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ASSOCIATION BETWEEN BIOCHEMICAL LEPTIN AND SOME BIOCHEMICAL MARKERS IN IRAQI PATIENTS WITH OBESITY AND PERIODONTAL

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ABSTRACT

The objective of the study is to correlate the relationship between body composition and the activity of the alpha-amylase enzyme in the saliva of healthy people and people with obesity which may provide further clues to investigate the potential regulation and biological mechanism of obesity. The results showed the gender of patients of the four groups revealed no significant differences in mean of gender (males and females) between the groups. Data showed that the there is a significant difference between the mean of patients with periodontal disease, the mean of age of patients of the four groups studied was ranged from 24.75 years in patients with obesity without periodontal disease group to 32.55 years in patients with obesity and periodontal disease group. Statements indicated that there is a significant difference between weight, BMI and groups studied. Data showed that ghrelin parameter varied in their effect on obesity and periodontal disease which seemed in their values of the tested four groups studied. The findings demonstrated that there is a significant difference in mean of insulin levels of groups studied; it seems that all the patients groups significantly decreased the level of insulin as compared with healthy control group. Patients with non-obesity and periodontal group recorded the lower value of insulin level (8.80 µU/ml) while patients with obesity and periodontal group recorded tha upper value of insulin level which recorded 14.48µU/ml with no significant differences with patients with obesity without periodontal group (14.48 vs. 10.24 µU/mL). Data demonstrated that there is a significant difference between the mean of FBS levels control group with the patients groups studied. The data revealed that the groups studied varied in their values of HbA1C levels with a normal range of this parameter (HbA1C).

Keywords: Obesity, Leptin, Ghrelin, Insulin resistance, TSH

1. INTRODUCTION

Obesity is a huge public health issue that affects people all over the globe. Since 1975, the prevalence of obesity has climbed by a factor of three, and this trend is expected to continue into the foreseeable future (Engin 2017). In spite of the fact that obesity is defined as an imbalance between a person's energy expenditure and the amount of food they consume, there are a number of variables, both hereditary and non-genetic, such as environmental and behavioral factors, that

contribute to obesity. Because of the inclusion of hereditary variables in addition to hormones, there may be a 40–70% inter-individual variation in body mass index (BMI) within a single family. This is linked to the fact that hormones have a role in the regulation of body weight (Ogden et al. 2016).

Leptin and ghrelin are two factors that have a major influence on the organization of the food that is consumed and the weight of the body. These hormones also have an effect on the dopaminergic transition that occurs in the brain's reward centers and the food-seeking behavior of animals, and they activate the reward sections of the brain in humans (Reilly et al. 2018).

Because leptin is a hormone that is essential to the intake of energy as well as the control of that consumption, we may say that leptin is a moderator of the long-term management of energy balance. Because it is the hormone that is responsible for providing certain messages to the brain that a person is feeling full and that he has taken in the adequate quantity of calories that his body requires, leptin is also known as hunger hormone or the satiety hormone (Skinner et al. 2018).

This is because it is the hormone that is responsible for sending these notifications. Or, in order for the process to operate in reverse, the person still need a greater number of calories. The majority of leptin that is released into the circulation comes from adipose tissue, where it is generated. Leptin levels in the blood are an accurate reflection of the amount of adipose tissue present, and they tell the brain how much energy is being stored (Boulet 2015).

The levels of leptin that circulate in the blood display diurnal oscillations and also alter in response to the nutritional condition of the body. Leptin levels in the blood are lowered when one fasts, whereas they are raised when one eats regularly or becomes obese. Common responses to fasting may be reversed by preventing the decline in leptin levels that is generated by fasting. This highlights the relevance of leptin levels for maintaining energy balance. In addition, when the stomach is empty, the gastrointestinal system produces ghrelin, which then travels via the blood to the hypothalamus in the brain to trigger the sense of hunger and boost appetite. This causes a person to consume more calories since they have a greater desire to consume food (Hruby et al. 2016).

LITERATURE REVIEW

Obesity

The human body is composed of fat-free mass and body fat in addition to other tissues. The mass that is not constituted of fat is called the fat-free mass, and it is made up of all of the body's non-fatty components, including bones, water, muscles, teeth, and connective tissues. There are fats that are important to the body as well as fats that are not needed. Adipose tissue, which is found both under the skin and all around the body's main organs, contains non-essential fat (Hruby et al. 2016).

The percentage of people who are overweight or obese is growing at a startlingly rapid pace throughout the whole planet. It is anticipated that by the year 2010, the number of children in the European Union who are overweight or obese would exceed 26 million, with 6.4 million of those children being classified as obese. The number of people in Europe who are overweight or obese

is growing by 1.3 million each year. In the 10 years that spanned from 1985 to 1995 in Australia, there was a significant rise in the percentage of children who were either overweight but not obese or obese. This rise occurred over the time in question (Smith et al. 2016).

Leptin

It is a 146 amino acid-long protein that is encoded by the obesity gene such as IL2 and IL12. Leptin is a hormone that is produced by fat tissue and that acts on the brain to regulate food intake and body weight. As a result, it is regarded to be a part of the cytokine superfamily of class I. It is produced largely in the adipocytes of white adipose tissue, and the quantity of circulating leptin is proportional to the overall amount of fat in the body. This hormone travels through the bloodstream and controls both the amount of food consumed and the amount of energy expended by acting on the hypothalamus (Friedman 2014, Izquierdo et al. 2019).

