

Effect of metabolic syndrome on hormone level Visfatin and the level of blood sugar in groups of women in the city of Samarra

Jehan Naji Shaker¹, Asma Hassan Jumaa²

¹Fatat Al-raafidayn Intermediate school

²Department of Biology, College of Education, University of Samarra, Iraq

Abstract

The current study aimed to study the effect of metabolic syndrome on a hormone level Visfatin and the level of blood sugar in groups of women, as this study was conducted in the period from (1/10/2022 - 1/12/2022) in the city of Samarra, and the groups of women included a total of (90) blood samples from females whose ages ranged between (18- 40) years old, where the study was divided into two groups, the first included (60) obese women, while the second group included (30) women of normal weight. The results of the study showed There is a significant decrease in the level of the hormone phosphatidine in the serum of a group of obese women compared to a group of normal weight women. And it showed that the level of phosphatidine decreased in people who suffer from the metabolic syndrome, That there is a significant increase in the level of the hormone phosphatidine in obese women in the case of severe obesity, while there are no significant differences in obese women in the case of obesity of the first and second degrees, which included the samples of the current study, and that the level of the hormone rises in the case of the presence of adipose tissue at very large levels, and we find A significant increase in the level of serum glucose in the group of obese women compared to the group of normal weight women.

1- Introduction

What we eat determines our attitude, diet, and nutrition reflects on the health, growth, and development of the individual. However, lifestyle changes such as eating habits and inactive lifestyle that emerged during the past few decades led to a rapid increase in obesity rates and associated metabolic imbalances. A[1] Since metabolic syndrome (Metabolic Syndrome) group of Metabolic disorders include visceral obesity, insulin resistance, glucose intolerance, and hypertension The pathophysiological studies of the metabolic syndrome are increasing Metabolic syndrome provides a practical tool for identifying patients with increased risk of cardiovascular disease and type 2 diabetes [2].

Phosphatine is a protein hormone with a molecular weight of 52 kDa discovered in 2005 AD. It was originally described as a substance secreted predominantly by visceral adipose tissue, bone marrow cells, active lymphocytes, liver cells, and skeletal muscle cells. It was also found to be present in In other fat depots in the body such as perivascular fat and pericardial fat [3] Phosphatine binds to the receptors of the hormone insulin, so insulin works in cells, as it stimulates the taking of glucose and its entry into fat cells and muscle cells and prevents its exit from liver cells, so its action is associated with the modification of insulin sensitivity and insulin resistance [4]

Phosphatine plays a role in the pathogenesis of cellular metabolic syndrome, impaired glucose tolerance, impaired fasting glucose, diabetes mellitus, cardiovascular disease, and obesity. [5] Its levels are positively associated with insulin concentration, and cellular metabolic syndrome. While the levels of phosphatidine decrease with the improvement of insulin resistance and the decrease in body weight, as the increase in plasma phosphatidine compensates for insulin resistance and hyperglycemia, and plays major roles in programmed cell death associated with obesity [6].

And the lack of consumption of glucose by fat cells reduces the storage of fat and causes an increase in the liberation of fat in addition to the fats present in the blood. Because of the inability of the body to completely oxidize fatty acids, it will lead to their accumulation in the blood in the form of ketone bodies that come out with urine.[7] This leads to kidney failure and heart disease[8] Therefore, obesity is one of the causes of high glucose and type 2 diabetes[9] Where is the incidence rate T2DM in people who suffer from the metabolic syndrome is 5 times more, and this happens because of the food style and personal life that contribute to weight gain and obesity.[10].

2-Sample collection

Collected (90) a sample of females only whose ages ranged between (18-40) years, which included (60) samples of females suffering from metabolic syndrome who had a BMI of more than (30), and (30) a sample of the control group whose average BMI was It has (18-24.9), which is within the normal range.

3- Collecting blood samples

blood samples were drawn (5) ml of brachial vein at different times (4-5) hours after eating meals, taking information through a questionnaire, and taking physical measurements (weight, height, waist circumference, hip circumference) and blood samples were placed in glass test tubes containing gel And free of anticoagulant, for a period of (10-15) minutes, then it was placed in a centrifuge for the purpose of separation at a speed of (3000) for a period of (10) minutes to obtain the blood serum and placed in Appendroff tubes for the purpose of conducting the required tests in the study And it was kept in the freezer (Deep Freezer) at (-20) degrees Celsius.

