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RESEARCH ARTICLE

Relationship between HOMA-Insulin Resistance and Serum Vaspin Levels in Individuals with Polycystic Ovary Syndrome

Charu Rathore*[1], Dr. Nita Sahi[2], Dr. Jairam Rawtani[3], Dr. Mukul Sharma*[4], Disha Sahi[5]
[1]Ph.D. Scholar, 2Senior Professor & Dean Academics (PMCH),
[3]Senior Professor, 4Assistant Professor, 5MBBS
[1,2,5]Department of Biochemistry, Pacific Medical College & Hospital, Udaipur; 3Dr. S N Medical College, Jodhpur, Rajasthan; 4Banas Medical College & Research Institute, Palanpur

*Corresponding Author
Charu Rathore

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Abstract: ***Aims & Background:*** PCOS, or polycystic ovarian syndrome, is regarded as a metabolic disease in addition to a reproductive endocrinopathy. Obesity, glucose intolerance, insulin resistance, hyperinsulinemia, and a changed lipid profile are all linked to it. It is believed to be the most common endocrine issue among women of reproductive age and one of the main causes of female infertility. Insulin resistance and gonadotropic dysfunction are the primary pathophysiological elements of PCOS, and they are both correlated with body mass index. Adipocytokines have proven to be quite useful in understanding the metabolic defects associated with PCOS. Vaspin (visceral adipose tissue-derived serine protease inhibitor) is an insulin-sensitizing adipokine, identified in visceral adipose tissue from Otsuka Long-Evans Tokushima Fatty rats. Vaspin has been proposed as a useful adipokine and potential treatment candidate for metabolic disorders due to its insulin-sensitizing properties during the hyperglycemic state and its protective effects in the vascular and adipose tissues. The purpose of the study is to assess blood vaspin levels and determine whether they are related to insulin resistance. ***Material & Methods***: Using The Rotterdam Criteria, we included 80 clinically diagnosed PCOS subjects with age-matched healthy controls, in the study. Anthropometric measurements were taken. Fasting blood glucose, insulin and serum vaspin levels were measured, and Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) was calculated. All data were analysed and presented as mean ± standard deviation (SD), with ANOVA and correlation(r) calculated using Pearson's Correlation Coefficient. ***Results:*** The mean of serum Vaspin level in PCOS (2674.67 ± 447.23) was higher than control (1346.35 ± 333.49) which was statistically significant. ($p<0.0001$).
Conclusion: In comparison to the controls, the PCOS groups had significantly higher levels of plasma insulin, fasting blood glucose, and HOMA-IR. In PCOS participants, there was a positive correlation between the serum vaspin level and HOMA-IR. PCOS women may be at risk for diabetes, atherogenesis, and steroidogenicity due to elevated serum Vaspin levels and their positive connection with HOMA-IR. Clinical Significance: Vaspin may be a useful diagnostic and prognostic inE3T5;P `dicator to stop PCOS complications.

Keywords: PCOS, Insulin, Vaspin, HOMA-IR, hyperandrogenemia, hyperinsulinemia, 6case-control study. Main Article.

INTRODUCTION

Polycystic ovary syndrome (PCOS) is a widespread endocrine disorder affecting 6-13% of women of reproductive age linked with metabolic complications. According to World Health Organization, PCOS affects approximately 116 million women (3.4%) globally as of 2020.[1] Although the exact cause of this condition is still unknown, the symptoms can vary greatly, but typically include 0020 biochemical or clinical hyperandrogenismUY,654UY,6542, oligo-amenorrhea, and polycystic ovaries on ultrasound, as stated in The Rotterdam Criteria, 2003.This diverse condition impacts numerous bodily processes, leading to a number of health issues such as infertility, irregular menstruation, hyperandrogenism, hirsutism, acne, obesity, metabolic syndrome, and autoimmune diseases. It is polygenic condition associated with psychological, metabolic and reproductive sequelae.[2] The onset and persistence of this condition are significantly influenced by insulin resistance (IR).[3] A synergistic link between hyperandrogenism and disturbed gonadotropin-releasing hormone (GnRH) pulsatility, likely accompanied by hyperinsulinemia, insulin resistance, and inflammation, is the suggested etiology of PCOS. The subtleties of these connections have not yet been completely explained, though.[4-6]