Plasma leptin levels drop as fat mass drops, and this stimulates hunger and reduces energy expenditure while waiting for fat mass to return to normal levels. This physiological mechanism guarantees that total energy storage are consistently maintained within a very small range. When fat mass grows, leptin levels also increase, which in turn suppresses hunger until weight loss occurs. In the center of the brain, where it lowers hunger by opposing the consequences of neuropeptide, which is a strong eating stimulant generated by cells in the stomach and in the hypothalamus, leptin operates on receptors to suppress appetite (D'souza et al. 2017).

The lack of leptin causes one to consume food without moderation, which ultimately results in obesity. Leptin levels may be lowered by fasting or by adhering to a very low-calorie diet (VLCD), as demonstrated in a number of studies. It's possible that leptin may act as a signal of energy balance in the near term. This mechanism is more susceptible to famine than it is to overfeeding; changes in leptin levels occur more often when food intake drops than when it rises (Seoane-Collazo et al. 2020).

Leptin and insulin

The beta cells that are found in the islets of Langerhans in the pancreas are responsible for the production of the hormone insulin. Not insulin itself, but the insulin precursor known as preproinsulin is what beta cells are responsible for producing. A signal peptide and the prosinsulin are the two components that make up this peptide, which has a total of 109 amino acids. It is in the Rough Endoplasmic Reticulum (RER) that the Preproinsulin is broken by a peptidase, and the signal peptide is responsible for directing it there. In addition, this enzyme is responsible for the formation of three sulphur bridges, which are necessary for the folding of the Proinsulin molecule into its mature state. The Trans-Golgi Network (TGN) is responsible for the transformation of proinsulin into mature insulin. In point of fact, peptidases sever Proinsulin in two distinct locations, which results in the production of two distinct substances, namely C-peptide and insulin. Insulin is made up of two distinct chains that are connected by sulfur bridges. Exocytosis is the process through which insulin and C-peptide leave the cell (Petersen and Shulman 2018).

Insulin increases the phosphorylation of Akt and endothelial NO synthase that is triggered by leptin, which in turn increases the amount of NO that is released. This brings up the potential of

insulin and leptin signals interacting with one another in some way. In contrast, leptin does not change the amount of blood flowing through the mesentery of conscious rats that have been treated with NO synthase inhibitors or adrenergic blockers. This is the case despite increased sympathetic activity. Based on these findings, it seems that leptin has an effect on the NO-dependent vascular reactivity of resistance arteries. Intriguingly, the injection of systemic leptin does not reduce the vasoconstriction that is generated by the stimulation of sympathetic nerves. This suggests that the direct vasodilator effects of leptin may not be adequate to resist the vasoconstriction that is caused by sympathetic nerve stimulation (Koch et al. 2014).

Materials

Tools and other medical requirements

The tools and instruments that used in this study are discriped in Table 3.1.

Table 3.1 Tools and instruments used in this study

No.	EquipmentandInstruments	Company	Country
1	Micropipettes (different volumes)	Eppendorf	Germany
2	Multichannelpipette (50-300µL)	Biohit	U.K
3	Absorbance microplate reader	Karl Kolb	Germany
4	Capped plastic tubes	Afco-Dispo	Jordan
5	Centrifuge	Kokusan	Germany
6	Ependroff tubes	Sigma	England
7	Deep freezer (-80 °C)	Angel Antoni	Italy
8	Gel electrophoresis apparatus	Bioneer	Korea
9	Gel tubes	Afco	Jordan
10	Hot plat stirrer	LabTech®	Korea
11	Incubator	Memmert	Germany
12	Microcentrifuge	Bioneer	Korea
13	Spectrophotometer	Shimadzu	Japan
14	Vortex mixer	CYAN	Belgium
15	Water bath	Kottermann	Germany
16	Refrigerator	Arjelic	Turky
17	Hormone analyzer	TOSOH 600	Japan
18	Elisa washer and reader	Serio ELISA	Italy

Subject selection

The subjects were included in four groups

1- 45 Healthy individuals (control group).
2- 55 İndividuals without obesity and with periodental disease.
3- 55 İndividuals with obesity and with periodental disease.
4- 55 İndividuals with obesity and without periodental disease.

Methods

Blood sample colection

Blood samples were collected from the veins of patients or individuals who recurred Ramad Teaching Hospital in Anbar Governorate, Iraq for hematological investigations that includes lipids profile (total choesterol, total glycerides, LDL, HDL, and VLDL), insulin level, random blood sugar (RBS), HbA1C, and leptin levels in obesity patients with periodonatal disease.

The blood samples that collected in a correct mannar were sent to the laboratory accompanied by a laboratory requisition form filled in by the clinician.Brief clinical information must be mentioned.Blood can be withdrawn from the vein by using sterile plastic syringe, the vein selected should be large then we cleanned the skin of the area around the identified vein by using 70% isopropyl alcohol, apply a tourniquet 3-4 inches above the venipuncture site, then insert the needle og syringe in the vein, release the tourniquet as soon as blood enters the syringe then withdraw the piston slowly to avoid frothing, deliver the blood gently into the suitable receiver, cap it to prevent leakage. Maintain light pressureon the gauze pad over venipuncture site till the bleeding stops and cover the puncture site with a small adhesive dressing then sent the blood samples to the laboratory for proceeding the tests.