The concentration of phosphatine was calculated according to the straight line equation as shown in the figure (1-1).

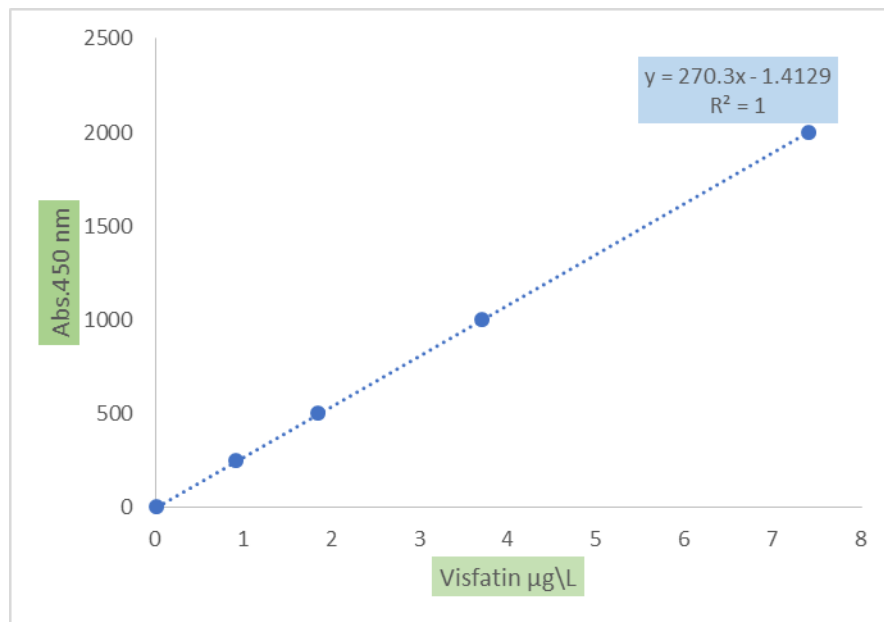


Figure (1-1) The straight-line equation for the concentration of the hormone phosphatine

4-Estimation of glucose concentration in the blood serum

Determination of glucose concentration in blood serum

Glucose concentrations were estimated using ready-made analysis kit(Kit), where blood sugar was estimated using the (Trinder method), where blood sugar is oxidized by the enzyme glucose oxidase GoD to hydrogen

peroxide and chloronic acid, through the interaction of hydrogen peroxide H₂O₂ with phenol and 4-amino-antipyrine and this step is done In the presence of the peroxidase enzyme, where the pink dye Quinone imine is formed (reagent evidence)[11].

Pipe	Bla	Sam	Stand
Reagent	1000	1000 liter	1000
Wa	10		
Sam		10	
Stand			10

Schedule (1-1) shows an estimate of the glucose concentration in the blood serum

Where the samples were mixed and incubated with temperature 37 ° for 10 minutes, after which the absorbance was read and the glucose estimate was calculated according to the following equation:

$$\text{Glucose mg/dl} = \frac{\text{Abs(Assay)}}{\text{Abs(standard)}} * \text{standard(100) concentration}$$

5-Results and discussion

5-1 Measurement of the concentration of the hormone phosphatidine

The results from Figure (1-2) showed that there was a significant decrease in the level of phosphatidine in the serum of a group of obese women compared to a group of normal-weight women. , which showed that the level of phosphatin was decreased in people with the metabolic syndrome, which indicates that the hormone phosphatin cannot be considered as a new inflammatory adipokinokine for the metabolic syndrome.[12]

