A cross-sectional tri-level study of the obesity effects on the salivary uric acid and total protein of gingivitis Iraqi subjects

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ABSTRACT

Background: Obesity is the fastest growing health-related problem in the world. It plays an affecting role in the biochemistry of many serious systemic diseases like diabetes and CVD. Periodontitis appeared to have a reciprocal relationship with both, obesity on one hand side and the serious systemic diseases on other hand. The aim of study: is to investigate the inter linking between obesity and gingivitis by screening the salivary uric acid and total protein.

Material and method: Control healthy periodontal and systemically-normal weight,27 male, (CG), gingivitis–normal weight, systemically healthy(GN) 16 male, Gingivitis-overweight-systemically healthy(GOV) 14 male).and Gingivitis-obese-systemically healthy(GO)12 male, aged 30-40 y, no smokers, Their weight measured according to BMI. Clinical data were recorded according to PL.I, GI, BOP, at the same hour of unstimulated saliva collection.

Results: Obese subjects showed increased PL.I, G.I, BOP scores and high significant increase of salivary total protein. BOP; showed significant increase in bleeding sites as the Body Mass Index goes up. Uric acid showed, always, negative correlation with the totality of gingival inflammation parameters, they were significant only with normal weight subjects.

Conclusion: Total protein. Appeared significantly positive correlated to gingivitis more than to the obesity. While Uric acid correlated insignificantly negative with the gingivitis but not to the increased body weight.The increased body weight also positively correlated to the gingival inflammation, these results could say that gingivitis modifies the salivary chemicals while the obesity enhances gingivitis.

Key words: Periodontal parameters, salivary flow, total protein, uric acid, gingivitis. (J Bagh Coll Dentistry 2012; 24(4):67-70).

INTRODUCTION

Obesity, defined as a body mass index (BMI) >30.0 kg/m^2, is a major public health problem today. The prevalence of obesity has increased substantially over the past decades in most industrialized countries. Obesity is a risk factor for several chronic diseases, most notably hypertension, type 2 diabetes, dyslipidemia and coronary heart disease.(1)Since adiposity can be considered a systemic disease that predisposes to a variety of co-morbidities and complications that affect overall health. Further, recent studies have suggested that obesity is also associated with oral diseases, particularly periodontitis.(2) In fact, the adipose tissue secretes several cytokines and hormones that are involved in inflammatory processes, suggesting that similar pathways are involved in the patho-physiology of obesity and periodontitis(3)

Obesity is characterized by the abnormal or excessive deposition of fat in the adipose tissueIts consequences go far beyond adverse metabolic effects on health, causing an increase in oxidative stress (4) which leads not only to endothelial dysfunction but also to negative effects in relation to gingivitis, and then to periodontitis, because of the increase in pro-inflammatory cytokines.(5) Thus obesity appears to participate in the multi-factorial phenomenon of causality of gingival and periodontal disease through the increased production of reactive oxygen species, However, the adipose tissue actively secretes a variety of cytokines and hormones that are involved in inflammatory processes, pointing toward similar pathways involved in the patho-physiology of obesity, periodontitis and related inflammatory diseases.(6)

The aim of this article is to get an overview of the association between obesity and gingivitis through the salivary uric acid and total protein values as an indicators.

MATERIAL AND METHODS

Sixty-nine volunteers, male, aged 30-40 years old, nonsmokers have been involved in this study. Females have been excluded to avoid hormone interferences. twenty –seven of them have been chosen as control group (CG)), whose showed healthy conditions; systemically as proved clinically and laboratory, within the healthy range of normal weight according to BMI index, and free of classic clinical signs of gingivitis according to the routine diagnostic parameters (PL.I, G.I, BOP.).Other 42 subject showed healthy systemic condition, variable body weight, variable gingivalesion, so, subdivided into three

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study groups based on their body weight; G1, sixteen subject of normal weight, 'GN'(BMI=18.5-24,99). G2, fourteen subjects of overweight, 'GOV (BMI=25-29.99).G3, twelve obese subjects, 'GO'(BMI= ≥30).

Routine clinical periodontal-gingival parameters have been used for diagnosis and data records that beenapplied in this study were; plaque index (PL.I).gingival index of loe and silness [7] (G.I), bleeding on probing (BOP).

Unstimulated salivary flow samples (5 ml) have been collected at 9-12 AM, after careful mouth rinse with potable water. Subjects have to be fasting at least one hour before collection. Timing the collection process was 5-10 minutes, collected samples were centrifuged (4000^{RPM}) for 10 minutes, freeze at $-20^{°C}$. Proteins were analyzed as react in acidic medium with Perogallol Red to be modulated to form a colored complex. The intensity of color is proportional to the protein concentration, read on spectrophotometer at 598 ^{mm}. (Reagent suppliers: Spinreact.co/ Spain).