Estimatin of leptin in the blood serum

This test is used to measures the amount of leptin the blood serum of sample to detect a deficiency that may be contributing to obesity, leptin is a hormone produced by fat cells in the body that helps regulate hunger and maintain normal weight on a long-term basis.

This test can be done by taking a blood sample from a vein, we used a commercial kit based on competitive enzyme –linked immunosorbent assay (ELISA). The quantitative measurement of leptin in blood serum was performed by using leptin enzyme immunoassay kit (DRG Diagnostic, Marburg, Germany) depending on the manufactions structions.

Insulin test

Insulin test is a blood test that helps evaluate insulin production by the beta cells in the pancreas insulin levels are most frequency ordered following a low blood glucose test result and when someone has acute or chronic symptoms of low blood glucose (hypoglycemia).

Procedure

Add 25 µL of calibrators, controls and samples in duplicate and incubate for 20 minutes then add 100 µL of enzyme conjugate and incubate for 10 minutes.After the set of ELISA plate is complete, continous the assay manually by covering the plate with plate cover and incubate for 60 minutes at room temperature on a horizantal orbital microplate shaker then set at 450 rpm.

Aspirate each well and wash with a microplate washer for five times, invert the plate and blot against clean paper towels between each wash then removed the wash buffer then invert the plate and blot it against clean paper towels.

Add 200 µL of TMB to each well, cover the plate with a new cover incubate for 15 minutes at 25 °C then add 50 µL of stop solurtion to each well using a 12-chennel pipet. Pipet up and down a few times to mix solutions then read the plate on a microplate reader at 450nm and calculate the results.

Statistical Analysis

SPSS (Statistical Package for Social Science – version 23) is use for statistical analysis. The results are present as mean SE (standard error). The significance of the difference is determine using a two-sample t-test for two independent means and a paired t-test for two dependent means. For the relationship between two quantitative variables, correlation and regression are use, with P 0.05 set

as the lowest level of significance. RESULTS AND DISCUSSION

Baseline Salivary Enzymes Analysis and some Biochemical Parameters of the Studied Groups

Aimed this study to knowledge to correlate the relationship between body composition and the activity of the alpha-amylase enzyme in the saliva of healthy people and people with obesity which may provide further clues to investigate the potential regulation and biological mechanism of obesity. The results of parameters showed that baseline of the tested parameters in this study revealed that the mean and SD as in below:

The Gender of Patients with Obesity

The results of Table 4.1 showed the gender of patients of the four groups revealed no significant differences in mean of gender (males and females) between the groups (control healthy individuals, patients without obesity, periodontal disease, patients with obesity and periodontal disease, patients with obesity without periodontal disease as shown in Table 4.2 and Figure 4.1.

These results were disagreement with the results of Jain and Bhavsar (2021) which found that the clinical parameters of periodontal disease were higher in females than females, while Al-Abdaly et al. (2019) indicated that the periodontal cases were significantly higher sensitivity in females as compared with to male's patients with periodontal disease, they suggested that periodontal disease were related to subclinical atherosclerosis in males but not in females.

Table 4.2 The gender parameter and study groups

Parameter		Group of study				Total
		Control healthy individuals	Patients without obesity with periodontal disease	Patients with obesity and periodontal disease	Patients with obesity without periodontal disease	
Gender	Male	25	27	28	29	109
	%	20.0%	22.0%	22.0%	24.0%	22.0%
	Female	27	26	25	23	101
	%	20.0%	18.0%	18.0%	16.0%	18.0%
Total		45	55	55	55	210

4.1 Age of Patients of Groups Studied

Data of Table 4.3 showed that the there is a significant difference between the mean of patients with periodontal disease, the mean of age of patients of the four groups studied was ranged from 24 - 68 years in patients with obesity without periodontal disease group to 30 - 60 years in patients with obesity and periodontal disease group (Table 4.3 and Figure 4.2). The standard deviation of the four groups recorded 3.84, 4.368, 5.362, and 3.574 respectively.

Table 4.3 Age of patients of groups studied

Group of study	Mean of age (Years)	N	Std. Deviation	Std. Error of Mean
Healthy individuals	25.884	45	3.846	0.05282
persons with non- obesity with periodontal disease	27.473	55	4.368	1.01793
persons with obesity and periodontal disease	30.309	55	5.362	1240951
persons with obesity without	22.584	55	3.574	1.04598

periodontal disease				
Total	27.9375	210	6.746	0.65079

The results of Table 4.4 revealed that no significant correlation between age of patients with periodontal disease and groups studied, this may due to the lack in number of patients in each group (165 patients) that lead to absence of correlation between age and groups studied.