The pancreatic islets' beta cells produce insulin, an anabolic hormone, into the hepatic portal circulation, where it affects both the liver and other peripheral tissues. An insufficient reaction of target tissues, including the liver, skeletal muscles, and adipose tissues, to the physiological plasma insulin levels is known as insulin resistance (IR). Postprandial hyperinsulinemia, fasting hyperinsulinemia, and finally hyperglycemia signal the onset of insulin resistance.[7] PCOS is a metabolic disorder that show hyperinsulinemia and peripheral IR as its central features. Although not part of the diagnostic criteria for PCOS, IR is considered to play a key role in the

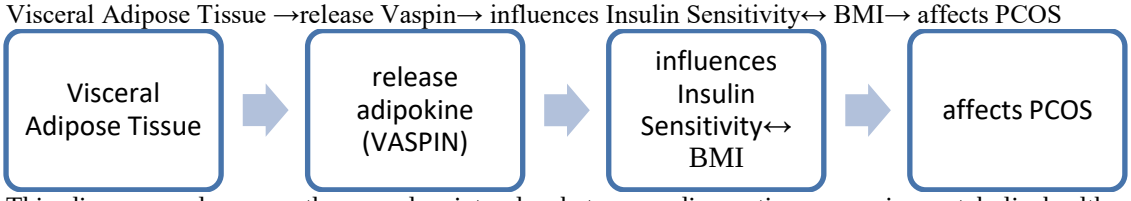
etiology of PCOS. Moreover, it is associated with metabolic abnormalities such as gestational diabetes, type 2 diabetes and cardiovascular complications.[8]
PCOS frequently manifests clinically and biochemically in women who are genetically prone to developing it due to weight gain and obesity. According to reports, 38% to 88% of women with PCOS are overweight or obese.[9–11]

Abdominal obesity, dysmetabolism, and reproductive abnormalities such irregular menstruation, unfavorable pregnancy outcomes, and infertility are all highly prevalent in PCOS. Increased incidence of obesity among PCOS women bring into light the role of adipokines that are known to influence the regulation of the hypothalamic–pituitary–gonadal axis or to locally change ovarian steroidogenesis.[12]

An insulin-sensitizing adipokine called Vaspin (visceral adipose tissue-derived serine protease inhibitor) was extracted from visceral adipose tissue in Otsuka Long-Evans Tokushima. Fatty rats are an animal model of type 2 diabetes mellitus (T2DM) with abdominal obesity.[13] According to reports, the degree of obesity and IR was correlated with both tissue expression and serum levels of vaspin.[14] Human vaspin mRNA has been detected in the epidermis, liver, pancreas, stomach, and visceral and subcutaneous adipose tissue. It is encoded by the SERPNA12 gene, which has six exons and five introns and is found on chromosome 14's long arm (14q32.13).[15] Vaspin has been proposed as a useful adipokine and treatment candidate for metabolic diseases because of its insulin sensitizing properties during hyperglycemic states and its protective effect in the vasculature and adipose states.[14] It contributes to the adipoinsular axis and may be linked to insulin resistance in obese individuals, such as those with polycystic ovarian syndrome and type 2 diabetes.

There has been only a few researches done in India[16-22] on VASPIN as it is one of the more recently identified adipokine. VASPIN has received the least attention, and as the majority of studies focus on obesity, metabolic syndrome, and diabetes mellitus, our goal is to examine its function and relationship to insulin resistance in PCOS patients; as both increased IR, a factor causing Diabetes and increased BMI, a factor causing Obesity seems to be present in most of the PCOS women.

