The relation of salivary antioxidants and lipid peroxidation biomarker to periodontal diseases among overweight and obese adult aged 55-65 year-old at Textile factory in Mosul city

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ABSTRACT

Background: Overweight and obesity might be a potential risk factor for periodontal diseases. The principle objective of this study was to identify the relationship of salivary antioxidants (vitamin E and uric acid) and lipid peroxidation biomarker (malondialdehyde/MDA) with periodontal diseases among overweight and obese adult aged 55-65 year-old subject at Textile factory in Mosul city.

Materials and methods: All subjects aged 55-65 year-old (thirty five subjects) at Textile factory in Mosul city took part in this study. Salivary flow rate was measured after collection of unstimulated saliva then salivary samples were analyzed for the measurement of salivary antioxidants (vitamin E and uric acid) and lipid peroxidation biomarker (malondialdehyde/MDA). Periodontal diseases were evaluated by using the gingival index (GI), periodontal pocket depth (PPD) and clinical attachment level (CAL). Body weight was determined by using the Body Mass Index (BMI).

Results: Malondialdehyde (MDA) level was higher among obese and overweight than non-obese with highly significant difference ($F=5.52$, $P<0.01$). Similarly vitamin E and uric acid levels were elevated among obese and overweight compared with non-obese though statistical differences were not significant ($P>0.05$). In contrast salivary flow rate was lower among obese and overweight than non-obese with highly significant difference ($F=8.11$, $P<0.01$). Regarding periodontal diseases, obese subjects showed higher periodontal destruction in comparison with non-obese.

Conclusions: Overweight and obese subjects could be considered as special group who need educational and preventive programs that include maintaining a normal body weight, eating a well-balanced diet and engaging in physical activity in addition to oral hygiene practices to improve their oral and general health.

Key words: Overweight, obesity, periodontal diseases, salivary antioxidants, malondialdehyde. (J Bagh Coll Dentistry 2012;24(1):90-95).

INTRODUCTION

Developed and developing countries are facing an obesity epidemic with various health consequences [1]. Obesity represents excessive body fat stores [2]. Overweight also defined as having more body fat than is optimally healthy but it is considered as a state of pre obesity [3]. For adult women, body fat is normally 20-25% of total body weight; for men, the range is 12-20%. Risk of chronic diseases rises dramatically when body fat exceeds 30% in women and 25% in men [4]. Obese people are at higher risk for heart diseases, diabetes mellitus, cancer, joint diseases, and psychological problems [2]. Overweight and obesity is associated with oral diseases, particularly periodontal disease [5].

Periodontal diseases are infections of multifactorial etiology; caused by overgrowth and differentiation of dental plaque bacteria [6]. While environmental and genetic influences affect disease severity [7]. *Gingivitis* is an inflammatory lesion confined to the tissues of the marginal gingiva [8]; whereas *periodontitis* results from extension of the inflammatory process initiated in the gingiva to supporting periodontal tissues [9].

It is a highly destructive and progressive inflammatory disease [10].

Several studies have addressed the relationship between obesity and periodontal diseases. Al-Zahrani *et al* [11] reported that obesity could be a potential risk factor for periodontal destruction. The same finding was also reported by other studies [12-15]. Also Zermeño-Ibarra *et al* [16] found that overweight and obese subjects showed significant periodontal alterations.

Normal salivary flow rate is essential for maintaining oral health and periodontal health in particular [17]. Also periodontal diseases involved a variety of molecular species among them reactive oxygen species (ROS). Oxidation of polyunsaturated fatty acids contained in the phospholipids portion of the cell membrane by reactive oxygen species leading to membrane destruction is termed *lipid peroxidation* [18]. The stable end product of lipid peroxidation is termed Malondialdehyde (MDA) [19]. Free radicals induced lipid peroxidation has been implicated in the pathogenesis of periodontal diseases [20,21]. Antioxidants have been shown to play a critical role in modulating reactive oxygen species-induced damage during periodontitis [22]. Since no previous Iraqi study was found concerning this subject; this study was carried out to explore the

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relationship of salivary antioxidants (vitamin E and uric acid), lipid peroxidation biomarker (malondialdehyde/MDA) and salivary flow rate with periodontal diseases among overweight and obese adult aged 55-65 year-old subject at Textile factory in Mosul city.