Table 4.4 Correlation between age of patients with groups studied

		Age (Years)	Group of study
Age	Pearson Correlation	1	-.001
	Sig. (2-tailed)		0.864
	N	210	200
Group of study	Pearson Correlation	-.001	1
	Sig. (2-tailed)	.0.864	
	N	210	200

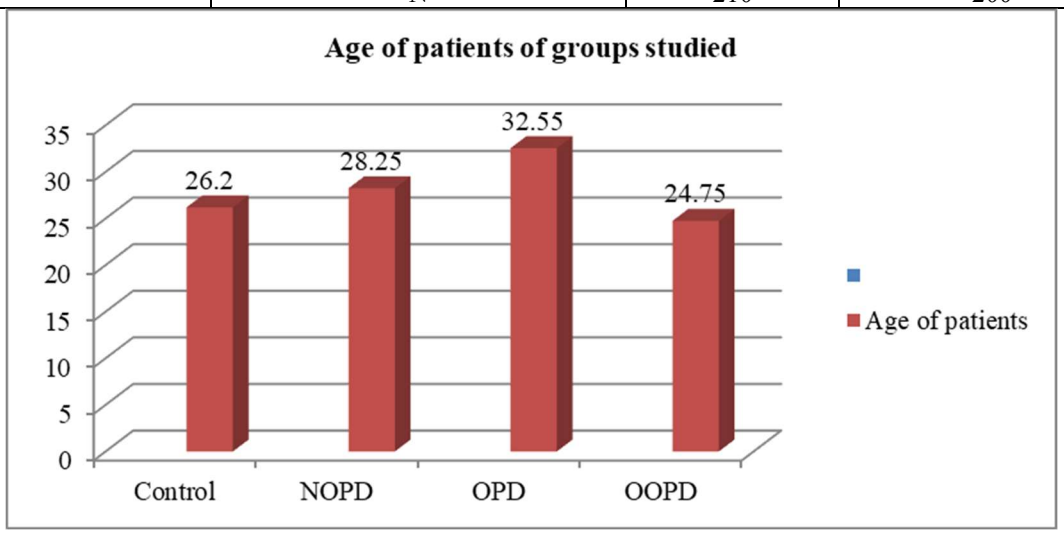

Figure 4.2 Age of patients of groups studied

4.2 The Weight and BMI of Patients of Groups Studied

Statements of Table 4.5 indicated that there is a significant difference between weight, BMI and groups studied, patients with obesity without periodontal disease significantly excellence in mean of height of patients and BMI which recorded 96.70 cm and 30.62 kg/m2 as compared with 82.40 cm and 23.89 kg/ m2 for control group respectively (Table 4.5 and Figure 4.3).It is clearly that periodontal disease may negative affected on weight and BMI index by decreasing the appetite to eating of foods which seemed in values of these parameters (weight and BMI) which recorded upper values between groups studied. Sunitha et al. (2010) indicated that the possible causal relationship between obesity and periodontal disease and potential underlying biological mechanisms remain to be established and the adipose tissue activity secretes a variety of cytokines and hormones that are involved in inflammatory processes. While, Khader et al. (2009) found that 14% of normal weight persons had periodontal disease while 29.6% of overweight and 51.9% of obese persons had periodontal disease.

Table 4.5 The weight and BMI of patients of groups studied

Group of study		Weight (kg)	BMI (kg/m^2)
Healthy individuals	Mean	82.4000	23.8932
	N	45	45
	Std. Deviation	5.81649	2.47180
	Std. Error of Mean	1.30061	.55271
Persons with non- obesity with periodontal	Mean	79.3500	24.1608
	N	55	55
	Std. Deviation	5.72414	1.94693
	Std. Error of Mean	1.27996	.43535
Persons with obesity and periodontal	Mean	92.0500	30.1222
	N	55	55
	Std. Deviation	10.79218	1.51542
	Std. Error of Mean	2.41320	.33886
Persons with obesity without periodontal	Mean	96.7000	30.6276
	N	55	55
	Std. Deviation	7.82775	1.60619
	Std. Error of Mean	1.75034	.35915
Total	Mean	87.6250	27.2010
	N	210	210
	Std. Deviation	10.43166	3.71441
	Std. Error of Mean	1.16630	.41528

Table 4.6 Correlation between weight and BMI and groups studied

		Group of study	weight	BMI
Group of study	Pearson Correlation	1	.600**	.793**
	Sig. (2-tailed)		.000	.000
	N	210	210	210
weight	Pearson Correlation	.600**	1	.770**
	Sig. (2-tailed)	.000		.000
	N	210	210	210
BMI	Pearson Correlation	.793**	.770**	1
	Sig. (2-tailed)	.000	.000	
	N	210	210	210
**. Correlation is significant at the 0.01 level (2-tailed).				

Data of Table 4.6 pointed that there is a strong positive significant correlation between weight and BMI of patients and groups studied which recorded 0.600** at a P level of 0.01.These results were in agreement with the results of Keller et al. (2015) which concluded that the is an association between periodontal disease and overweight and obesity at baseline and risk of developing periodontal disease, while Al-Zahrani et al. (2003) found that the abnormal obesity are associated with increased prevalence of periodontal obesity could be a potential risk factor for periodontal disease.

