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ORIGINAL ARTICLE

Evaluation of the Role of Vaspin and Insulin Resistance in Metabolic Syndrome

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ABSTRACT

Background: At least three of the following five medical factors must be present in order for someone to be diagnosed with metabolic syndrome. These conditions include abdominal obesity, high blood pressure, high blood sugar, high serum triglycerides, and low serum high-density lipoprotein (HDL). There is a correlation between having metabolic syndrome and having an increased risk of getting cardiovascular disease and type 2 diabetes. The percentage of older people who are members of racial and ethnic minority groups is growing at an alarming rate. Insulin resistance, metabolic syndrome, and prediabetes are conditions that are intricately connected to one another and share characteristics in common. It is believed that the condition is caused by an underlying disease of energy use and storage called MetS, which is closely related to the already existing weight gain, also known as obesity, and sloth. The blood levels of vaspin, insulin, and the HOMA IR value all increase as a result of this, and the levels of a number of other parameters also change as a result.

Patients and methods: A case control study was conducted in the form of a comparison between individuals who went to the consulting clinic for internal diseases during the study period, which lasted from December 2021 to February 2022. There were a total of 60 confirmed cases with metabolic syndrome and 30 healthy people, and their ages ranged from 24 to 67. Patients from the Salah El-Din Governorate and the surrounding areas had their insulin and vaspin levels tested using the Elisa instrument. Patients with metabolic syndrome were classified as having the condition based on their fasting blood glucose levels, triglyceride levels, HDL-C levels, systolic blood pressure, and waist circumference. This information was obtained from an organization affiliated with the International Diabetes Federation.

The results: The following findings emerged from the research conducted, and it was discovered that the level of vaspin and the HOMA IR value were significantly higher in individuals diagnosed with metabolic syndrome as compared to healthy people. And a negative correlation with HDL-C, a positive correlation between vaspin, HOMA IR, and insulin levels, as well as a positive correlation with the level of fasting glucose; moreover, HOMA IR value was significantly higher in patients with the metabolic syndrome than in the controls without the metabolic syndrome.

Keywords: metabolic syndrome, insulin resistance, and vaspin

INTRODUCTION

Patients with diabetes mellitus (D.M.) have an increased prevalence rate that is adjusted for age of 23.7 percent, metabolic syndrome (M.S.), defined as an elevated risk of type 2 diabetes and heart disease, is becoming a more well-known disorder. M.S. is defined by an increased risk of both conditions. The conditions of metabolic syndrome include diabetes and its precursor, prediabetes, as well as abdominal obesity, high cholesterol, and high blood pressure. According to statistics provided by the World Health Organization, the disorder affects more than one-quarter of the world's adult population. People who have this illness have a mortality risk that is two times higher than that of people who do not have this condition. People who suffer from metabolic syndrome have a five times increased risk of developing type 2 diabetes[1]. Because of the high prevalence of cardiac risk factors and the accompanying morbidity, coronary artery disease (CAD) is one of the most common causes of death in industrialized countries[2].

An endocrine organ, adipose tissue produces a number of biological mediators that regulate a variety of functions, including blood pressure, reproductive function, appetite, glucose homeostasis, angiogenesis, and immune system function, among other things[3]. Adipose tissue is responsible for the production of both pro- and anti-inflammatory mediators, which have an effect on inflammation both locally and systemically. One of these mediators is called adipokines, and these are hormone-like compounds that are produced by cells in white adipose tissue[4]. Adipokines, which are created by white adipose tissue (WAT), have been discovered as pleiotropic substances that lead to low-grade systemic inflammation in obese persons. White adipose tissue (WAT), in addition to operating as an energy storage tissue, is now recognized as an active endocrine organ[5]. Changes in metabolism brought on by an increase in body fat, particularly visceral obesity, when paired with a sedentary lifestyle, have the potential to bring about malfunction in adipose and muscle tissue. This would start off a chain reaction of events, the first of which would be insulin resistance. This would be followed by low-grade chronic inflammatio[6].

Vaspin One of the most important serine protease inhibitors (serpins) is Serpin A12 (OL-64; Vaspin, Visceral adipose-specific serpin, Ser A12)[7]. Visceral adipose tissue was the location where the gene vaspin (SERPINA12) was found in rats with type 2 diabetes. This was proven to be true in 2013 by using a crystal structure as evidence. Vaspin is helpful in bringing down the levels of sugar in the blood. Enhancing the half-life of Insulin is the primary focus of the mechanism that regulates glucose levels. This is in contrast to the traditional method of increasing glucose absorption via the insulin-mediated pathway[8]. Obesity, type 2 diabetes, metabolic syndrome, and atherosclerosis were all linked to increased expression of the vaspin mRNA and blood levels[9]. Vaspin improved glucose tolerance and insulin sensitivity while also acting as an insulin sensitizer and had anti-inflammatory characteristics. This was done in an effort to counteract the insulin resistance that is caused by obesity[10].