Hence, this will be the first study trying to explore its relation directly with PCOS in our region.
Visceral Adipose Tissue →release Vaspin→ influences Insulin Sensitivity↔ BMI→ affects PCOS



This diagram underscores the complex interplay between adipose tissue, vaspin, metabolic health and reproductive disorders like PCOS.

MATERIALS AND METHODS

Inclusion Criteria: Clinically diagnosed patients with PCOS attending the obstetrics & gynecology OPD / clinics with age between 18-40 years were recruited. Diagnosis was based on the presence of any two out of three of the following criteria as proposed at the Rotterdam Consensus Meeting (2003)

- Oligomenorrhoea or amenorrhoea;
- Clinical hyperandrogenism and/or hyperandrogenaemia; and
- Polycystic ovaries on ultrasound

Exclusion Criteria: Patient with endocrine diseases (eg. diabetes mellitus, thyroid diseases etc.), chronic diseases, acute inflammation, on hormone replacement therapy (HRT), Pregnant and lactating women, alcoholics and smokers or who had undergone prior any form of Surgery especially of reproductive organs, liver and pancreas
Prior to the study, institutional ethics committee approval was acquired.
Every participant had their height, weight, and BMI measured using the following formula: BMI = weight (in kilograms) divided by height (in meters squared). Categorisation of BMI was as under:

PCOS, Insulin, Vaspin, HOMA-IR, hyperandrogenemia, hyperinsulinemia, 6case-control study.
Main Artic

Body Mass Index	Classification
< 18.5	Under Weight
18.5 - 24.9	Normal Weight
25 - 29.9	Over Weight
30.0 - 34.9	Obesity Class 1
35.0 - 39.9	Obesity Class 2
40 or above	Obesity Class 3

Using aseptic methods, 5 ml of venous blood was drawn from each subject's antecubital vein. Whole blood was collected in vials with anticoagulants for estimation of plasma glucose levels and the remaining blood sample was allowed to clot and Using commercially available reagents and kits, and following conventional protocol, separated serum was used for a variety of biochemical tests which were run on fully automated biochemistry analyzer Beckman Coulter AU680, Access 2 Immunoassay Analyzer, ELISA Analyzer. . Methods used for tests are as follows:

- Fasting plasma glucose- Hexokinase method
- Serum Triglycerides- Glycerol Phosphate Oxidase and Peroxidase Endpoint Method
- Serum Insulin - Chemiluminescence Immunoassay
- Serum Vaspin - ELISA technique
- Insulin Resistance was calculated using Homeostatic model assessment of Insulin Resistance (HOMA-IR) by formula-

$$IR = \frac{Fasting\ blood\ glucose\ (mmol/L) \times Fasting\ Insulin\ (\mu IU/mL)}{22.5}$$

(Fasting blood sugar in mg/dL was converted into mmol/L by multiplying with conversion factor of 0.05551).

Following table depicts the normal range and method used to assess the parameters:

S.No.	Parameter	Normal Range	Method
1.	Fasting Blood Glucose	70-110 mg/dl	Hexokinase
2.	Serum Triglycerides	<150 mg/dl	Glycerol Phosphate Oxidase and Peroxidase Endpoint Method
3.	Serum Insulin	2.30-26.0 µIU/ml	Chemiluminescence Immunoassay
4.	HOMA-IR	<2.5	Homeostatic Model Assessment of Insulin Resistance
5.	Vaspin	200-2500 pg/ml	ELISA

RESULTS

Table 1: Mean values of Anthropometric parameters among healthy controls and PCOS subjects

S.No.	Parameter studied	Healthy Controls	PCOS Subjects
1.	Age (years)	24.25 ± 4.07	25.75 ± 5.25
2.	Height (m)	1.56 ± 0.03	1.58 ± 0.05
3.	Weight (kg)	52.81 ± 3.79	63.76 ± 8.84
4.	BMI (kg/m^2)	21.68 ± 1.79	25.20 ± 3.01