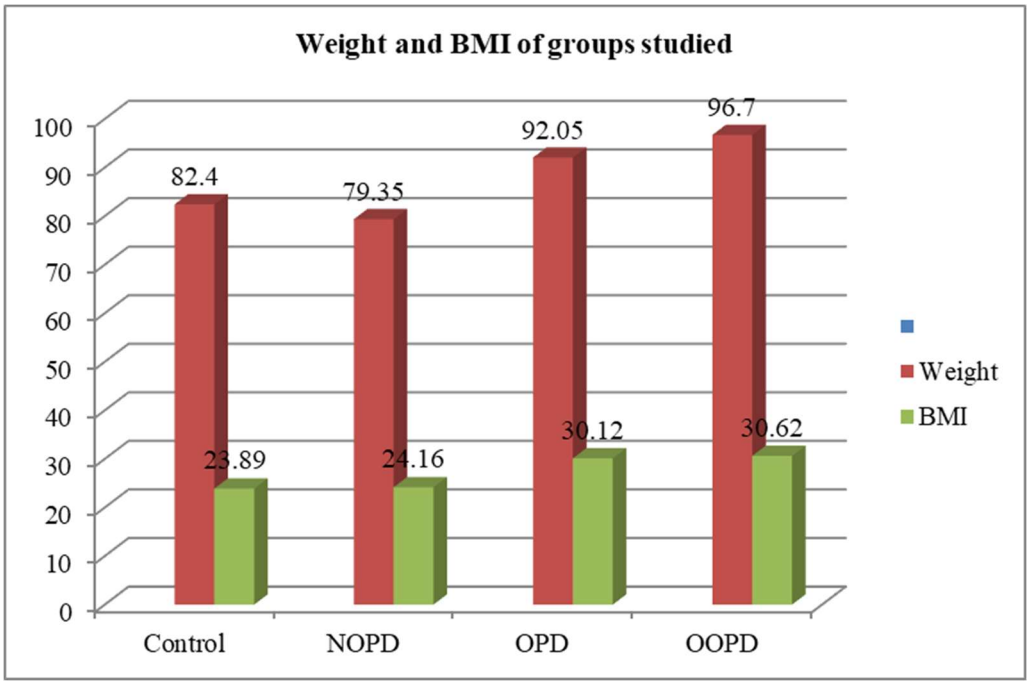

Figure 4.3 Weight and BMI of patients of groups studied

4.3 Leptin Level and Groups Studied

The results of Table 4.11 and Figure 4.6 indicated that the four groups studied varied in their levels of leptin, the three patients' groups mostly increased the levels of leptin as compared with healthy individuals, patients with periodontal without obesity significantly elevated the level of leptin which recorded 13.85 ng/mL as compared with 8.21 ng / mL for control group while persons with obesity and periodontal group showed no significant differences with persons with non-obesity and periodontal group (13.85 ng/ml vs. 15.92 ng/ml).

The standard deviation of patients with periodontal without obesity recorded 6.64 as compared with 3.55 for healthy control group.The elevated of leptin levels are related with obesity and inflammation related diseases including high blood pressure, metabolic syndrome and heart disease.Leptin is a single – chain protein hormone with a molecular mass of 16 KDa that is thought to play a key role in the regulation of body weight, it is produced by differentiated adipocytes, although production has been demonstrated in other tissue such as the fundus of the stomach, skeletal muscle, liver, and the placenta (Friedman and Halaas 1998).

Leptin is a hormone that plays as a mediator in the stomach – hypothalamus pathway and provides information about the body's energy storage in adipocytes and its level is linked with obesity (Edmann et al. 2005).

Karam (2013) showed that the level of leptin was significantly higher in patients with chronic periodontitis which recorded 9.81 ng/mL as compared with healthy control which recorded 5.61 ng/mL and found the level of leptin in saliva of chronic periodontitis patients was significantly lower (0.17 ng/mL) than that of its salivary levels in healthy control (0.29 ng/mL). Also, the results

of Table 4.12 showed that there in a weak positive correlation between leptin level and groups studied which recorded 0.121.

Table 4.11 Leptin level of groups studied

Group of study	Mean ng/mL	N	Std. Deviation	Std. Error of Mean
Healthy individuals	8.2196	45	3.55080	0.79398
persons with non -obesity with periodontal	13.8549	55	6.64710	1.48634
persons with obesity and periodontal	15.9258	55	5.92458	1.32478
persons with obesity without periodontal	9.6470	55	3.57872	0.80023
Total	11.9118	210	5.91319	0.66112

Al-Ardhi and Ibraheem (2019) found that there is a significant elevation (P<0.01) level of leptin hormone in obese groups in compared with control group, they concluded that the leptin hormone has a positive correlation with obesity.

While, Vincent et al. (2003) showed that the individual's overweight has high levels of leptin than healthy weight and underweight individuals.

While, Husain et al. (2020) indicated that the leptin level of patients group recorded 44.48 ng/mL as compared with 38.75 ng/mL for healthy subjects.

Table 4.12 Correlation between leptin level and groups studied

		Group of study	Leptin
Group of study	Pearson Correlation	1	0.121
	Sig. (2-tailed)		0.285
	N	210	210
Leptin	Pearson Correlation	0.121	1
	Sig. (2-tailed)	0.285	
	N	210	210

