Experimental gingivitis on Iraqi over weight adult male: A clinical and microbiological study

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ABSTRACT

Background: The aim of this study was to investigate the influence of over weight on the initiation of gingival inflammation compared to normal weight subjects on mankind.

Material and methods: Thirty six adult male, aged 20-30 years, systemically proved as healthy, sub-grouped in three, twelve in each, as normal weight, pre-obese and obese according to BMI index. All have been subjected to a vigorous oral hygiene control up to getting as low as possible full mouth Plaque and Gingival scores (PL.I= less than 0.3 and G.I. less than 0.2).taken as Base line records. Subjects have been advised to continue their usual life-style, withdrawing the daily brushing, and to be subjected to a weekly oral examination up to getting a clinically visible gingivitis, then re-establishing the oral hygiene to watch the improvement of gingival lesion under the influence of over weight.

Results: Showed a significant increase of PL.I for the three groups in first week. A positive significant difference in favor of obese and Pre-obese, But it was insignificant between Pre-obese and Obese. Identical results were obtained in G.I. scores. Bacteriologic findings showed significant increase of pseudomonades species in obese group.

Conclusions: Results suggested great influences of Over-weight on plaque accumulation and thus facilitate the virulent bacterial invasion and disease acceleration, daily oral hygiene control is a successful preventive measures regardless the body weight

Key Wards: BMI, PL.I, G.I. Obesity, Gingivitis. (J Bagh Coll Dentistry 2011;23(4):101-106).

INTRODUCTION

The plaque induced periodontits is an evolution of pre-existed gingivitis, this condition could stay years without progression to involve the deeper structures of periodentium unless aggravated by a risk factor [1]. Obesity has been highlighted as a predictor of many serious systemic disorders [2], like Diabetes, hyperlipidimia, Hypertension, cholelithiasis, arteriosclerosis, cardiovascular, and cerebrovascular [3]. The imbalance of the host immune system commonly detected among the periodontits subjects as well as among the over-weighted subjects could explain the observed association of obesity with certain diseases including periodontitis [4,5].Visa-versa, recent studies have shown that periodontal disease could exert an impact on systemic health such to modify the blood chemistry [6-8] with a raise in inflammatory mediators[9] which at its role affect the metabolic of glucose and lipids, thus increasing the risk of diabetes and/or cardiovascular disorder [10]. In last decade, the prevalence of obesity is increased, becoming worldwide, same as periodontitis [11,12]. Environmental, socio-behavioral influences, genetics, and metabolic syndromes are likely the most important risk factors for obesity [13-16].

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Genetic factors have been suggested to be contributed in many systemic disorders including obese ability and aggressive periodontitis [17,18]. Hundreds of recent studies have been cited specking about the relation ship of overweight and already clinically existed periodontits as secondarily to metabolic syndrome diseases or via immune deterioration [9, 19]. But what about the start point of the disease, the gingivitis..? The precedent to periodontits, what is the influence of obesity on the initiation of the disease..?

The experimental gingivitis has mainly been experimented on animals, or very rarely on mankind [20, 21] without taking in any consideration the body weight in all experimental trials [22, 23].

The aim of this study is to investigate the influence of overweight on gingivitis initiation on humans in Iraq.

MATERIALS AND METHODS

Subject selection: Traditional life styled Iraqi adult male were taken, Thirty six volunteers, aged 20-30 years, systemically have been examined by the legal state hospital of Al-Yarmook (sugar level, H.D.L, L.D.L, and Triglyceride). Those had been suspected unhealthy were excluded. Fasting and random Blood sugar and lipids should be within the normal levels. The volunteers were sub-grouped in three according to their BMI (obese ≥30. over weight ≥ 25 and normal weight 18-24.99) [24,25

Clinical examinations: Full mouth scaling with ultra sonic devise have been practiced for all subjects, demonstrating BASS technique, inter dental brush and flossing, advising them to brush and floss the teeth after each meal for several weeks with weekly follow up and clinical checking up to get as low as possible and stable PL.I [26] and G.I. [27], this record was taken as a base line records. The volunteers were advised to withdraw the brushing, continuing their habitual life stile, and to be attended weekly to record the readings.