MATERIALS AND METHODS

Study participants were all subjects aged 55-65 year-old (Thirty five subjects:- Non-obese: 13 (37.14%), overweight: 9 (25.71%), and obese: 13 (37.14%)) at Textile factory in Mosul city. They should be non-smoker, with no medical history that compromises salivary secretory mechanism (depending on the medical report supplied by the medical unit at the factory), shouldn't take any medications with xerogenic effect or any nutritional supplementation, and shouldn't wear any fixed or removable dental prostheses. The collection of unstimulated salivary samples was performed according to the instructions cited by Tenovuo and Lagerlöf **(23).** Salivary flow rate was expressed as milliliter per minute (ml/min). Then salivary samples were taken to the laboratory for biochemical analysis at the College of Veterinary and College of Dentistry, University of Mosul.

Salivary antioxidants were determined colorimetrically by using the spectrophotometer (Cecil Instrument Limited CE 1021, England). Vitamin E was measured by manual method depending on Emmerie-Engel reaction **(24).** While uric acid level was measured by using ready kit (BioMérieux sa, France). Salivary MDA level was determined by using the method of Beng and Aust **(25)**.

Plaque index (26) (PlI) and calculus index (27) (CalI) were used for recording oral cleanliness. Periodontal disease can be evaluated by using the gingival index (28) (GI), periodontal pocket depth (9) (PPD) and clinical attachment level (27) (CAL). Body weight was measured by using the Body Mass Index (BMI) which is the ratio of body weight in Kilogram to body height in meter squared (4). BMI was divided into three categories that included non-obese (<25 Kg/m^2), Overweight (≥25-<30 Kg/m^2), and obese (≥30 Kg/m^2) (29). Data analysis was conducted through the application of the SPSS (version 12). Analysis of variance was applied. The confidence limit was accepted at 95% (P <0.05).

RESULTS

Malondialdehyde (MDA) concentration was higher among obese and overweight compared with non-obese with highly significant difference (F=5.52, P<0.01). Further investigation using L.S.D. test showed that MDA level was highly significantly (m.d.=-0.16, P<0.01) and significantly (m.d.=-0.14, P<0.05) higher among obese than non-obese and overweight subjects respectively. Although no significant differences were recorded concerning vitamin E and uric acid levels but the concentrations of both were elevated among obese and overweight in comparison to non-obese (P>0.05) (**Table1**). Salivary flow rate showed higher value among non-obese subjects with highly significant difference (F=8.11, P<0.01) as revealed in **Table 2**. In addition L.S.D. test showed highly significant difference in salivary flow rate between non-obese and overweight (m.d.=0.27, P<0.01) and between non-obese and obese (m.d.=0.28, P<0.01). It is worth to mention that salivary flow rate was the same in both overweight and obese subjects (P>0.05). Regarding oral cleanliness and gingival health, **Table 3** reveals statistically no significant differences in PlI, CalI, and GI mean values among the three BMI categories (P>0.05). Also no significant differences were found regarding PPD and CAL (P>0.05) among BMI categories as shown in **Table 4**. However, CAL mean value was significantly higher among obese than overweight using L.S.D. test (m.d.= -1.24, P<0.03).

The extent of PPD with different thresholds of severity according to BMI categories are shown in **Table 5**. Highly significant difference was found in ≥ 5mm PPD threshold among BMI categories (P<0.01). Further investigation using the L.S.D. test revealed that ≥ 5mm threshold was highly significantly higher among non-obese than overweight (m.d.=0.76, P<0.01) and obese individuals (m.d.=0.76, P<0.01). Regarding other PPD thresholds no significant differences were found (P>0.05). The extent of CAL with different thresholds of severity according to BMI categories is shown in **Table 6**. ≥2 mm threshold showed highly significant difference among BMI categories (P<0.01). Similarly, ≥3, ≥7 and ≥9 mm thresholds showed significant difference among BMI categories (P<0.05). Further investigation using L.S.D. test revealed that ≥2, ≥3 and ≥7 mm were highly significantly higher among obese than overweight (m.d.=-41.56, P<0.01; m.d.=-31.35, P<0.01; m.d.=-3.22, P<0.01 respectively), in addition ≥4 and ≥5 mm thresholds were significantly higher among obese than overweight (m.d.=-22.77, P<0.05; m.d.=-15.60, P<0.05 respectively). The most severe threshold (≥9 mm) was significantly higher among obese compared to non-obese and overweight (as it was absent in both) (m.d.=- 0.37, P<0.05).