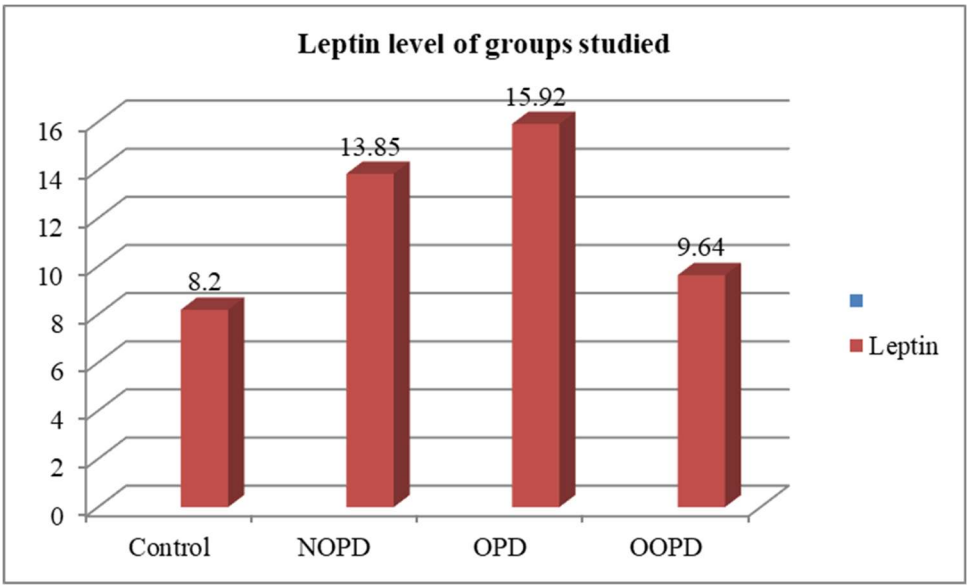

Figure 4.6 Leptin level of groups studied

4.4 Insulin Level of Groups Studied

The findings of Table 4.15 and Figure 4.8 demonstrated that there is a significant difference in mean of insulin levels of groups studied; it seems that all the patients' groups significantly decreased the level of insulin as compared with healthy control group. Patients with non-obesity and periodontal group recorded the lower value of insulin level (8.80 µU/ml) while patients with obesity and periodontal group recorded tha upper value of insulin level which recorded 14.48µU/ml with no significant differences with patients with obesity without periodontal group (14.48 vs. 10.24 µU/ml).

The decreasing in insulin level in patients' groups may be due to to the removal of pathogens by treatment leads to a decreased of inflammation which lead in reduces insulin resistance and reduced the sugar level or the decreasing in insulin level of periodontal disease may be due to a small size of the samples taken (55 patient for every group) or may be to the patients taken some drugs that lead to lowering the insulin level.

It was found that during obesity, the periodontal bacterial components may induce chronic inflammation and oxidative stress contributing to the development of insulin resistance (Blasco-Baque et al. 2017). Also, the results of Table 4.16 showed that there is a weak negative correlation between insulin level and groups studied which recorded-0.140. Recently obesity has been recorded to be associated with periodontitis, obesity induces macrophage accumulation in adipose tissue, promotes chronic low- grade inflammation and increase adipokines derived from adipocytes (Khan et al. 2020).

Table 4.15 Insulin level of groups studied

Group of study	Mean mcU/mL	N	Std. Deviation
Healthy individuals	15.8368	45	5.54403
persons with non-obesity with periodontal	8.8091	55	4.05303
persons with obesity and periodontal	14.4895	55	14.74590
persons with obesity without periodontal	10.2425	55	5.35184
Total	12.3445	210	8.89175

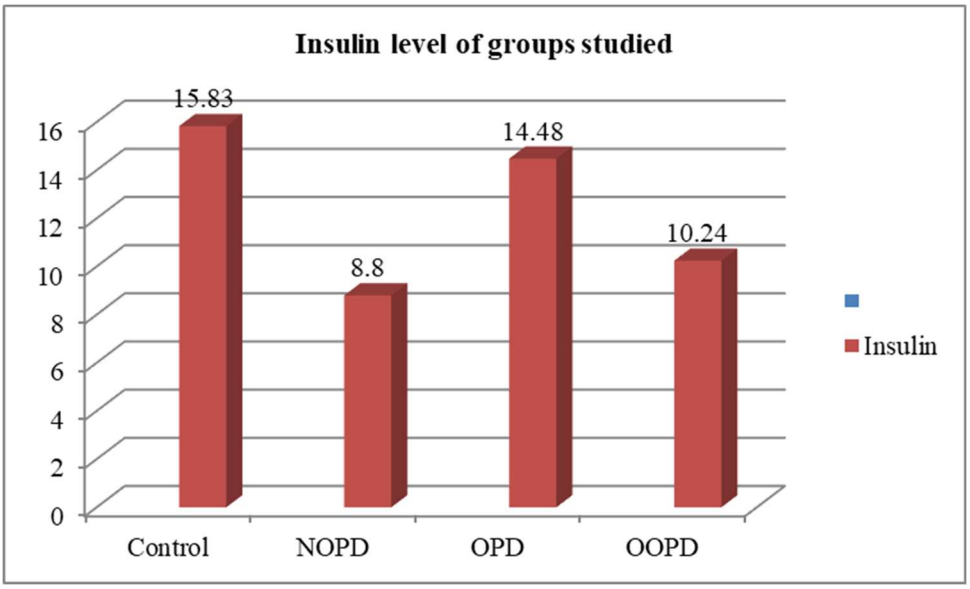

Figure 4.8 Insulin level of groups studied

Table 4.16 Correlation between insulin level and groups studied

		Group of study	Insulin
Group of study	Pearson Correlation	1	-.140
	Sig. (2-tailed)		.214
	N	210	210
Insulin	Pearson Correlation	-.140	1
	Sig. (2-tailed)	.214	
	N	210	210

4.5 FBS Level of Groups Studied

Data of Table 4.17 demonstrated that there is a significant difference between the mean of FBS levels control group with the patient's groups studied, patients' groups significantly elevated the levels of FBS as compared with healthy control group, patients with obesity without periodontal disease group significantly superior in increasing the level of FBS which recorded 121.50 mg/dL as compared with 91.35 mg/dL for control group (Table 4.17 and Figure 4.9). The standard deviation of patients with obesity without periodontal disease recorded 42.30 as compared with 15.48 for control group. Also, the results showed that patients with non- obesity and periodontal group has no significant differences with patients with obesity and periodontal (102.30 vs. 107.5) mg/dL while patients with obesity without periodontal group recorded 121.50 mg / dL.