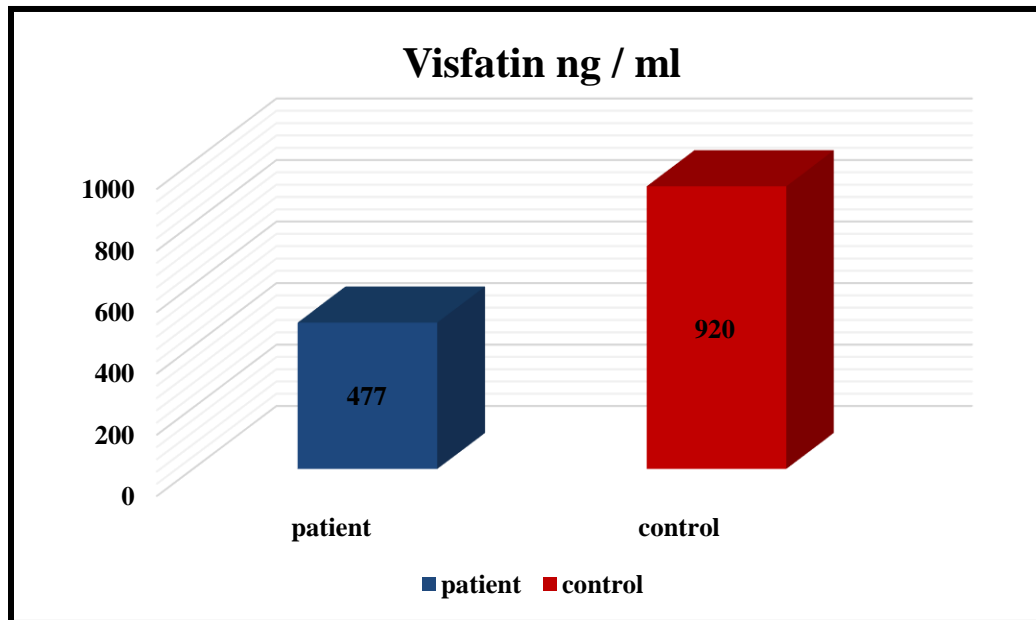
The results of the current study also agreed with each of the students Where the results of the current study agreed with a number of previous studies[13] [14].

This is due to the high level of glucose In the blood, there is a defect in the secretion of insulin and the failure to perform its functions completely, and the reason for this is due to damage to the beta cells. Insulin-producing B-cell in the pancreas, as well as a defect in the cellular receptors for insulin, in addition to the development of insulin resistance in the body[15] All of which lead to an increase in glucose levels in the blood, and the breakdown of glycogen Glycolysis found in the liver, in addition to its presence in other fatty and protein sources, works to add more amounts of glucose. to the bloodstream[16].

5) Which showed no association between the level of phosphatidine and the metabolic syndrome.

It can be said from previous studies that there is a significant increase in the level of the hormone phosphatidine in obese women in the case of severe obesity, while there are no significant differences in obese women in the case of obesity of the first and second degree who included the samples of the current study, that is, it is possible to explain these results to The level of the hormone rises in the presence of adipose tissue at very high levels.

This was confirmed by the study[17] Which showed that phosphatidine is secreted from visceral adipose tissue and has an insulin-mimetic effect, so it is associated with obesity, especially central obesity.



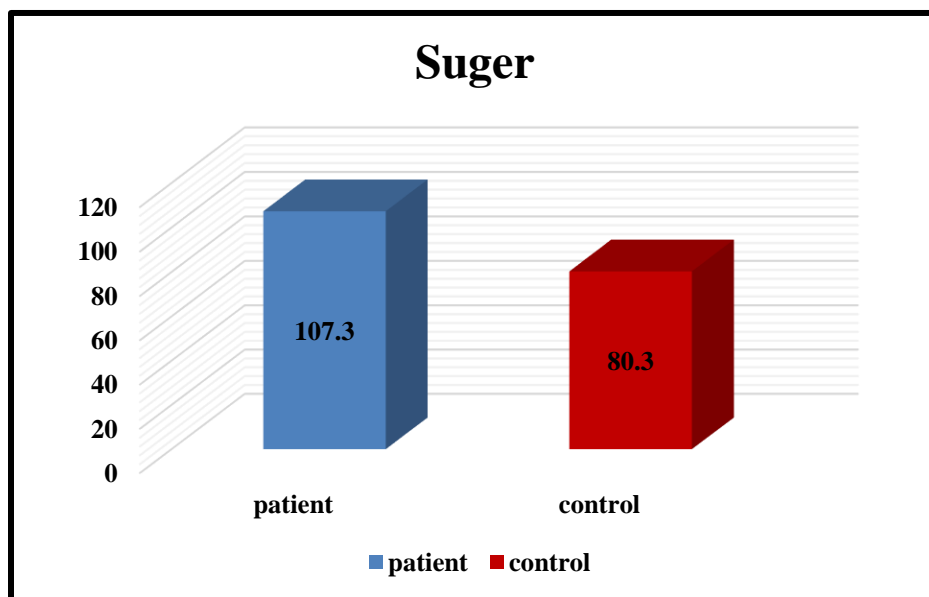
The shape (1-2) The level of phosphatine hormone in the blood serum of the samples under study