Body Mass and Weight assessments: The BMI used is a gender and age independent, defined as weight in Kilogram divided by the square of the Height in meters, scaled as normal weighted 18.5-24.99. Over-weighted ≥ 25 (25-29.99) and obese ≥ 30.

Dental plaque samples: As the gingivitis appeared clinically, a dental plaque smears were taken after rinsing the mouth several times to remove the material alba and food debris, isolating the upper anterior teeth with cotton rolls, drying the area with cotton, and by a sterilized small scalar gather gently the plaque from the apical third of the crown next to the gingival margin. The collection then transferred to a sterilized test tube containing phosphate buffer saline to be sent to the laboratory within 2-3 hours. Cultivation and Incubation applied as usual protocols using blood Agar one for aerobic, other for anaerobic (BD, Diagnostic) McConky Agar (BD, Diagnostic), one for aerobic and second for anaerobic, Chocolate Agar, Sabauroud Agar (BD, Diagnostic). Gram stain and colonial reading were recorded.

Statistical rules: T- test and Chi-square test have been used to realize the results.

RESULTS

The statistical analyses of base line records showed insignificant variations among the three groups in PL.I and G.I. readings. (table1, 2, 3) BMI records showed high significant differences among the three groups as compared with each others (Table 4and 5). All three groups showed a significant increase of PL.I. and G.I. scores as compared to the base line records in the first and second weeks. These records became insignificant in the third week (Tables 1, 2, 3).

As the gingivitis evolutes, the PL.I and G.I. showed a progressive significant increase in their values in the obese group, while the pre-obese and the normal weight as well showed insignificant increase of plaque accumulation between first and second weeks with significant gingival index evolution from first up to the second week (Tables1 and 2).

Plaque accumulation (PL.I) and inflammation evolution (G.I), showed a significant progression from base line records as healthy gingiva up to inflamed gingiva in pre-obese and obese groups, with a successful regression to the normal healthy state in third week when the oral hygiene have been established (Table 6 and 7). The records of normal weight group showed that the evolution of plaque accumulation and gingival index were insignificants from base line up to second week (Table 8) inversely it showed a significant evolution from second to third week and showed a significant regression to the base line records with oral hygiene control insignificant variations between third week and base line records (Table 8 and 9).

Gingival index showed a significant increase in their values in obese and pre-obese peoples than normal weight peoples, plaque accumulation was heavier in the obese group at the end of second week (Table 9)

A positive significant correlation have been found between obesity and gingival inflammation and plaque accumulation, while the correlation was only positively significant in Plaque reading in pre-obese peoples (Table 10).

The bacterial content of normal weight plaque was 45% streptococcus species, 45% staphylococcus species, 5% klebsiella, and 5% pseudomonades.

In the pre-obese group the percentage of bacteria present was 41% streptococcus species, 42% staphylococcus, and 17% klebsiella.

While for the obese the percentage of bacteria present was 34% streptococcus, 33% staphylococcus, and 33% pseudomonades (Table 11).

DISCUSSION

Being overweight or obese is important risk factor for several adult diseases including diabetes type 2, hyperlipimia, hypertension, cholelithiasis, arteriosclerosis, cardiovascular, and cerebrovascular disease [28-30].

In fact, periodontal disease was more prevalent in subjects with a serum high density lipoprotein cholesterol concentration< 60mg/dl suggesting that periodontal disease is exacerbated by the metabolic syndrome associated with obesity [9, 31-32]. Buhlin et al [1] had reported that a high BMI (≥26 in men and ≥25 in women) is significantly correlated with high collestrolimia and periodontits in multivariate model of a case-control study. Merchand etal. 2003, [33], concluded that decreasing the serum lipid by increasing the

physical activity could lead to decrease the periodontal risk in men.

Of a different types of obesity (waist-to-hip or upper body) could react with periodontal disease differently, upper body obesity (UBO) is related to visceral fat accumulation, increase the risk of various adult disease especially type 2 diabetes [34, 35].

UBO was also reported to have a significant relationship with periodontal disease [36]. Multivariate analysis using known risk factor of periodontal disease showed a significant relationship between BMI and periodontal disease only in subjects with high waist-to-hip ratio [37]. Similar studies results were obtained using Body Fat Index instead of BMI [38].These findings suggest that visceral fat accumulation in upper body obesity is associated with periodontal disease [39].Most recent studies were reported that BMI and waist-to-hip ratio were associated with various periodontal parameters, such as attachment level, pocket depth, gingival bleeding and calculus index [40].

