obese having the ability to develop plaque accumulation easier and faster than normal subjects (P ≤ 0.007) this is because the dietary habits and increasing in the frequency of food eaten in addition to the type of food which’s more sticky and containing more sugar that affects the formation of dental plaque. When the gingivitis is created the all three groups showing an almost equal amount plaque accumulation (non significant differences) (P ≤ 0.204).

That’s mean the length of the period after dental plaque formed will not affect the amount.

The GI parameter showed a higher significant score of inflammation on the pre-obese and obese samples during the initiation of the disease (P ≤ 0.000) especially after the 1st week of the beginning of the experiment, that’s mean the overweight subjects will develop gingivitis more faster than normal weight.

Almost all the obese subjects were developed mild form of gingivitis after 7 days of the experiment, this result was disagreed with L e et al in 1965, in their experiment all the subjects did not develop gingivitis in the first week, this may be due to the effect of obesity on the inflammatory response leading to increasing the levels of some cytokines like: TNF-α, IL-1, IL-6, and IL-8, all these cytokines will intensify the inflammation in addition to the thickening of the
blood vessels and increase the level of PAI-1, all these effects will accelerate gingivitis.

These results were agreed with wood N. studies in 2003 that indicates that there is increase in bleeding tendency of gingiva in relation to the BMI.

During the 2nd week of the experiment the differences remain significant between the three groups (Normal weight, Pre-obese, and Obese), this mean that in spite of the development of gingivitis in all the subjects in the three groups but the obese and pre-obese having higher GI than normal weight ($p \leq 0.002$), this mean that the overweight will not affect the initiation of gingivitis only but also has an effect on the progression of the disease.

Overweight subjects showed a prolong improvement response after the recovery of the oral hygiene control ($p \leq 0.001$), after one week of oral hygiene re-establishment the overweight subjects returns to the normal state gingiva but within higher limits, this result could suggest a negative interference of the obesity on the gingival healing that’s maybe due to the injurious effect of some adipokines, Bazari et al in 2007 suggests that obesity causing poor wound healing and slowing the healing process.

A significant positive correlation of GI with BMI, and PLI with BMI could suggest that overweight increasing the risk to initiate gingivitis and could interfere in the course of the disease progression.

The bacterial finding showed an equal percent of streptococci and staphylococci but with predominance on other species in normal weight subjects. The pre-obese showed an increase percent of klebsiella and undetectable number of pseudomonades. The obese subjects showed insignificant differences and almost equal percent of streptococci, staphylococci and pseudomonades with undetectable number of klebsiela.

These differences in bacterial species and presence or absence of species suggest that there are differences in the dental plaque composition, maybe due to changes in food habits between normal weight and overweight.

REFERENCES

Table 1: Fisher test of the PLI for the 3 groups during the experiment

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<tr>
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<th>Fisher F-value</th>
<th>P-value</th>
<th>S/NS</th>
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<td>in the 1st week</td>
<td>5.944</td>
<td>0.007</td>
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<tr>
<td>in the 2nd week</td>
<td>1.685</td>
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<td>one week after oral hygiene re-establishment</td>
<td>1.293</td>
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Table 2: Fisher test of the GI for the 3 groups during the experiment

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<tr>
<td>2nd week</td>
<td>8.111</td>
<td>0.002</td>
<td>S</td>
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<td>one week after oral hygiene re-establishment</td>
<td>11.019</td>
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Table 3: Coefficient of correlation for 3 groups

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<td>Normal weight</td>
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