The number of people living with diabetes has skyrocketed in the previous 20 years, from an estimated 150 million in 2000 to an estimated 425 million in 2017, with projections suggesting that this number could climb to 629 million by 2045. It is now estimated that this epidemic will result in around 4 million more fatalities annually. Persons who with type 2 diabetes have a higher risk of dying than people without the condition because they are more likely to experience complications such as cardiovascular disease, renal failure, and infection as time goes on. Those with poor glucose tolerance were demonstrated to be at a high risk of developing the illness, and it was discovered in the middle of the 1980s that obesity and a lack of physical exercise were key risk factors that could be modified for type 2 diabetes[11]. Insulin resistance, often known as I.R., is thought to be the pathogenic state that underlies metabolic syndrome. Insulin resistance can be described as a diminished tissue sensitivity or responsiveness to circulating insulin. MetS is assumed to be caused by a pathogenic condition that is behind this spike in glucose level[12,13].

An impaired physiological response to insulin stimulation in organs such the liver, muscle, and fat is characteristic of insulin resistance. Insulin resistance is a contributing factor in the development of type 2 diabetes. Insulin resistance makes it more difficult for the body to get rid of glucose, which causes beta cells to produce more insulin and results in hyperinsulinemia as a compensatory reaction. Visceral obesity, hypertension, hyperglycemia, and dyslipidemia are The metabolic consequences of insulin resistance include, but are not limited to, obesity, hyperuricemia, elevated inflammatory cytokines, endothelial dysfunction, and a prothrombic state. During this activity, the causes and symptoms of insulin resistance, as well as the role that an interdisciplinary team plays in managing the condition, will be investigated[14]. The most frequent consequence of insulin resistance is type 2 diabetes; however, many people do not progress straight from "non-diabetes" to "diabetes," but rather, they establish an intermediate position in which glucose concentrations are not high enough to be classed as diabetic, but are also not normal; this state is known as "prediabetes" or "impaired fasting glucose." People who have prediabetes have an increased risk of developing type 1 diabetes, cardiovascular disease, and stroke, as well as eye difficulties[15].

PATIENT AND METHODS

Study Design: From the first of November 2021 to the end of February 2022, the city of Tikrit was the location of a case-control research that was conducted. Understudy included ninety people, including patients and controls, whose ages ranged from 24 to 67, and the study was carried out by and received approval from the board of the Tikrit University College of Medicine. These patients, as well as the control group, were seen at Salahaddin General Hospital. An interview was conducted with each of these individuals using a questionnaire developed by the researcher that inquired about their age, gender, current health status, and past medical experiences (previous diabetes, hypertension). In order to acquire all of the relevant information, the investigator conducted a personal interview. After the interview was over, a venous blood sample of 5 milliliters was taken from each participant.

The participants in this study had their waist circumference and blood pressure measured as part of this investigation.

MATERIALS AND METHODS

The standards set out by the International Diabetes Federation (IDF) in order to choose a patient sample - To be considered obese in the abdominal region, a man's waist circumference must be greater than 94 cm, and a woman's waist circumference must be greater than 80 cm (women). Furthermore, you must possess at least two of the following:

1. T.G. level that is greater than or equivalent to 1.7 mmol/L or therapy that is specifically intended for hypertriglyceridemia.
2. The HDL-c level is below 1.03 mmol/L in men and below 1.3 mmol/L in women, unless they are receiving specialized therapy.
3. A systolic blood pressure that is equal to or more than 130 mm Hg, or a diastolic blood pressure that is equal to or greater than 85 mm Hg, or the use of medication to manage hypertension.
4. A previously established diagnosis of diabetes type 2 or a fasting plasma glucose level that is equivalent to or higher than 5.6 mmol/L.

