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EVALUATION OF VISFATIN HORMONE LEVEL IN BASRAH OBESE WOMEN

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Abstract

Background: Visfatin is an adipokine secreted mainly by visceral adipose tissue and has been linked to obesity. Visfatin has insulin mimetic properties. Visfatin also play an important role in the development of several chronic diseases and inflammation.

Aim of the study: To evaluate serum visfatin concentrations in Basrah women of different body weights to determine the relationships with obesity and diabetes mellites in women in Basrah city.

Methodology: In this study, 60 women of different body weights were chosen between October and February, from Basrah University staff and students. They were divided into two groups according to their body mass index (BMI). Anthropometric measurements were recorded for all participants. Blood samples were collected to assay the biochemical parameters, including the levels of visfatin, insulin, glucose and lipid profile.

Results: Obese women shows significantly higher visfatin level than lean women. Furthermore, obese women showed significant increase of total cholesterol (T.C), triglyceride (T.G) and low-density lipoprotein-cholesterol (LDL-C) than lean women. However, obese women had significantly lower high-density lipoprotein-cholesterol (HDL-C) than lean women. Whereas, no significant differences of glucose and insulin levels between two group.

Conclusions: The results of this study revealed that visfatin levels were increased in obese women. This suggests that visfatin levels strongly associated with obesity.

Key words Visfatin, Obesity, Adipose tissue, Adipokines

1. Introduction

Obesity is chronic medical condition characterized by excessive fat accumulation in body (Blüher,2020). It is one of the most important factors that lead to many metabolic complications include type 2 diabetes, insulin resistance and cardiovascular disease (Barazzoni et al.,2018).

The spread of obesity globally has risen significantly among developing and, more recently, countries with medium and low income (Mancuso and Bouchard,2019). The World Health Organization (WHO) reports that in 2016, about 2 billion people were overweight, with 650 million meeting the obesity criterion (Jiménez et al.,2020). In the United States, the Middle East and Europe, in particular, the highest level of obesity is found and the lowest in East Asia and Sub-Saharan Africa (Balistreri et al.,2010).

Obesity is measured by using Body Mass Index (BMI) that determined by dividing the weight of the person in kilograms by their square height in meters, therefore, individuals can be classified into three categories, normal (BMI= 18-24.9 kg / m2), overweight (BMI= 25-29.9 kg / m2) and obese (BMI= 30 kg / m2) (Berthoud and Klein,2017).

There are many reasons that contributes to the evolution of obesity such as genetic variation, individual and environmental factors. Moreover, the prevalence of obesity is often influenced by racial disparities such as sex, age and race (Hales et al.,2020; Alqarni,2016).

The main source of fatty acids (FFA) in the fasting state is adipose tissue which is used for energy use and heat production (Balistreri et al.,2010). Adipose tissue also recognized as large endocrine and paracrine organ in human body which is secretes hundreds of bioactive molecules called adipokines (Zhang and Sairam,2014). These molecules are proteins secreted mainly by adipocytes and have role in several function in the body including energy metabolism, glucose homeostasis, inflammation, insulin resistance, immunity, appetite and satiety (Unamuno et al.,2018).

Visfatin hormone, the subject of our current research, is one of the important adipokines secreted from adipose tissue. Visfatin was first described by Fokohara in 2005, this hormone is predominantly found in visceral fat of obese mice and humans (Makhoumi et al.,2014). Visfatin has insulin mimic properties, its play an important role in the homeostasis of energy, glucose metabolism and inflammation by regulation the production of some inflammatory cytokines including tumor necrosis

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factor-a (TNF-a) and interleukin-6 (Yu et al.,2018). Visfatin is also implicated in the pathogenesis of multiple metabolic disorders such as obesity, diabetes mellitus (DM), blood pressure and insulin resistance (IR) (Zhang et al.,2019).

2. The aim of the study

Evaluate visfatin levels in obese women. Study the relationship between visfatin and insulin. To establish the relationship between visfatin and biochemical parameters.

3. Methods

Study population

In this study, a total of 60 Iraqi women samples aged from 25 - 55 years were collected from Basrah University's staff and students during the period from October 2020 to February 2021. A brief explanation of the project was explained to the participants before sample collecting. Written informed consent has been obtained from all participants before their inclusion. The medical histories of the study population and some required data such age and geographical area was obtained by direct interview with women by using a questionnaire.