DISCUSSION

In the current study, the results obtained showed no significant difference in gingival inflammation among BMI categories which might be related to non-significant differences in dental plaque and calculus accumulations among the three categories of BMI. Similarly no significant differences were recorded in PPD and CAL mean values among BMI categories. However, when PPD and CAL are considered according to thresholds of severity (extent) the results regarding PPD could not be explained because ≥1, ≥2 and the severe threshold ≥5 mm were higher among non-obese than overweight and obese; while ≥3 and ≥4 thresholds were higher among obese and overweight than non-obese and among obese than non-obese and overweight respectively. Concerning CAL the results were more obvious because CAL at almost all thresholds of severity was higher among obese than non-obese (in particular the severe threshold ≥9 mm). However, overweight subjects revealed the lowest CAL value probably due to sample size. Severe attachment loss among obese subjects was also found by other studies [11-15]. CAL findings were more obvious than PPD results probably due to the fact that the reference points for the assessment of attachment loss and probing depth are the cementoenamel junction and the gingival margin respectively. Since the cementoenamel junction is fixed whereas the gingival margin is not, attachment loss represents a cumulative effect of overall disease experience and the resulting loss of periodontal support, whereas the probing depth measurement is more variable and also may depend on the magnitude of gingival recession at the site of measurement [30].

Higher clinical attachment level among obese persons than non-obese probably related to higher calculus accumulations among obese than non-obese but with no significant difference. Dental calculus plays a role in periodontal disease pathogenesis since it is a mineralized dental plaque with a layer of unmineralized plaque on its surface also it acts as a retentive factor for dental plaque [31]. Another cause is elevated lipid peroxidation (i.e. MDA) level among obese than non-obese with highly significant difference. The role of ROS-induced lipid peroxidation and damage of periodontal tissues was reported by many studies [20, 21, 32]. Reduced salivary flow rate among obese compared to non-obese with highly significant difference may further explain higher periodontal destruction among obese subjects. The flushing effect of salivary flow is the most important one, not only because it so effectively removes oral microorganisms and their products into the gut but also because a steady supply of saliva ensures continuous presence of both non-immune and immune-factors in the mouth [33]. Also higher periodontal disease severity among obese might be related to unhealthy diet with insufficient micronutrients and excess fat content that increasing the risk for periodontal disease and obesity as well [11]. In addition it was found that adipocytes have been identified as active producers of cytokines including TNFα, IL-6 and to some degree IL-1 [34]. These inflammatory mediators increased the inflammatory state that sets the stage for increased levels of periodontal disease triggered by oral pathogens [35]. Furthermore, psychological stress associated with obesity may affect periodontal health through physiological and behavioral pathways. Physiological response to stress may alter blood and salivary flow, decreasing the immune response to oral pathogens [36]. Psychological stress may affect oral health behaviors such as regular brushing and flossing in addition to seeking preventive dental care [37].

Also data analysis showed that vitamin E and uric acid levels were higher among obese and overweight compared with non-obese though statistical differences were not significant. This might be related to the fact that the body raises the level of its antioxidant systems to combat the oxidative damage [38]. These antioxidants enhance periodontal health by providing protection against ROS-induced damage of periodontal tissues especially gingival hyaluronic acid and proteoglycans [39]. Vitamin E prevents ROS-induced lipid peroxidation of the cell membrane because of its localization in the cell membrane [40]. Accordingly it has immunenhancing effect since it protects cells of the immune system against the oxidative damage [41]. Uric acid is a relatively powerful scavenging antioxidant of water soluble radicals [42]. It can also bind copper and iron ions that catalyzed ROS formation [43].

Table 1: Salivary antioxidants and lipid peroxidation biomarker (MDA) (Mean ±S.D.) according to BMI categories.

Variable (mg/dl)	Non-obese (<25 Kg/m$^{2)}$			Over weight (≥25-<30 Kg/m^2)			Obese (≥30 Kg/m^2)			ANOVA test df=2	
	No.	Mean	±SD	No.	Mean	±SD	No.	Mean	±SD	F-value	P-value
Vitamin E	13	0.16	0.09	9	0.17	0.08	13	0.21	0.19	0.52	0.60
Uric acid	13	6.20	1.04	9	6.41	1.60	13	6.79	1.49	0.61	0.55
MDA (µmol/L)	13	0.01	0.072	9	0.11	0.09	13	0.26	0.19	5.52	0.009**

** Highly Significant

Table 2: Salivary flow rate (Mean ±S.D.) according to BMI categories.