Alzahrani et. al. [41]), reported that BMI and waist circumference were associated with periodontal disease for both attachment loss(≥ 3mm) and probing pocket depth(≥4mm) especially in the subjects of young adult 18-34 years old. Aging is associated with an increase in body fat mass [42], this mean that some older obese subjects gain weight as part of aging process [43,44], on other hand, old age and obese showing two risk factors at the same time, both significantly associated with aggravation of priory existed or directing the initiated periodontal disease [16, 41, 43].

Periodontits is a bacterial invasion, started as a gingivitis due to bacterial plaque accumulation of several days, this gingivitis could stay years in different inflammation form involving only the gingival unit apparatus, or in some susceptible individuals, could progress into more advanced lesion to involve more deeper periodontal unit apparatus to appear clinically as a bone and soft tissue destructive disease with attachment loss and pocket formation [2]. This transgression to the periodontal support needs an additional factor more than the bacterial virulence, that is the risk of a systemic factor included the immune responses and host defense deteriorations [4, 5, 7, 8, 45].

The totality of the studies since 1977 on animals or mankind up to date proved a significant correlation between obesity and periodontits through the systemic disorder due to the increase serum lipids and serum glucose levels via the metabolic syndrome [13, 15, 16, 30, 31, 32]. Indeed, according to our knowledge, we could not find in the recent literature any study deal with obesity and plaque induced gingivitis either as usual finding or experimentally developed. In fact, the few experimental gingivitis studies on human or on animals have never take in consideration the body weight that prevented us to deal with their results [19-23].

Our study dealing with experimental gingivitis on adult mankind uses the clinical parameters (plaque index and gingival index), existent bacterial flora, and oral hygiene improvement on normal weight, pre-obese and obese subjects showing healthy systemic condition, using the body mass index (BMI) and inters parametric comparisons. We found a significant differences obtained between normal weight subjects (BMI ≥21.085), pre-obese (BMI ≥27.085) and the obese (BMI ≥ 32.385) in favor of the over weighted in their Plaque index which signifying that over weighted subjects could have the ability to develop plaque accumulation easier and faster than normal weighted subjects. The gingival index showed a significant higher scores of inflammation on the pre-obese and obese subjects during the initiation of the disease and also showed a significant evolution with time during three weeks as showed a positive reflex in returnee to the normal healthy state after the oral hygiene have been controlled.

The bacterial finding showed an equal percent of Streptococci and Staphylococci which predominated on other species in normal weighed subjects. The pre-obese showed an increased number of Klebsiella with undetectable numbers of Pseudomonades, Obese showed equal predominant percent of Streptococci, Staphylococci and pseudomonades, these species have showed a significant variations of percent as the body weight increase.

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Table 1: obese group

	Base line		1st week		2nd week		3rd week	
	PL	GI	PL	GI	PL	GI	PL	GI
Mean ±SE	0.441±0.004	0.167±0.005	1.1593±0.03	1.129±0.041	1.619±0.009	1.361±0.018	0.307±0.017	0.183±0.005
SD	0.197	0.034	0.2147	0.127	0.289	0.182	0.084	0.06

Table 2: Pre-obese group

	Base line		1st week		2nd week		3rd week	
	PL	GI	PL	GI	PL	GI	PL	GI
Mean ±SE	0.513±0.005	0.181±0.001	1.239±0.031	0.963±0.009	1.595±0.032	1.33±0.041	0.413±0.004	0.28±0.008
SD	0.207	0.083	0.3883	0.22	0.374	0.152	0.167	0.0625

Table 3: Normal group

	Base line		1st week		2nd week		3rd week	
	PL	GI	PL	GI	PL	GI	PL	GI
Mean ±SE	0.462±0.002	0.11±o.oo1	1.076±0.004	0.411±0.006	1.377±0.018	1.023±0.05	0.373±0.016	0.1356±0.005
SD	0.241	0.054	0.1817	0.266	0.398	0.239	0.209	0.0925

Table 4: BMI

	Obese group	Pre-obese group	Normal group
Mean and SE	32.31± 2.3	26.99± 1.9	21.056±1.27
SD	2.934	1.037	1.628