These results were in agreement with the results of Agrawal et al. (2017) which concluded that the fast blood glucose level and BMI are positively correlated and the patients are at a risk of obesity, they showed that from among 150 participants, only 15 persons were suffering from periodontitis and 75 persons were overweight showed the presence of periodontitis, but 68% shown prevalence of periodontitis by obese persons. They found that the mean of blood glucose level was increased among obese persons as compared to normal persons. Thus, the obesity may become one of a main

cause of deterioration of life expectancy in the future (Al-Zahrani et al. 2003).

The obesity has been proposed to be a risk factor for periodontitis, it has been found that blood glucose level is positively associated with periodontal disease (Katz et al. 2000). The results of Table 4.18 found that there is a strong positive significant correlation between FBS level and groups studied which recorded 0.363** at a P value of a level of 0.01.

Table 4.17 FBS level of groups studied

Group of study	Mean mg/dL	N	Std. Deviation
Healthy individuals	91.3500	45	15.48607
persons with non-obesity with periodontal	102.3000	55	29.17659
persons with obesity and periodontal	107.4500	55	16.40756
persons with obesity without periodontal	121.5000	55	42.30404
Total	105.6500	210	29.60760

Table 4.18 Correlation between FBS level and groups studied

		Group of study	FBS
Group of study	Pearson Correlation	1	.363**
	Sig. (2-tailed)		.001
	N	210	210
FBS	Pearson C orrelation	.363**	1
	Sig. (2-tailed)	.001	
	N	210	210
**. Correlation is significant at the 0.01 level (2-tailed).			

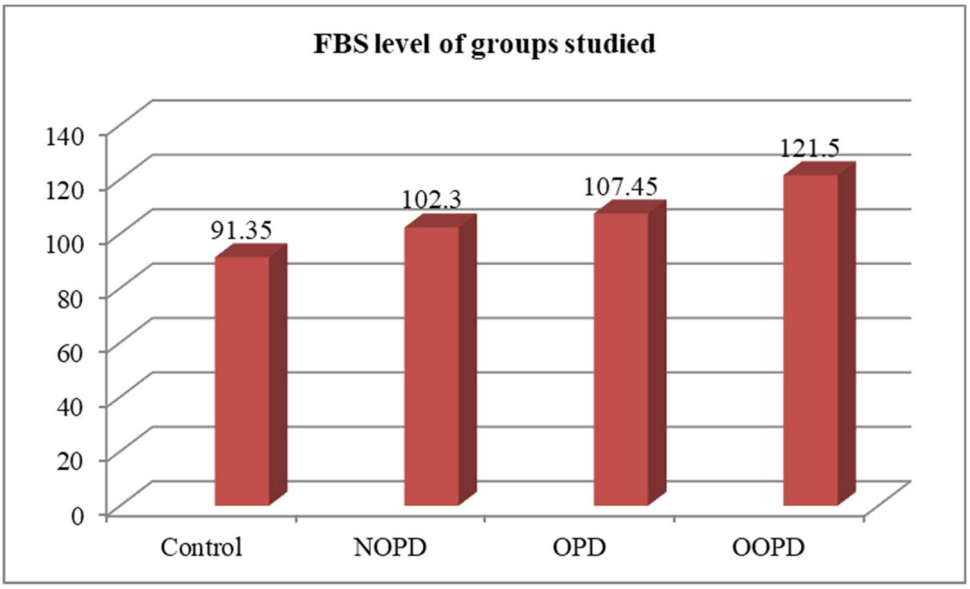

Figure 4.9 FBS level of groups studied

4.6 HbA1C Level of Groups Studied

The data of Table 4.19 revealed that the groups studied varied in their values of HbA1C levels with a normal range of this parameter (HbA1C). There are no significant differences between the mean of HbA1C level of groups studied.

Chen et al. (2021) found that after 3-6 months, periodontal therapy could significantly reduce the level of HbA1C and they concluded periodontal therapy significantly contributed to glycemic control in T2DM patients especially in patients with higher HbA1C levels.

While Samings et al. (2022) found that there is a significant correlation between BMI and HbA1C, and they concluded that obesity is positively correlated with HbA1C level in young adults without diabetes.

Kantharin et al. (2022) found that out of 100 patients, among obese there were 11, 9, 13, and 7 subjects with HbA1C level < 6.5 %, 6.5-8 %, 8-10 %, and > 10 %, while among normal BMI subjects there were 12, 6, 3, and 4 had HbA1C level <6.5 %, 6.5-8%, 8-10 %, and > 10 % respectively. Altamach et al. (2016) indicated that diabetes with periodontal and without Periodontal showed reductions in HbA1C level with a mean value of 0.3% surgical and non-surgical treatment. Also, Bag et al. (2023) showed that among the study population, 49% had HbA1C level less than 8% and they concluded that no association was found between HbA1C and BMI among patients with DM2. Husain et al. (2020) found that the level of HbA1C was recorded 7.83 in patients with typoe 2 diabetes while healthy subjects recorded HbA1C level 5.47.