Uric acid has been oxidized by Uricaseto allentoine and hydrogen peroxide, when 1mlof reconstituted react with 20 ^{ul} of prepared saliva then incubated at $37^{°C}$ for 5 minutes. Oxidation read on spectrophotometer at 520 ^{mm}. (Reagent supplier: Biomagreb.co/ Tunisia)

Results were analyzed statistically with student-t test, F-fisher test, chi- square, and spearman coefficient of correlation.

RESULTS

The control group (CG) showed in-equivalent values of uric acid and total protein to that of international standard, that were ranged for uric acid 3.5-7.2$^{mg/dl}$, and that of T. protein as 6.0-8.3$^{mg/dl}$ (Table 1). Gingivitis group showed a higher significant GI, PL.I, to the control group.

The obese subjects (GO) showed a high significant PLI as well as G.I. to the normal weight group (GN). The statistical compare between overweight (GOV) and obese (GO) showed insignificant increase in PL.I value, as well as of G.I. Identical results also obtained between normal weight (GN') and overweight (GOV') in their PL.I., while G.I showed a slight significant increase in its value in favor to GOV. (Table 2).

Bleeding on probing also showed significant increased percent in bleeding sites as the BMI goes up from normal to obese, while the overweight –obese showed insignificant increase in percent of bleeding sites (table2).

Salivary bio-chemicals showed insignificant variations of uric acid and total protein among the gingivitis groups. While when compared to the control (CO),only the total protein showing high significant increase with the whole three groups of gingivitis (Table 3,4).

The total protein showed significant positive correlation with PL.I and BOP of GN and showed high significances of positively correlated with PL.I, G.I. and BOP. Of GOV.While the GO showed positive significant correlations between T. proteinand PL.I, G.I, while theBOP appeared insignificantly positive.

Uric acid showed Negative significant correlation with PL.I, G.I, and BOP in gingivitis cases of Normal weight (GN) but insignificant Negative correlations with same parameters as the body weight goes up (overweight or obese.).

BMI of control group as well as of the three groups of gingivitis showed negative insignificant correlations with uric acid and positive insignificant correlations with total proteins. (Table 5, 6)

DISCUSSION

It has been approved that obesity is second only to smoking as the strongest risk factor for inflammatory periodontal tissue destruction [8] when Perlstein et al [9] observed histo-pathologic changes in the periodontium in hereditary obese Zucker rats. it seemed that under healthy oral conditions, obesity does not promote pathologic periodontal alterations; however, in response to bacterial plaque accumulation, periodontal inflammation and destruction were more severe in obese animals [10]

In 1998, Saito et al [11] analyzed 241 healthy Japanese individuals and showed an association between obesity and periodontal disease in humans. In addition, studies have indicated that the fat distribution pattern plays a crucial role in the association with periodontitis.[12]Another recent study by Saito et al.[13] concluded that obesity is associated with deep periodontal pockets, independent of glucose tolerance status. Genco et al [14] analyzed National Health and Nutrition Examination Survey (NHANES III) data and demonstrated that BMI was positively correlated with the severity of periodontal attachment loss; they found that this relationship is modulated by insulin resistance [15]

However, adipose-tissue–derived cytokines and hormones may play a key role. Fat tissue is not merely a passive triglyceride reservoir of the body, but also produces a vast amount of cytokines and hormones, collectively called adipokines or adipocytokines, which in turn may modulate periodontitis. Indeed, the majority of the recent international studies have been conducted

on the chronic periodontitis, very rare, particularly on humans, were studied the relationship of obesity and gingivitis. This may be due to the reversibility of gingivitis facing the irreversible damages on periodontitis conditions making the follow-up of cases uncertain.