Study design

The participants were divided into two main groups according to their BMI. The first group I including 30 lean women with BMI range (18-24.9), the second group including 30 obese women with BMI more than 30. The exclusion criteria for were the presence of any chronic diseases, endocrine diseases, treatment with any medication, pregnancy, and irregular menstrual cycle.

Anthropometric measurements

Anthropometric measurements, including body weight, height, and waist and hip circumferences, were measured. BMI values were calculated by dividing the person's weight in kilograms to height in meters square, and the waist-to-hip ratio (WHR) was calculated by dividing the waist circumference to the hip circumference in centimeters.

Serum preparation

Five ml of venous blood was collected in the morning between 8:00 and 10:00 after an overnight fasting, and placed in sterilized serum separation tube (gel tube). Leave it for a period (about 10 minutes) until the clot formation is occurred. After clot formation, the samples were placed in centrifuge (3500 rpm for 10 minutes at room temperature) to obtain the serum. The serum obtained were withdraw and placed in Eppendorf safe-lock tubes (1ml) which used for dividing the samples before storage in deepfreeze at (-20°) until the time of assay.

Biochemical analysis

Glucose and lipid profile concentrations were measured by enzymatic colorimetric method, by using commercial Kit (COBAS INTEGRA 400 plus, Roche, Germany). Enzyme-linked immunosorbent assay kits (ELISA) were used to determine serum visfatin and insulin levels.

Statical analysis

The data were statistically analyzed using SSPS software and the significance of the observed differences, associations, or calculations was determined at p-value <0.05. Chi^2 statistical test was used to investigate the significance of associations, Kruskal-Wallis and Mann-Whitney tests were used for differences between the groups of non-parametric data, and Spearman's test to examine nonparametric correlations.

4. Results

Table 1 shown that BMI and WHR were highly significant ingrease (p=0.000) in obese than control group. However, the results appeared that T.C, LDL, T.G and VLDL had significant increase in obese group than control (p=0.010, p=0.002, p=0.017 and p=0.016) respectively. Moreover, significant decrease of HDL in obese than control group (p=0.012). While, there was no significant differences of glucose concentrations (p=0.929) between two group. In table 2 visfatin level had significant increase in obese group than control (p=0.011). In contrast, there was no significant differences of insulin (p=0.160) between two group.

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Table 1 Comparison between group I and group II regarding BMI, WHR, glucose and lipid profile.

Variables	Group I Control n=30		Group II Obese healthy n=30		P value
	Mean ±SD	Median	Mean ±SD	Median	
BMI (kg/m²)	23.380 ± 1.6658	24.050	34.140± 3.1399	33.200	0.000*
WHR	0.7857 ± 0.0606	0.8000	0.8663± 0.07761	0.8850	0.000*
Glucose (mmol/l)	93.963 ± 6.2184	92.800	95.617± 11.8287	93.550	0.929 NS
Total cholesterol (mmol/l)	180.063± 31.7482	182.000	206.202± 35.7653	204.150	0.010*
Triglycerides (mmol/l)	82.157± 37.2205	78.800	107.493± 50.3963	88.700	0.017*
HDL-C (mmol/l)	48.210± 8.8651	48.050	41.580± 10.4861	40.750	0.012*
LDL-C (mmol/l)	92.967± 23.5520	95.450	115.647± 27.0835	120.100	0.002*
VLDL (mmol/l)	16.433± 7.4358	15.750	21.493± 10.0788	17.700	0.016*

* Significant; P-value <0.05 NS; not significant

Table 2 Comparison between group I and group II according to visfatin and insulin levels.

Variables	Group I Control n=30		Group II Obese healthy n=30		P value
	Mean ±SD	Median	Mean ±SD	Median	
Visfatin (ng/ml)	6.083 ± 1.7046	5.950	8.797± 9.4173	7.050	0.011*
Adiponectin (ng/ml)	56.047± 10.180	54.350	54.603± 8.1799	54.450	0.647 NS
Leptin (ng/ml)	8.080± 8.8648	4.350	46.327± 23.2231	42.450	0.000*
Insulin (µIU/ml)	14.897± 5.1885	13.400	18.003± 10.1209	15.050	0.160 NS

* Significant; P-value <0.05 NS; not significant

5. Discussion

One of the most frequent medical disorders is obesity. Obesity is characterized by low-grade inflammatory responses and increased oxidative stress (Wnuk et al.,2020). Obesity has developed into a hazardous condition that involves a variety of interventions, treatments, and preventions. Adipokines

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are small polypeptide growth factors released primarily by white and brown adipose tissue adipocytes such as visfatin, adiponectin, leptin and resistin (Gui et al.,2017).