Data of Table 4.20 showed no significant correlation between HbA1C level and groups studied which recorded 0.111.

Table 4.19 HBA1C level of groups studied

Group of study	Mean	N	Std. Deviation
Healthy individuals	4.8269	45	1.25881
Persons with non-obesity with periodontal	5.0754	55	1.54875
Persons with obesity and periodontal	4.9631	55	1.17838
Persons with obesity without periodontal	5.3197	55	1.54827
Total	5.0463	210	1.37897

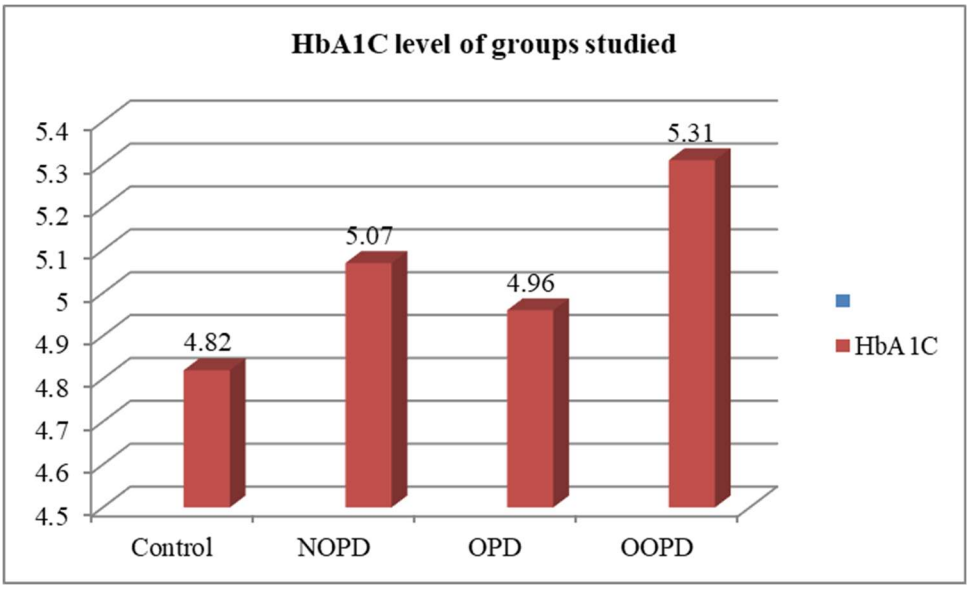

Figure 4.10 HbA1C level of groups studied

Table 4.20 Correlation between HbA1C level and groups studied

		Group of study	HBA1C
Group of study	Pearson Correlation	1	.111
	Sig. (2-tailed)		.325
	N	210	210
HBA1C	Pearson Correlation	.111	1
	Sig. (2-tailed)	.325	
	N	210	210

CONCLUSIONS AND RECOMMENDATION

Conclusions

The results showed the gender of patients of the four groups revealed no significant differences in mean of gender (males and females) between the groups (control healthy individuals).

Data showed that the there is a significant difference between the mean of patients with periodontal disease.

The statements indicated that there is a significant difference between weight, BMI, and groups studied, patients with obesity without periodontal disease significantly excellence in mean of height of patients and BMI.

The results indicated that the four groups studied varied in their levels of leptin, the three patient's groups mostly increased the levels of leptin as compared with healthy individuals, patients with periodontal without obesity significantly elevated the level of leptin which recorded 13.85 ng/mL as compared with 8.21 ng/mL for control group.

The results pointed that no significant differences between amylase levels of the four groups studied, mostly patients groups deceased the mean of amylase levels as compared with healthy

control, patients with obesity and periodontal disease revealed upper decreasing in amylase level which recorded 48.85 U/L as compared with 65.95 U/L for control group.

The findings demonstrated that there is a significant difference in mean of insulin levels of groups studied, it seems that all the patients' groups significantly decreased the level of insulin as compared with healthy control group.

Data demonstrated that there is a significant difference between the mean of RBS levels of groups studied, patients groups significantly elevated the levels of RBS as compared with healthy control group, patients with obesity without periodontal disease group significantly superior in increasing the level of RBS which recorded 121.50 mg/dL as compared with 91.35 mg/dL for control group.

The data revealed that the groups studied varied in their values of HbA1C levels with a normal range of this parameter (HbA1C). There are no significant differences between the mean of HbA1C level of groups studied.

Recommendations

- Patient's overweight and obese population seems to be at risk of low amylase activity which correlates with their obesity.
- Monitoring of the fast blood glucose level and BMI are positively correlated, and the patients are at a risk of obesity.
- Obesity has been proposed to be a risk factor for periodontitis.
- It has been found that blood glucose level is positively associated with periodontal disease.
- Determination of thyroid function (T3, T4, and TSH) may be useful in diagnosis of periodontal and obesity disease.
- Monitoring of lipids profile in blood of patients with periodontal and obesity may be important in diagnosis this disease.

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