Table 2: Mean values of Fasting Glucose, serum Insulin, HOMA-IR and Vaspin in healthy controls and PCOS

S. No	**PARAMETERS STUDIED**	**HEALTHY CONTROLS** (Mean ± SD)	**PCOS SUBJECTS** (Mean ± SD)			
			Normal Weight	**Overweight**	**Obese Class 1**	**Total**
1.	Fasting Blood Glucose (mg/dl)	81.50 ± 8.74	83.39 ± 10.28	89.36 ± 8.00	96.66 ± 8.45	87.07 ± 9.91[a]
2.	Serum Triglycerides (mg/dl)	104.77 ± 21.83	115.21 ± 18.79	137.05 ± 23.90	165.00 ± 10.77	128.77 ± 25.40[b]
3.	Serum Insulin (µIU/ml)	9.69 ± 2.13	10.56 ± 2.49	14.83 ± 2.02	16.4 ± 1.71	12.92 ± 3.19[b]
4.	HOMA-IR	1.95 ± 0.47	2.17 ± 0.56	3.23 ± 0.47	3.89 ± 0.31	2.78 ± 0.79[b]
5.	Vaspin (pg/ml)	1346.35 ± 333.49	2439.89 ± 412.14	2885.69 ±358.32	2895.5 ± 454.5	2674.67 ± 447.23[b]

subjects with different categories based on BMI

[a]p=0.0002- extremely significant
[b]p<0.0001- extremely significant

Table 3: Correlation of BMI, serum insulin and HOMA-IR with Vaspin among Control group and PCOS subjects

S.No.	PARAMETERS COMPARED	VASPIN	
		Healthy Controls (r)	PCOS Subjects (r)
1.	BMI	0.68	0.52
2.	INSULIN	0.42	0.52
3.	HOMA-IR	0.26	0.55

Pearson's Correlation Coefficient (r) was applied for calculation of correlation between different parameters.