Excluded Criteria:

1. Pregnancy women
2. Patient on Insulin therapy
3. Patients have Immune diseases

Sampling: Five milliliters of blood was drawn from each research participant's vein. After centrifugation at 3000 rpm for 20 minutes, blood samples were deposited in disposable gel test tubes and centrifuged. The resulting serum was then aspirated with a mechanical micropipette and transferred into clean test tubes that were labeled. The samples were separated into two groups: one group was used for biochemistry measurements (including determining HDL C, Triglyceride, and fasting serum glucose), and the other group was used for ELISA measurements (including vaspin, and Insulin). Both groups were stored in the deep freeze at a temperature of –80 c0.

Statistical analysis: In order to do a computerized statistical analysis, the statistic program known as SPSS (Statistical Package for Science Services version 26) was utilized. The comparison was made with the use of the T-test probability (P-value), as well as correlation. P-values that were lower than 0.05 were considered to be statistically significant, whereas P-values that were higher than 0.05 were considered to be statistically insignificant.

RESULTS

Table 2: Patients with metabolic syndrome are divided into age groups.

AGE			
Class	Frequency	Percent	Cumulative Percent
26-35	3	5	5
36-45	10	17	22
46-55	23	36	58
56-65	24	42	100
Total	60	100	

The table shows an increase in the metabolic syndrome with age, and this appears clearly as shown in the table, where there is an increase in the number of patients with an increase in age according to the age range. metabolic syndrome 40% with the age group more than 56 years, followed by those within the age group (46-55) years 38%, then group (36-45) years17% and least rate metabolic syndrome in this study was 5%within the age group < 36 years.

Table 3: Serum vaspin mean and standard deviation in metabolic syndrome and without control group

Biochemical Parameter	Group Statistics	No.	Mean± S.D	P-Value
vaspin (ng/mL)	Patient	60	1.32±0.92	P =0.000006
	Control	30	0.48±0.35	

Within the focus of this research, an investigation into the connection between serum vaspin concentration and metabolic syndrome was carried out. The level of vaspin in a patient with metabolic syndrome was substantially greater than the level of vaspin in a control subject who did not have metabolic syndrome [mean+SD (1.32± 0.92 ng/ml vs. 0.48± 0.35 ng/ml)] respectively, when compared to the data presented in table (3).

A comparison of metabolic syndrome patients and controls using the homeostatic model assessment for insulin resistance (HOMA IR).

The connection between having a high HOMA-IR score and having metabolic syndrome. the HOMA IR value was considerably greater in patients diagnosed with metabolic syndrome when compared to values obtained from controls who did not have metabolic syndrome . When compared to the control group, those with metabolic syndrome had substantially higher mean blood levels of HOMA IR [mean±SD (3.36 ±2.87 vs 1.35± 0.51) correspondingly at a p-value of 0.0002, as shown in the table] (3).

Table 3: Compare the HOMA IR Mean and Standard Deviation of the metabolic syndrome group and the non-metabolic group.

Biochemical Parameter	Group Statistics	No.	Mean± S.D	P-Value
HOMA IR	Patient	60	3.366±2.87	P =0.0002
	Control	30	1.35±0.51	

Vaspin and insulin levels in the blood have a correlation with one another: Vaspin and insulin both have a positive and strong association with each other. As can be seen in figure(1), the P-value is 0.000002 and the R-value is 0.568.

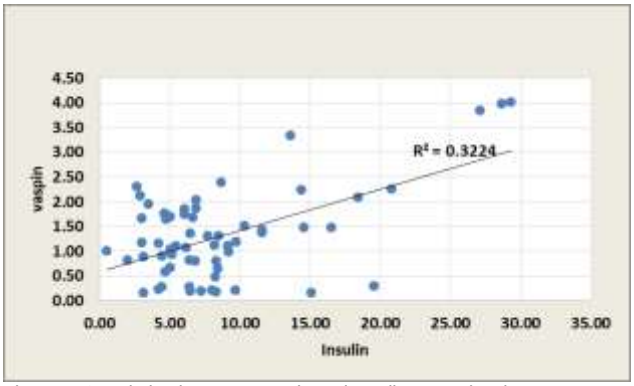

Figure 1: Correlation between Vaspin and Insulin serum levels.

Correlation between Vaspin serum level and HOMA IR value: The results of this study indicate that there is a substantial positive association between HOMA IR and Vaspin. The P-value was 0.000001, and the R-value was 0.583, as seen in the figure (2)

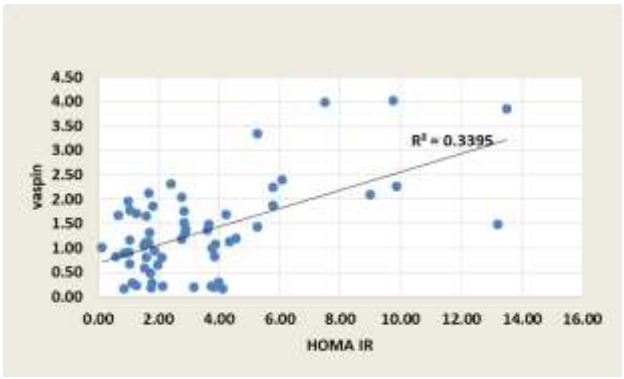

Figure 2: Correlation between Vaspin serum level and HOMA IR value.