A cross-sectional tri-level study has been conducted to investigate the connection among the obesity as a systemic risk factor, with gingivitis on one side and as a salivary chemicals modifying factor. The reality of being the obesity as a conductive risk factor for many serious systemic diseases and to periodontal disease as well now is clear enough [17]. An increasing prevalence of obesity is well documented in all ages and ethnicities worldwide [18]. Obesity commonly accompanied by elements of the metabolic syndrome, including insulin resistance, hypertension, and dyslipidemia, is associated with increased risk of chronic inflammatory diseases such as periodontitis [19]. Both periodontal disease and obesity are of multifactor etiology related to dietary habits but also closely correlated with socio-demographic background of the individuals. [20] Most of the studies regarding association between obesity and periodontal disease are based on clinical data [21,22]

The increased amount of adipokines from visceral fat may induce agglutination of blood in the microvasculature, decreasing blood flow to the Gingiva in obese people and facilitating the progression of gingival disease [23]. Obese subjects in this study showed increased PL.I, G.I and BOP scores.Also showed a high significant increase of salivary Total protein. This chemical, the total protein, appeared significantly correlated to gingivitis more than to the obesity. Bleeding on probing showed significant increase in bleeding sites as the BMI goes up from normal to obese. Uric acid showed, always, negative correlation with the totality of gingival inflammation parameters, but they were significant only with normal weight subjects. This result suggests that salivary uric acid may be correlated with the gingival inflammation not to the increased body weight, in addition our results could suggest that increased body weight also positively correlated to the gingival inflammation, this results could say that gingivitis modifies the salivary chemicals while the obesity enhance gingivitis.

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Table 1: Mean ± SE records of control group (CG), gingivitis groups, GN, GOV and GO

Groups	PL.I	G.I	BOP	SFR	T. Protein	U. Acid
CG	0.021±0.001	0.121±0.002	0%	0.34±0.0141	35.791±1.044	1.986±0.066
GN	0.817±0.075	0.74±0.06	5.5%	0.331±0.017	66.44±2.471	1.829±0.097
GOV	1.09±0.012	1.025±0.096	15%	0.368±0.018	71.47±2.747	1.757±0.098
GO	1.201±0.01	1.055±0.093	16.3%	0.422±0.019	76.61±3.339	1.571±0.011

Table 2: Significances Records.

	PL.I P. Value	Sign.	G.I P. Value	Sign.	BOP Chi square	Sign.
GN<>GOV	0.057	NS	0.0164	S	92.3	HS
GN<>GO	0.0065	HS	0.00778	HS	99.3	HS
GOV<>GO	0.494	NS	0.823	NS	0.222	NS

Table 3: Salivary chemicals' T. protein and U. acid.

	U. Acid Mg/dl		T. Protein Mg/dl	
	Mean ± SE	**SD**	**Mean ± SE**	**SD**
CG	1.986 ± 0.066	0.345	35.791 ± 1.044	5.427
GN	2.1 ± 0.058	0.247	61.87 ± 2.602	11.039
GOV	2.149 ± 0.059	0.222	66.163 ± 3.395	12.703
GO	2.081 ± 0.068	0.235	61.943 ± 3.420	11.848

Table 4: Salivary biochemical's statistics.

	U. Acid Mg/dl		T. Protein Mg/dl	
	P value	**Sign.**	**P value**	**Sign.**
CG <> GN	0.365	NS	0.0027	HS
CG<> GOV	0.116	NS	0.0031	HS
CG <> GO	0.390	NS	0.0016	HS
GN<> GOV	0.524	NS	0.315	NS
GN <> GO	0.877	NS	0.987	NS
GOV <> GO	0.453	NS	0.392	NS

Table 5: Correlations statistics gingival parameters vie saliva chemicals

		PL.I		G.I.		B.O.P.	
		Value	**Sign.**	**Value**	**Sign.**	**Value**	**Sign.**
GN	U. Acid	0.55	- ve S	0.47	- ve S	0.672	- ve HS
	T. Protein	0.58	+ ve S	0.147	+ ve NS	0.563	+ ve S
GOV	U. Acid	0.257	- ve NS	0.234	- ve NS	0.124	- ve NS
	T. Protein	0.784	+ ve HS	0.807	+ ve HS	0.822	+ ve HS
GO	U. Acid	0.0035	- ve NS	0.049	- ve NS	0.047	- ve NS
	T. Protein	0.776	+ ve HS	0.652	+ ve S	0.452	+ ve NS

Table 6: Correlation statistics, BMI vie saliva chemicals

	BMI	U. Acid			T. Protein		
	Mean	**Mean**	**P. value**	**Sign.**	**Mean**	**P. value**	**Sign.**
CG	18.5 ± 0.334	1.986 ± 0.066	1.987	- ve NS	35.79 ± 1.04	0.992	+NS
GN	23.153 ± 0.832	2.1 ± 0.0581	1.092	-ve NS	61.87 ± 2.601	1.381	+NS
GOV	27.905 ± 1.462	2.149 ± 0.059	2.190	-ve NS	66.163 ± 3.395	1.098	+NS
GO	35.09 ± 3.871	2.081 ± 0.0677	2.053	-ve NS	61.943 ± 3.42	2.539	+ NS