Fig 1. Graph showing correlation between BMI and serum Vaspin

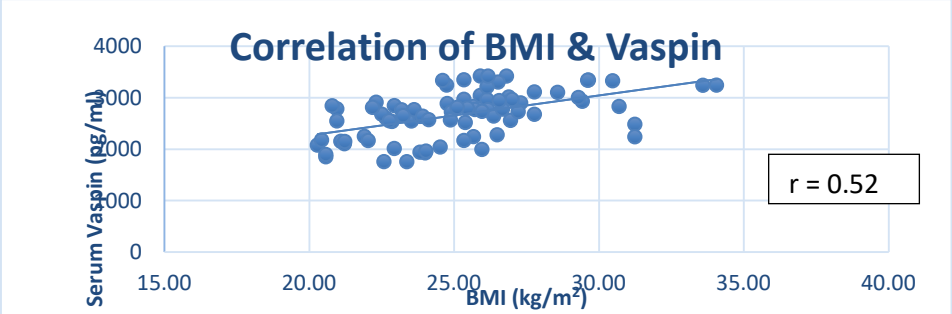


Fig 2. Graph showing correlation between serum Insulin and serum Vaspin

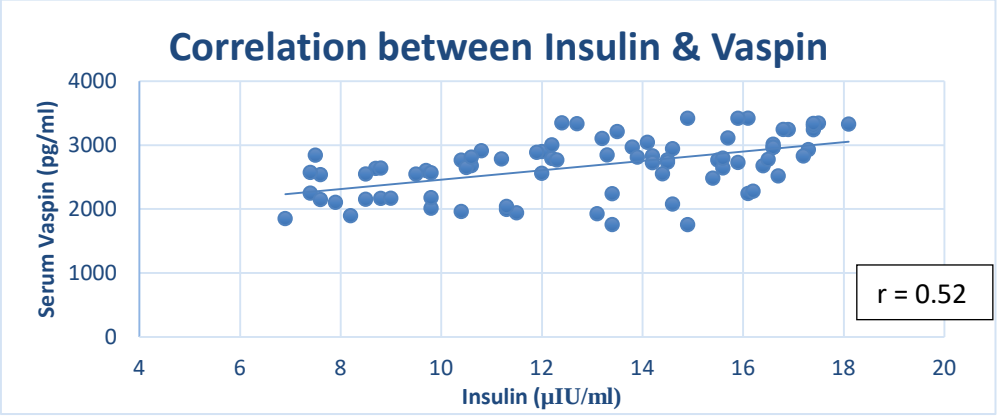
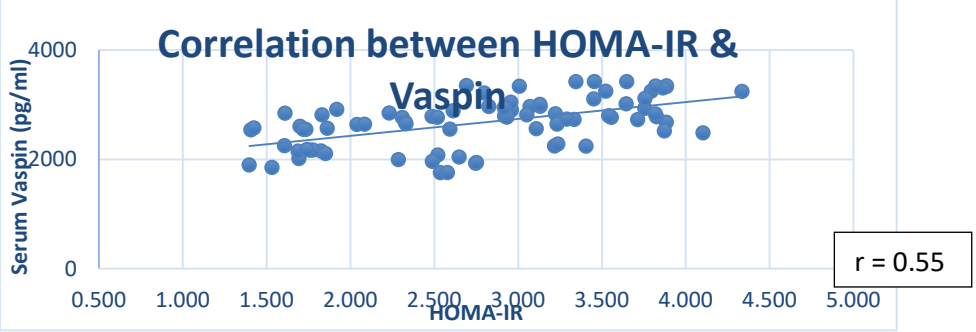


Fig 3. Graph showing correlation between HOMA-IR and serum Vaspin



Results: The PCOS group's mean and standard deviation of BMI, fasting blood glucose, serum triglycerides, serum insulin, HOMA IR, and serum Vaspin were statistically significantly ($p<0.0001$) greater than those of the healthy controls (table 1, 2).
When comparing PCOS patients to healthy controls, hyperinsulinemia and insulin resistance were noted.

BMI was more positively associated with Vaspin among healthy controls but such good association was not seen among PCOS subjects while insulin values were more correlated to vaspin among PCOS than healthy group. Among PCOS group women, serum Vaspin levels were found to correlate more positively with HOMA-IR with r = 0.55 than in control group where r=0.26 (table 3).

DISCUSSION

According to Teede et al. (2010)23, Polycystic Ovarian Syndrome is the most prevalent endocrinopathy affecting older women who are fertile and one of the main causes of low female fertility. However, its physiopathology is still poorly understood due in part to its highly variable phenotype (oligo-ovulation, hyperandrogenism, hirsutism, acne, alopecia, acanthosis nigricans, peripheral insulin resistance, hyperinsulinemia, metabolic syndrome, obesity, and type 2 diabetes) (Fauser et al. 2012).24 For instance, if we refer to 1990 NICHD definition, PCOS may be present as three phenotypes:

(i) Oligo+HA+Hirsutism, oligo-ovulation, and hyperandrogenemia;
(ii) Oligo+HA, oligo-ovulation and hyperandrogenemia, but no obvious hirsutism; and
(iii) Oligo+Hirsutism, oligo-ovulation, and hirsutism, but no detectable hyperandrogenemia.

The aforementioned criteria state that variations in the underlying metabolic pathophysiology are reflected in the clinical presentation of PCOS. On the other hand, the existence of the disorder's clinical and/or biochemical characteristics (such as hyperandrogenism, acne, and hirsutism) is more likely to be the result of other variables unrelated to PCOS's metabolic pathogenesis.25

Vaspin is a recently identified adipokine that has a strong correlation with both insulin resistance (IR) and obesity (Blüher 2012).26 Only the blood concentration and adipose tissue mRNA expression of vaspin are now known to be associated with PCOS (Tan et al. 2008, Escobar-Morreale et al. 2009, Cakal et al. 2011, Koiou et al. 2011a, Guvenc et al. 2016, Dogan et al. 2020).27 However, its role in PCOS has not yet been well investigated in India, and there is a limited information about it.