DISCUSSION

The results of the examination are presented in the table below (1). The age group of more than 56 years old had the greatest prevalence of metabolic syndrome, at 40 percent, according to this study. Those in the age group of (46-55) years had the next highest rate, at 38 percent, followed by those in the age group of (36-45) years. 17 percent, while the group aged 36 years or younger had the lowest risk of metabolic syndrome at 5 percent, according to this study.

This research included participants ranging in age from 26 to 65 years old, all of whom were diagnosed with metabolic syndrome.

This line of reasoning, along with Hirode et al.[16], Farmanfarma et al[17].

The explanation for the rise in the prevalence of metabolic syndrome with increasing age is that it is related to a reduction in exercise and mobility as well as an increase in inflammation; this is one of the factors that contributes to obesity and insulin resistance.

According to the findings presented in table (2), our research indicates that there is a significant difference between the levels of vaspin found in patients and those found in controls.

Our findings have been confirmed by a number of other studies that looked at the circulation levels of vaspin in people who had metabolic syndrome. These studies found that the levels of vaspin were higher in people who had metabolic syndrome. Researchers have shown a correlation between the expression of vaspin in visceral and subcutaneous adipose tissue of obese patients and obesity, as well as glucose metabolism and insulin resistance[18].

The MetS components as a whole can't be connected to Vaspin, but she is linked to a portion of them. Vaspin is an independent MetS predictor. The primary factors contributing to this relationship are insulin resistance as well as chronic inflammation. Numerous investigations have shown that there is a correlation between the expression of the vaspin gene and the MetS components[19]. It has been shown that vaspin, when applied to endothelium and smooth muscle cells, can reduce the risk of atherosclerosis as well as obesity-related inflammation. Vaspin has qualities that are anti-inflammatory as well as anti-apoptotic. The inhibition of TNF-induced VSMC adhesion molecule synthesis by vaspin may lead to a reduction in lymphocyte adhesion through the production of reactive oxygen species (ROS) and the activation of nuclear factor-b and protein kinase C. Vaspin inhibits the generation of pro-inflammatory adipocytokines in adipose tissue, which contributes to the tissue's inherent anti-inflammatory properties (such as resistin and Tnf-)[20].

Correlation of Metabolic Syndrome with HOMA IR value.

HOMO-IR is an index that may be used as an indicator for the detection of insulin resistance or insulin sensitivity in type 2 diabetes. It is generated by analyzing the connection between fasting blood sugar and fasting insulin[21]. A patient with metabolic syndrome had a value for HOMA IR that was noticeably greater than that of the control group, which did not have metabolic syndrome. This was the conclusion drawn from the findings of the current investigation. Insulin resistance may play a crucial role in the development of metabolic syndrome as a pathological condition. The connection between insulin resistance and the myriad other metabolic syndrome problems is a complicated one that frequently works both ways. Therefore, insulin resistance is a contributing factor in both hyperglycemia and dyslipidemia, which both serve to exacerbate insulin resistance. If validated, the insulin resistance and pathogenesis of the metabolic syndrome and obesity-associated type 2 diabetes have major implications for the management and prevention of these metabolic disorders.

The outcome of our research is consistent with the findings of other clinical investigations carried out by Nolan et al[22], Gluvic, Zoran, et al[23].

Correlation Between vaspin and insulin-resistant. This study also indicated that there is a significant connection between high levels of vaspin serum, elevated levels of serum insulin, and HOMA IR. In this analysis, vaspin was discovered to be connected to insulin resistance in a favorable direction, a conclusion that was validated by previous studies. This discovery suggests that vaspin is responsible for improving glucose intolerance by functioning as an insulin-sensitizing agent.

A positive association was found between vaspin, insulin, and HOMA IR in the study that came before this one. Several researchers, including El-Lebedy et al.[24], Rashad et al.[25], and Yang et al[26].