Results of the current study varied with outcome[18]Which showed that the level of the hormone is much higher in obese women compared to women of normal weight, and this increase in the level of the hormone in obesity may be aAn anti-regulatory system that prevents an increase in glucose. As phosphatidine is most associated with obesity and fat accumulation[19] [20]Especially the visceral tissue, which is considered more harmful than the subcutaneous adipose tissue, because it secretes many substances that lead to irregular accumulation of fat, causing obesity.[21]. It was found that weight loss in individuals who suffer from obesity can control the secretion of these substances, as the hormone phosphatine prevents the secretion of glucose in the liver by acting similarly to insulin, where glucose absorption increases in fat cells and muscle cells, which leads to its accumulation in the form of triglycerides in the body. fat cells[22]Thus, the elimination of visceral adipose tissue depends on the cells' sensitivity to insulin.[23]This leads to avoiding many of the problems caused by metabolic syndrome, including type 2 diabetes, high blood fats and blood pressure.

Results of the current study varied with study[24]Which showed that phosphatine levels are higher among obese individuals compared to non-obese individuals.

5-2Glucose concentration measurementGlucose concentration

The results of the current study showed a significant increase in the level of blood glucose in the group of obese women, compared to the group of normal-weight women, as shown in the figure (1-3).



The shape (1-3) glucose level in the blood serum of the samples under study

soI agree-The results of the study h-Aya with A-D from previous studies[25] [26]

References:

- Pasupulati, AK Reddy, BG (2013). Nutrition & metabolic syndrome. Rev Endocr Metab Disord 14:217. DOI 10.1007/s 11154-013-9267
- Thaman, R. & Arora, G. (2013). Metabolic Syndrome: Definition & Pathophysiology- the discussion goes on ! . J Phys Pharm Adv 2013,3(3):48-56.
1. Malamitsi-Puchner A, Briana DD. (2013). Visfatin as an Adipokine. Adipokines 2016 Apr 19 ; 144: 249.
 2. Sun Y, Wu Z, Wei L, et al. (2015). High-Visfatin levels in women with polycytic ovary syndrome: evidence from a meta-analysis. Gynecological endocrinology. 2015 Oct 3;31(10): 808-14.
 3. Davut Baltacı1, Mert Can Tuncel2, Mert Cetinkaya2, Muhammet Talha Gunduz2, Zemze Ozbey1, Ozlem Admis3, Ismail Hamdi Kara1, H& an Ankarali4. (2016). Acta Medica Anatolia, Volume 4 Issue 2 .
 4. Qun Cheng1,2, Weipin Dong1, Lei Qian1, Jingcheng Wu1 & Yongde Peng1. (2000). Visfatin inhibits apoptosis of pancreatic b-cell line, MIN6, via the mitogen-activated protein kinase/phosphoinositide 3-kinase pathway. Diabetes Research Laboratory, Department of Endocrinology & Metabolism, Shanghai Jiaotong University Affiliated First People's Hospital, 100 Hainin Road, Shanghai 200080, People's Republic of China 2 Shanghai Geriatric Institute, Fudan University Affiliated Huadong Hospital, Shanghai 200040, People's Republic of China (Correspondence should be addressed to Y Peng; Email: pengyongdetx@medmail.com.cn)
 5. Rorsman, P. Review (2005). Insulin secretion: function and therapy of pancreatic beta-cells in diabetes. British Journal of Diabetes & Vascular Disease. Vol. 5, No. 4, July. 187-191. 6.
 6. Uma, K. and Michael, W. (2001). Glycohemoglobin: A primary predictor of the development or reversal of complications of diabetes mellitus. Clinical Chemistry. 47 ; 1157-1165.