According to the findings of our study, women with PCOS had significantly higher serum vaspin levels than the control group. Additionally, elevated plasma HOMA-IR and glucose levels have been found, suggesting that insulin resistance may be the cause of elevated vaspin levels. Tan et al.'s28 investigation produced findings that were comparable. He found that obese women with PCOS had higher levels of vaspin mRNA and protein in their omental adipose tissue. In contrast, Dogan et al., 2020,29 suggested that women with PCOS had significantly higher levels of vaspin, but that the PCOS group did not have a statistically significant rise in serum vaspin levels despite a statistically significant increase in BMI. Franik G et al 2020, 30 in a study among PCOS women also reported a decrease in their serum vaspin level. Moreover, Hida et al31 concluded that increased vaspin level was caused by obesity and insulin resistance among the Otsuka Long Evans Tokushima Fatty rats.

A number of studies have been conducted on assessing circulating vaspin level in PCOS females and its correlation is explored with BMI and insulin, but the relation between Vaspin and IR remains unexplored. The result of our study on PCOS group showed that a positive association exists between their serum Vaspin level and HOMA-IR, that suggests the expression of vaspin in tissues is a defense mechanism against insulin resistance. It might suppress the production of IR-related genes, a phenomenon that is especially noticeable in abdominal fat; as it is already postulated that the raised Vaspin levels is a compensatory response against Insulin resistance (IR) and obesity and/or it could be a compensatory mechanism to maintain glucose tolerance and insulin sensitivity. Similar results were observed by Das M et al22 in their study conducted over 90 participants (including 60 PCOS & 30 healthy controls).

CONCLUSION

Our study's findings demonstrated that PCOS participants showed a positive correlation between serum VASPIN level and HOMA IR. As an adipokine that correlates positively with Insulin Resistance, VASPIN may serve as a marker for understanding this syndromic disorder and could assist in the developing specific therapeutic targets aimed at reducing the risk factors for diabetes mellitus, coronary artery disease, and infertility in patients with PCOS, especially considering the increase in obesity and insulin resistance associated with PCOS. Elevated Vaspin could be an indication of increased diabetogenic, atherogenic and steroidogenic risk among them. Given

the challenges in identifying PCOS, our study's findings imply that measuring these women's serum vaspin levels could be a unique way to diagnose PCOS, independent of BMI.

Clinical Significance: Vaspin may be a useful diagnostic and prognostic indicator to prevent PCOS complications.

List of Abbreviations:
ANOVA: Analysis of Variance
BMI: Body Mass Index
ELISA: Enzyme-linked Immunosorbent Assay
GnRH: Gonadotropin Releasing Hormones
HA: Hyperandrogenemia
HOMA-IR: Homeostatic model assessment for insulin resistance
HRT: Hormone Replacement Therapy
IR: Insulin Resistance
NICHD: Eunice Kennedy Shriver National Institute of Child Health and Human Development
OPD: Outpatient Department
PCOS: Polycystic Ovary Syndrome
r : Pearson's Correlation Coefficient
SD: Standard Deviation
T2DM: Type 2 Diabetes Mellitus