Adipokines are hormones that can influence a variety of physiological and pathological processes, particularly those linked to immune and inflammatory activities. Skeletal muscle, kidney, pancreas and immune systems, can all benefit from adipokines. Adipokines' extensive impacts may explain (at least in some part) the systemic issues that are commonly linked with obesity (Saeidi et al., 2021).

The results of this study shown that there were significant increases of visfatin in obese group in comparison with control group. On the other hand, data was showed no significant differences in the level of insulin between groups.

Several studies agreed with this study results, its clarified that obesity causes increase the release of visfatin from adipocytes. Mabrouk was found significantly higher levels of visfatin in obese diabetics compared to healthy normal weight group (Mabrouk et al.,2013).

Moreover, another research was conducted that serum visfatin concentration was significantly raised in obese mice than control (Naz et al.,2017).

Alnowihi et al., (2020) and Berndt et al., (2005) were recorded that those obese women showed significantly higher visfatin than lean women. Furthermore, another study illustrated that serum level of visfatin was significantly higher in obese women when compared to controls (Zahorska-Markiewicz et al,2007; de Luis et al.,2008; Kamińska et al.,2010). Haider et al., (2006) and Garcia-Fuentes et al., (2007) also found significantly higher visfatin levels in patients with morbid obesity in comparison with lean individuals.

The hypothesis that this resulted from a compensatory mechanism developed in response to impaired insulin action, which confirms insulin mimetic effect of visfatin. This theory seems to be confirmed by other studies, which demonstrated that plasma visfatin concentration was dependent on the degree of insulin resistance. However, it should be noted that the relationship between serum visfatin level and insulin resistance remains unclear and studies revealed conflicting results (Shaker et al.,2011; Cheng et al.,2011; Picu et al., 2017).

In contrast, several studies demonstrated that obese subjects had significant lower visfatin levels compared to subjects with normal body weight (Pagano et al.,2006; Jian et al.,2006). Another research was reported no significant differences of visfatin concentration between obese and lean subjects (Hofsø et al.,2009).

Although the statistical analysis shown no significant differences in insulin level between all group, the data was recorded elevated in mean of insulin in obese more than control group (18.003 and 14.897) respectively. These elevated insulin levels may be due to increased insulin resistance due to insulin over secretion to overcome the tissue resistance (Schultz et al, 2013).

The data also shows that BMI and WHR were highly significant ingrease in obese than control group. However, the results appeared that T.C, LDL, T.G and VLDL had significant increase in obese group than control. Moreover, significant decrease of HDL in obese than control group. While, there was no significant differences of glucose concentrations between two group

Many previously studies revealed similar results. BMI and WHR had significant increase in obese subjects in compared with control group, the levels of T.C, T.G and LDL-C were significant increase in obese group than control while HDL-C degrease in obese than control group (Ayman et al.,2019).

Alnowihi et al., (2020) and Berndt et al., (2005) were recorded that those obese women showed significantly higher lipid profile than lean women. In contrast, obese women had significant lower HDL-C than lean women. Moreover, HDL shown significant decrease in obese group than control (Zahorska-Markiewicz et al,2007).

Moreover, (Baltacı et al., 2016) was conducted that the variables BMI, WHR, T.C, LDL-C, T.G) were significant increase in obese group in comparison with control. Conversely, HDL-C level was significantly decrease in obese subjects than lean group.

Furthermore, previous study on obese children was revealed that BMI, T.C, LDL-C, T.G) were significantly higher in obese group than control, HDL-C level was significantly decrease in obese subjects than normal weight group (Li et al., 2013).

On the other hand, several studies were appeared different result. There were no significant differences of T.C, LDL-C and T.C concentrations between obese and control group (Zahorska-Markiewicz et al,2007).

The results also shown no significant differences of glucose concentrations between obese and control group. This conducted was agreed with several studies. However, there were no significant differences of fasting glucose concentrations between obese and control group (Zahorska-Markiewicz et al,2007).

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Similarity, no significant variation of fasting glucose between obese and control group (Mabrouk et al.,2013).

Conclusions

Visfatin levels was higher in obese and diabetic women than lean women. It is difficult to decide if obesity intensifies these effects or if it is the opposite.

Recommendations

Make further investigations are being carried out to determine the levels of visfatin hormone in both gender and at various ages. Their relationship to obesity and role of visfatin in development and diagnosis of obesity DM. We prefer to conducting genetic studies on visfatin in large population samples of obese individuals and functional characterization of the genetic variations.

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