Insulin resistance makes it harder for triglyceride lipolysis to be inhibited in diabetic patients whose condition is only partially under control. An increase in the breakdown of triglycerides allows for a greater entry of fatty acids into the liver. Insulin causes an increase in SREBP-1c activity, which in turn causes an increase in fatty acid synthesis enzyme expression in insulin-resistant persons. (A transcription factor known as ChREBP is also active in hyperglycemia, and its activation increases the transcription of enzymes involved in the synthesis of fatty acids.) Czech et al[27].

Based on these data, it appears that diabetics may have diminished SOD activity, which leads to increased oxidative stress and impaired beta-cell function. Low SOD activity has been linked to a number of negative health outcomes, including increased insulin resistance, hypertriglyceridemia, and dysfunctional beta cells. Diabetes patients have lower levels of superoxide dismutase (SOD), according to some study. Oxidative stress occurs when there is a disruption in the balance that exists between the production and clearance of reactive oxygen species (ROS). Beta cells are vulnerable to the effects of oxidative stress because they do not have antioxidant defense enzymes. Ma et al[28].

Insulin sensitivity can be improved with the use of vaspin, in

addition to glucose tolerance. According to Aibara et al., insulin may thus have the ability to control insulin sensitivity in peripheral tissues via vaspin, which is released by the liver[29]. Giomisi et al. found that there is a negative association in their investigation[30].

CONCLUSION

Patients in this research had significantly higher vaspin and HOMA IR values compared to controls, suggesting that these biomarkers may be useful in the identification of metabolic syndrome. All these issues may be traced back to insulin resistance, making it the central symptom of metabolic syndrome.