REFERENCES

1. Bulsara J, Patel P, Soni A, Acharya A. A review: brief insight into polycystic ovarian syndrome. Endocr Metab Sci.2021; 3:100085.
2. Escobar-Morreale HF. Polycystic ovary syndrome: definition, aetiology, diagnose and treatment. Nat Rev Endocrinol.2018; 14:270–84.
3. Amato MC, Vesco R, Vigneri E, Ciresi A, Giordano C. Hyperinsulinism and polycystic ovary syndrome (PCOS): role of insulin clearance. J Endocrinol Invest.2015; 38(12):1319–1326.
4. Cassar S, Misso M, Shaw C, Hopkin W, Teede H, Stepto N. Insulin resistance in Polycystic Ovary Syndrome: A systematic review and meta-analysis of euglycaemic-hyperinsulinaemic clamp studies. Hum. Reprod. 2016; 31: 2619–2631.
5. Nestler JE, Jakubowicz DJ. Decreases in ovarian cytochrome P450c17 alpha activity and serum free testosterone after reduction of insulin secretion in polycystic ovary syndrome. N. Engl. J. Med. 1996; 335: 617–623.
6. Stepto NK, Moreno-Asso A, McIlvenna LC, Walters KA, Rodgers RJ. Molecular Mechanisms of Insulin Resistance in Polycystic Ovary Syndrome. Unraveling the Conundrum in Skeletal Muscle? J. Clin. Endocrinol. Metab. 2019; 104: 5372–5381.
7. Manneras-Holm L, Leonhardt H, Kullberg J. Adipose tissue has aberrant morphology and function in PCOS: enlarged adipocytes and low serum adiponectin, but not circulating sex steroids, are strongly associated with insulin resistance. J Clin Endocrinol.2011; 96(2):304.
8. Korenberg KM, Melmed S, Polonsky KS, Larsen PR. Williams textbook of endocrinology. Mc Graw Hill 2008; 11 (16): 585.
9. Barber TM, McCarthy MI, Wass JA, Franks S. Obesity and polycystic ovary syndrome. Clin Endocrinol (Oxf). 2006; 65(2):137- 145.
10. Legro RS. The genetics of obesity. Lessons for polycystic ovary syndrome. Ann N Y Acad Sci. 2000; 900:193-202.
11. Balen AH, Conway GS, Kaltsas G, et al. Polycystic ovary syndrome: the spectrum of the disorder in 1741 patients. Hum Reprod. 1995; 10:2107-2111
12. Biomedicines10102503 https://doi.org/10.3390/ Heiker JT, Klöting N, Kovacs P, Kuettner EB, Sträter N, Schultz S, Kern M, Stumvoll M, Blüher M, Beck-Sickinger AG. Vaspin inhibits kallikrein 7 by serpin mechanism. Cell. Mol. Life Sci. 2013: 70, 2569–2583.
13. Suliga E, Wawszczak M, Gluszek S. The roles of vaspin, chemerin, and omentin in the determination of metabolic syndrome. Med Studies. 2018; 34(2):160-177.
14. Pilarski Ł, Pelczy ´nska M, Koperska A, Seraszek-Jaros A, Szuli ´nska M, Bogda ´nski P. Association of Serum Vaspin Concentration with Metabolic Disorders in Obese Individuals. Biomolecules 2023; 13:508. https:// doi.org/10.3390/biom13030508.
15. Suliga E, Koziel D, Ciesla E, Rebak D, et al. Associations between Vaspin Rs2236242 Gene Polymorphism, Walking time and the risk of Metabolic Syndrome. Balkan J Med Genet. 2019 Aug 28; 22(1):41-48.
16. Bharty R, Anand. Assessing Relationship between Vaspin and Biochemical Parameters and its Association with Diabetic Mellitus (T2DM): A Clinical Study. International Journal of Current Pharmaceutical Review and Research. 2023; 15(10); 791-795.
17. Rathwa N, Parmar N, Palit SP, Patel R, Ramachandran AV, Begum R. Intron specific polymorphic site of vaspin gene along with vaspin circulatory levels can influence pathophysiology of type 2 diabetes. Life Sciences.2020; 243: 117285. https://doi.org/10.1016/j.lfs.2020.117285
18. Sathyaseelan AJ, Adole PS, Wyawahare M, Saya RP. Assessment of Serum Vaspin Levels among Type 2 Diabetes Mellitus Patients with or without Acute Coronary Syndrome. Journal of Clinical and Diagnostic Research. 2016; 10(12): BC07-BC10. DOI: 10.7860/JCDR/2016/22417.8952
19. Kumari P, Bansal SK, Sharma DK, Yadav BK. Correlation of Serum Vaspin with Cardiovascular Risk Factors in T2DM. Int J Cur Res Rev. 2020; 12(5):13-18.
20. Harshitha S, Renuka P, Vengatesh M, Sriram V, Pavithra RB, Vinodhini VM.Correlation of Serum Vaspin Level with the Severity of Cardiovascular Risk in Patients with Acute Coronary Syndrome: A Cross-sectional Study. Journal of Clinical and Diagnostic Research. 2024; 18(8): BC09-BC12.
21. Sihag S, Mathur R, Bissa MS, Bhawani J. Assessment of Serum Insulin and VASPIN Levels Among Type 2 Diabetes Mellitus Patients with or Without Obesity: A Cross-sectional Analytical Study. J