7. Galicia-Garcia, U., Benito-Vicente, A., Jebari, S., Larrea-Sebal, A., Siddiqi, H., Uribe, KB, ... & Martín, C. (2020). Pathophysiology of type 2 diabetes mellitus. *International J. of molecular sciences*, 21(17), .6275.
8. Al-Attaby, AKT, & Al-Lami, MQD (2019). Role of calcium-regulating hormones, adipocytokines and renal function test in the progression of type 2 diabetes mellitus in a sample of Iraqi patients. *The Iraqi Journal of Agricultural Science*, 50(1), 343–351.
9. Kaplan LA, Pesce AJ (1989). *Clinical Chemistry*, Mosby Ed.
10. M Foroughi, MJ Hosseinzadeh, S Zahediasl, F Hoseinpanah, AA Momenan, MR Esharaghivan. (2009). *Iranian Journal of Endocrinology & Metabolism* 11 (2), 151-223.
11. Kim JH, Cho GJ, Choikm, Han JH, yoohd, Kim sm. (2009). Korean jobs. The relationship between plasma Visfatin level, obesity & Metabolic syndrome in women - without Diabetes
12. Canan Ersoy al, Ganime Sadikoglu, Hasan Orhan. (2010). Body fat distribution no effect on serum Visfatin levels in healthy female subjects . volume 49, issue 3, March 2010, pages 275-278.
13. Lasiod, Mabrouka Abdul Qadir Abdul Latif. (2015). A study of the prevalence of metabolic syndrome among hypertensive patients in Ibrik and Al-Ghuraifa regions. Sebha University. Libya.
14. Soedling, H., Hodson, DJ, Adrianssens, AE, Gribble, FM, Reimann, F., Trapp, S., & Rutter, GA (2015). Limited impact on glucose homeostasis of leptin receptor deletion from insulin-or proglucagon-expressing cells. *Molecular metabolism*, 4(9), 619-630.
- 15.
16. Asmat, U., Abad, K., and Ismail, K. (2016). Diabetes mellitus and oxidative stress—a concise review. *Saudi Pharmaceutical Journal*, 24(5), 547-553.
17. Jang- young Kim, Dhananjay yadav, song vogueahn, sang - Baek koh. (2015). A prospective study of serum Adiponectin & regression of Metabolic syndrome: The ARIRNA NG study. *Biochemical & Biophysical Research* 201-communications 466(2) 201-205
18. Zahorska-Markiewicz, B., Olszanecka-Glinianowicz, M., Janowska, J., Kocelak, P., Semik-Grabarczyk, E., Holecki, M., ... & Skorupa, A. (2007). Serum concentration of visfatin in obese women. *Metabolism*, 56(8), 1131-1134.
19. Burstein M, Scholnic HR, & Sc& MR (1970). Rapid method for the isolation of lipoproteins from human serum by precipitation with polyanions. *Journal Clinical Lab. Invest*, 11(6), 583-595.
20. Friebe D, Neef M, Kratzsch J, Erbs S, Dittrich K, Garten A, Petzold-Quinque S, Bluher S, Reinehr T, Stumvoll M, Bluher M, Kiess W, Komer A. (2011). Leucocytes are a major source of circulating nicotinamide phosphoribosyltransferase (NAMPT) / PRE-B cell colony (PBEF) / Visfatin linking obesity & inflammation in humans. *Diabetologia*, 54, 1200-11 (2011), DOI: 10.1007/ s00125-010-2042-z.
21. Alirezn E, Alamdari A, Ali Z, Seerat E, Omid K, Manouchehr N, Alipasha M. (2011). Serum Visfatin is associated with type 2 diabetes mellitus independent of insulin resistance & obesity. p 154-158.
22. Fukuhara A, Matsuda M, Nishizawa M, Segawa K, Tanaka M, Kishimoto K. (2005). Visfatin: a protein secreted by visceral fat that mimics the effects of insulin. *Science* 307,426-30 DOI: 10.1126/ science.1097243. Epub 2004 Dec 16 .
23. Alirezn E, Alamdari A, Ali Z, Seerat E, Omid K, Manouchehr N, Alipasha M. (2011). Serum Visfatin is associated with type 2 diabetes mellitus independent of insulin resistance & obesity. p 154-158.
24. Irfan Younus, Shama Iqbal, Muhammad Shahid, Hamid Hassan (2023). Serum Visfatin levels in obese & non-obese individuals. *The Professional Medical Journal* 30 (01), 40-44.
25. Lasiod, Mabrouka Abdul Qadir Abdul Latif. (2015). A study of the prevalence of metabolic syndrome among patients with high blood pressure in Ibrik and Al-Ghuraifa regions. Sebha University. Libya.