REFERENCES

1. Al-Rubaye FG. Frequency of metabolic syndrome in a sample of adult diabetics in Baghdad. Iraqi J Community Med. 2011;24:12-16.
2. Montazerifar F, Bolouri A, Mozaffar MM, Karajibani M. The Prevalence of Metabolic Syndrome in Coronary Artery Disease Patients. Cardiology Research. 2016;7(6):202-208. doi:10.14740/cr507w
3. Booth A, Magnuson A, Fouts J, Foster MT. Adipose tissue: an endocrine organ playing a role in metabolic regulation. Horm Mol Biol Clin Investig. 2016;26(1):25-42. doi:10.1515/hmbci-2015-0073
4. Ouchi N, Parker JL, Lugus JJ, Walsh K. Adipokines in inflammation and metabolic disease. Nature Reviews Immunology. 2011;11(2):85-97. doi:10.1038/nri2921
5. Coelho M, Oliveira T, Fernandes R. Biochemistry of adipose tissue: an endocrine organ. Arch Med Sci. 2013;9(2):191-200. doi:10.5114/aoms.2013.33181
6. Dalamaga M. Interplay of adipokines and myokines in cancer pathophysiology: Emerging therapeutic implications. World J Exp Med. 2013;3(3):26-33. doi:10.5493/wjem.v3.i3.26
7. Kyrou I, Mattu HS, Chatha K, Randeva HS. Fat Hormones, Adipokines. In: Endocrinology of the Heart in Health and Disease. Elsevier; 2017:167-205. doi:10.1016/B978-0-12-803111-7.00007-5
8. Suliga E, Wawszczak M, Gluszek S. The roles of vaspin, chemerin, and omentin in the determination of metabolic syndrome. Medical Studies. 2018;34(2):160-177. doi:10.5114/ms.2018.76878
9. Yang HW, Huang YG, Gai CL, Chai GR, Lee S. Serum vaspin levels are positively associated with diabetic retinopathy in patients with type 2 diabetes mellitus. J Diabetes Investig. 2021;12(4):566-573. doi:10.1111/jdi.13385
10. Weiner J, Zieger K, Pippel J, Heiker JT. Molecular Mechanisms of Vaspin Action - From Adipose Tissue to Skin and Bone, from Blood Vessels to the Brain. Adv Exp Med Biol. 2019;1111:159-188. doi:10.1007/5584_2018_241
11. Gong Q, Zhang P, Wang J, et al. Morbidity and mortality after lifestyle intervention for people with impaired glucose tolerance: 30-year results of the Da Qing Diabetes Prevention Outcome Study. Lancet Diabetes Endocrinol. 2019;7(6):452-461. doi:10.1016/S2213-8587(19)30093-2
12. Gallagher EJ, Leroith D, Karnieli E. Insulin resistance in obesity as the underlying cause for the metabolic syndrome. Mt Sinai J Med. 2010;77(5):511-523. doi:10.1002/msj.20212
13. Raimi TH, Dele-Ojo BF, Dada SA, et al. Triglyceride-Glucose Index and Related Parameters Predicted Metabolic Syndrome in Nigerians. Metab Syndr Relat Disord. 2021;19(2):76-82. doi:10.1089/met.2020.0092
14. Freeman AM, Pennings N. Insulin resistance. In: StatPearls [Internet]. StatPearls Publishing; 2021.
15. Abbass LB, Ezzaddin F. Detection of insulin resistance in overweight and obese individuals. Zanco Journal of Medical Sciences (Zanco J Med Sci). 2011;15(1):74-79.
16. Hirode G, Wong RJ. Trends in the Prevalence of Metabolic Syndrome in the United States, 2011-2016. JAMA. 2020;323(24):2526. doi:10.1001/jama.2020.4501
17. Kalan Farmanfarma K, Kaykhaei MA, Adineh HA, Mohammadi M, Dabiri S, Ansari-Moghaddam A. Prevalence of metabolic syndrome in Iran: A meta-analysis of 69 studies. Diabetes Metab Syndr. 2019;13(1):792-799. doi:10.1016/j.dsx.2018.11.055
18. Choi SH. Association of Vaspin with Metabolic Syndrome: The Pivotal Role of Insulin Resistance (Diabetes Metab J 2014;38:143-9). Diabetes & Metabolism Journal. 2014;38(3):240. doi:10.4093/dmj.2014.38.3.240
19. Dimova R, Tankova T. The Role of Vaspin in the Development of Metabolic and Glucose Tolerance Disorders and Atherosclerosis. BioMed Research International. 2015;2015:1-7. doi:10.1155/2015/823481
20. Heiker JT. Vaspin (serpinA12) in obesity, insulin resistance, and inflammation. J Pept Sci. 2014;20(5):299-306. doi:10.1002/psc.2621
21. Baek W, Lee JW, Lee HS, et al. Concurrent smoking and alcohol consumers had higher triglyceride glucose indices than either only smokers or alcohol consumers: a cross-sectional study in Korea. Lipids Health Dis. 2021;20(1):49. doi:10.1186/s12944-021-01472-2
22. Nolan CJ, Prentki M. Insulin resistance and insulin hypersecretion in the metabolic syndrome and type 2 diabetes: Time for a conceptual framework shift. Diab Vasc Dis Res. 2019;16(2):118-127. doi:10.1177/1479164119827611
23. Gluvic Z, Zaric B, Resanovic I, et al. Link between Metabolic Syndrome and Insulin Resistance. Curr Vasc Pharmacol. 2017;15(1):30-39. doi:10.2174/1570161114666161007164510
24. El-Lebedy DH, Ibrahim AA, Ashmawy IO. Novel adipokines vaspin and irisin as risk biomarkers for cardiovascular diseases in type 2 diabetes mellitus. Diabetes Metab Syndr. 2018;12(5):643-648. doi:10.1016/j.dsx.2018.04.025
25. Rashad NM, Ahmed HS, Ashour WMR, Yousef MS. Association of vaspin gene expression and its serum level on the risk of ischemic stroke in type 2 diabetic Egyptian patients: Prospective case-control study. Biotechnol Appl Biochem. 2020;67(6):912-919. doi:10.1002/bab.1850
26. Yang HW, Huang YG, Gai CL, Chai GR, Lee S. Serum vaspin levels are positively associated with diabetic retinopathy in patients with type 2 diabetes mellitus. J Diabetes Investig. 2021;12(4):566-573. doi:10.1111/jdi.13385
27. Czech MP, Tencerova M, Pedersen DJ, Aouadi M. Insulin signalling mechanisms for triacylglycerol storage. Diabetologia. 2013;56(5):949-964. doi:10.1007/s00125-013-2869-1
28. Ma M, Liu H, Yu J, et al. Triglyceride is independently correlated with insulin resistance and islet beta cell function: a study in population with different glucose and lipid metabolism states. Lipids Health Dis. 2020;19(1):121. doi:10.1186/s12944-020-01303-w
29. Aibara D, Matsuo K, Yamano S, Matsusue K. Insulin induces expression of the hepatic vaspin gene. Endocr J. 2020;67(1):9-14. doi:10.1507/endocrj.EJ19-0276
30. Giomisi A, Kourtis A, Toulis KA, et al. Serum vaspin levels in normal pregnancy in comparison with non-pregnant women. Eur J Endocrinol. 2011;164(4):579-583. doi:10.1530/EJE-10-1020