Pharm Bioallied Sci. 2024; 16 (3):S2094-S2096. doi: 10.4103/jpbs.jpbs_62_24.
22. Das M, Devi N, Behera L, Jena P et al. Study of serum VASPIN level and its effect on insulin sensitivity in polycystic ovary syndrome patients, in southern Odisha. International Journal of Life Sciences, Biotechnology and Pharma Research. 2024; 13 (5):394-399.
23. Teede H, Deeks A, Moran L. Polycystic ovary syndrome: a complex condition with psychological, reproductive and metabolic manifestations that impacts on health across the lifespan. BMC Medicine.2010; 8: 41.
24. Fauser BCJM, Tarlatzis BC, Rebar RW, Legro RS, Balen AH, Lobo R, Carmina E, Chang J, Yildiz BO, Laven JSE, et al. Consensus on women's health aspects of polycystic ovary syndrome (PCOS): the Amsterdam ESHRE/ASRM-Sponsored 3rd PCOS Consensus Workshop Group. Fertility and Sterility.2012; 97(25):28–38.
25. Chang WY, Knochenhauer ES, Bartolucci AA, Azziz R. Phenotypic spectrum of polycystic ovary syndrome: clinical and biochemical characterization of the three major clinical subgroups. Fertility and Sterility. 2005; 83(6): 1717-1723. https://doi.org/10.1016/j.fertnstert.2005.01.096.
26. Blüher M. Vaspin in obesity and diabetes: pathophysiological and clinical significance. Endocrine.2012; 41:176–182.
27. Bongrani A, Mellouk N, Ramé C et al. Vaspin, a novel adipokine in woman granulosa cells physiology and PCOS pathogenesis? Journal of Endocrinology. 2021; 249:57–70.
28. Tan BK, Heutling D, Chen J, et al: Metformin decreases the adipokine vaspin in overweight women with polycystic ovary syndrome concomitant with improvement in insulin sensitivity and a decrease in insulin resistance. Diabetes. 2008;57:1501– 1507
29. Dogan K, Helvacioglu C, Baghaki S, Ekin M. Comparison of body mass index and metabolic parameters with serum vaspin levels in women with Polycystic ovary syndrome. Diabetes Metab Syndr 2020; 14: 137-139 [PMID: 32087564 DOI: 10.1016/j.dsx.2020.01.008]
30. Franik G, Plinta R, Madej P, Owczarek A et al. Circulating vaspin levels and nutritional status and insulin resistance in polycystic ovary syndrome.Ginekol Pol. 2020;91(5):251-255.doi:10.5603/GP.2020.0056. PMID: 32495930.
31. Hida K, Wada J, Eguchi J, et al: Visceral adipose tissue-derived serine protease inhibitor: a unique insulin-sensitizing adipocytokine in obesity. PNAS. 2005; 102: 10610-10615.