Table 5: t-test between groups OF BMI

BMI	t-test	P-value	Sig
Obese & pre-obese	5.612	P<0.01	HS
Obese & Normal	10.451	P<0.01	HS
Pre- & Normal	8.823	P<0.01	HS

Table 6: t-test between obese group

	PL		GI	
	t-test	P-value	t-test	P-value
Base line&1st week	9.692	P<0.01	28.567	P<0.01
Base line &2nd week	10.598	P<0.01	25.281	P<0.01
Base line & 3rd week	2.523	0.028	0.819	0.430
1st & 2nd week	5.564	P<0.01	4.355	P<0.001
1st & 3r week	13.954	P<0.01	28.554	P<0.01
2nd &3rd week	15.791	P<0.01	22.521	P<0.01

***P<0.01 High significant, **P<0.05 Significant, ***P>0.05 Non significant**

Table 7: t-test between Pre-obese group

	PL		GI	
	t-test	P-value	t-test	P-value
Base line&1st week	7.464	P<0.01	12.078	P<0.01
Base line &2nd week	10.04	P<0.01	21.644	P<0.01
Base line & 3rd week	3.839	0.003	2.740	0.019
1st & 2nd week	3.653	0.004	9.743	P<0.01
1st & 3rd week	8.530	P<0.01	10.165	P<0.01
2nd &3rd week	11.856	P<0.01	24.022	P<0.01

***P<0.01 High significant, **P<0.05 Significant, ***P>0.05 Non significant**

Table 8: t-test between Normal group

	PL		GI	
	t-test	P-value	t-test	P-value
Base line&1^{s}week	9.873	P<0.01	4.522	P<0.001
Base line &2ndweek	5.815	P<0.01	15.102	P<0.01
Base line & 3rdweek	1.369	0.198	0.922	0.376
1st & 2ndweek	2.463	0.030	0.143	0.339
1st & 3rdweek	9.714	P<0.01	3.422	P<0.01
2nd &3rd week	7.020	P<0.01	14.226	P<0.01

***P<0.01 High significant, **P<0.05 Significant, ***P>0.05 Non significant**

Table 9: t-test between groups of PL, GI

		Base line		1st week		2nd week		3rd week	
		PL	GI	PL	GI	PL	GI	PL	GI
Obese & pre-obese	t-test	0.989	0.657	0.611	2.427	0.183	0.468	3.479	3.679
	p-value	0.344	0.514	0.544	P<0.01 S	0.858	0.649	P<0.01 S	P<0.01 S
Obese & Normal	t-test	0.190	2.606	1.790	8.514	1.778	3.623	0.886	2.901
	p-value	0.853	P<0.01 S	0.101	P<0.001 HS	0.103	P<0.01 S	0.394	P<0.01 S
Pre- & Normal	t-test	0.522	2.095	1.860	4.995	1.379	3.633	0.484	4.750
	p-value	0.612	P<0.01 S	0.090	P<0.01 HS	0.195	P<0.01 S	0.638	P<0.01

P<0.05 Significant, **P>0.05 Non significant

Table 10: Correlation (r) between BMI with PL, GI

r	Base line		1st week		2nd week		3rd week	
	PL	GI	PL	GI	PL	GI	PL	GI
Obese	0.20	0.156	0.510	0.714	0.678	0.521	0.171	0.514
p-value	0.532	0.628	P<0.01 S	P<0.01 S	P<0.01 S	P<0.01 S	0.596	P<0.01 S
Pre-obese	0.837	0.537	0.413	0.409	0.280	0.315	0.852	0.222
p-value	0.654	0.985	0.182	0.191	0.379	0.319	P<0.001 HS	0.487
Normal weight	0.272	0.301	0.380	0.130	0.071	0.122	0.150	0.055
p-value	0.392	0.342	0.223	0.688	0.826	0.707	0.641	0.866

Table 11: chi-square test showing the significant variations among bacterial species

	Chi square	df	p-value	
Normal & pre-obese	11.83	3	0.008	Significant
Normal & obese	29.01	3	0.0001	Significant
Pre-obese & obese	51.73	3	0.0001	Significant
Normal, pre-obese, and obese	74.27	6	0.0001	Significant