Variable	Non-obese (<25 Kg/m$^{2)}$			Over weight (≥25-<30 Kg/m^2)			Obese (≥30 Kg/m^2)			ANOVA test d.f.=2	
	No.	Mean	±SD	No.	Mean	±SD	No.	Mean	±SD	F-value	P-value
Flow rate (ml/min)	13	0.56	0.24	9	0.28	0.10	13	0.28	0.21	8.11	0.001**

** Highly Significant

Table 3: Plaque, calculus and gingival indices (Mean ±S.D.) according to BMI categories.

Variable	Non-obese (<25 Kg/m$^{2)}$			Over weight (≥25-<30 Kg/m^2)			Obese (≥30 Kg/m^2)			ANOVA test df=2	
	No.	Mean	±SD	No.	Mean	±SD	No.	Mean	±SD	F-value	P-value
PlI	13	0.93	0.37	9	0.78	0.31	13	0.87	0.37	0.52	0.60
CalI	13	0.50	0.43	9	0.77	0.39	13	0.75	0.42	1.57	0.22
GI	13	1.18	0.03	9	1.16	0.13	13	1.16	0.07	0.33	0.72

Table 4: PPD and CAL (Mean ±S.D.) according to BMI categories.

Variable	Non-obese (<25 Kg/m$^{2)}$			Over weight (≥25-<30 Kg/m^2)			Obese (≥30 Kg/m^2)			ANOVA test df=2	
	No.	Mean	±SD	No.	Mean	±SD	No.	Mean	±SD	F-value	P-value
PPD	13	1.50	0.44	9	1.29	0.43	13	1.31	0.39	0. 90	0.42
CAL	13	1.80	1.22	9	1.10	0.98	13	2.34	1.43	2.62	0.09

Table 5: Extent of PPD (Mean ±S.D.) according to BMI categories.

PPD	Non-obese (<25 Kg/m$^{2)}$			Over weight (≥25-<30 Kg/m^2)			Obese (≥30 Kg/m^2)			ANOVA test df=2	
	No.	Mean	±SD	No.	Mean	±SD	No.	Mean	±SD	F-value	P-value
≥ 1	13	100.00	0.00	9	94.05	11.30	13	95.36	9.95	1.65	0.21
≥2	13	35.19	22.65	9	22.84	17.60	13	22.52	18.93	1.59	0.22
≥3	13	6.07	9.33	9	10.57	17.87	13	11.08	14.76	0.49	0.62
≥4	13	1.83	3.02	9	1.55	2.56	13	2.20	3.08	0.14	0.87
≥5	13	0.76	1.002	9	0.00	0.00	13	0.00	0.00	6.35	0.005**
≥7	13	0.00	0.00	9	0.00	0.00	13	0.00	0.00	-	-

*Significant ** Highly Significant

Table 6: Extent of CAL (Mean ±S.D.) according to BMI categories.

CAL	Non-obese (<25 Kg/m$^{2)}$			Over weight (≥25-<30 Kg/m^2)			Obese (≥30 Kg/m^2)			ANOVA test df=2	
	No.	Mean	±SD	No.	Mean	±SD	No.	Mean	±SD	F-value	P-value
≥ 1	13	62.70	28.27	9	64.91	18.10	13	62.44	33.46	0.02	0.98
≥2	13	40.56	25.13	9	17.08	22.11	13	58.64	35.08	5.60	0.008**
≥3	13	29.84	25.42	9	9.00	21.28	13	40.35	26.93	4.20	0.024*
≥4	13	20.89	20.96	9	7.56	18.35	13	30.32	21.33	3.29	0.050
≥5	13	14.96	15.58	9	6.33	16.09	13	21.93	17.63	2.38	0.11
≥6	13	7.93	9.06	9	4.78	12.86	13	15.26	14.22	2.23	0.12
≥7	13	2.30	2.25	9	0.44	1.33	13	3.67	3.47	4.02	0.03*
≥8	13	0.78	0.67	9	0.00	0.00	13	0.91	1.76	1.85	0.17
≥9	13	0.00	0.00	9	0.00	0.00	13	0.37	0.57	4.47	0.02*

*Significant ** Highly Significant

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