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ORIGINAL ARTICLE

Correlation between Insulin Resistance with severity of vasomotor symptoms in Postmenopausal Women

Ibrahim Adel ELnaptiti*[1], Mohamed S S Saad[1], Amany Mohamed Ahmed Abdelghany [2], Azza Hassan Abdelfattah[1]

[1] Internal Medicine Department, Faculty of Medicine, Zagazig, Egypt
[2] Obstetrics and Gynecology Department, Banha Teaching Hospital, Al-Qalyubia, Egypt

Corresponding author:
Ibrahim Adel ELnaptiti

Email:
ibrahim.adel693@gmail.com

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ABSTRACT:

Background: Hormonal fluctuations could affect vasomotor symptoms (VMS), particularly the decline in estrogen levels that accompany menopause. This research aimed to assess the correlation between insulin resistance and the severity of VMS in postmenopausal women. **Methods:** Seventy-four postmenopausal women (at least 12 consecutive months of amenorrhea) aged 40 years or more in this comparative cross-sectional study were classified regarding VMS into two groups (37 in each group): Group I: Postmenopausal women without VMS, Group II: Postmenopausal women with VMS. A menopausal Vasomotor Symptoms (MVS) survey was administered to all study participants. Laboratory investigations included Hemoglobin A1c, fasting blood glucose, and Insulin resistance index (HOMA-IR). **Results:** There were statistically significant positive correlations between HOMA-IR score and body mass index, Fasting Insulin, HBA1C, FBS, PBS, HDL, TGs, cholesterol, and estimated glomerular filtration rate to VMS (P<0.001) with a significant negative correlation between VMS and age. (P<0.05). HOMA-IR at cutoff value = 1.67 was a good predictor test for the severity of VMS among postmenopausal women. HOMA-IR, Age, BMI, and HbAlc were independent predictors for the severity of VMS among postmenopausal women (P<0.05) with Confidence interval (CI): (1.401 – 2.009, 0.564 – 0.917, 0.771 – 2.016, 0.564 – 0.917 respectively) and odd Ratios (OR) of (3.678, 1.719, 9.247, and 7.719 respectively).). **Conclusions** In postmenopausal women, there was a correlation between IR and VMS providing a strong basis for clinical diagnosis and treatment to improve the quality of life of postmenopausal Female patients.

Keywords: Insulin Resistance, severity, Vasomotor symptoms, Postmenopausal Women.

INTRODUCTION:

When ovarian follicular activity stops, menstruation stops permanently. This condition is known as menopause. The climacteric period begins with the onset of ovarian senescence and continues until the final stages of the process have taken place. A decline in ovarian steroid levels in the blood is the most notable of the many endocrine alterations that mark the ovaries' gradual decline in activity and, eventually, menopause. Vasomotor symptoms, urogenital atrophy, bone loss, and an increased risk of cardiovascular disease and metabolic syndrome can result from a decrease in estradiol levels caused by follicular exhaustion [1].

One of the primary factors contributing to the severity of vasomotor symptoms is hormonal

fluctuations, particularly the decline in estrogen levels that accompany menopause. Estrogen plays a central role in regulating body temperature and the body's response to temperature changes. As estrogen levels decline, the body's thermostat becomes dysregulated, leading to the hallmark heat surges of hot flashes. However, while hormonal changes are a key factor, they do not tell the full story [2].

The anti-inflammatory and slightly immunosuppressive properties of estrogen are well-documented. Its function as a pregnancy-related steroid hormone is essential. The anti-inflammatory effects of estrogen are largely responsible for the common observation that pregnant women with rheumatoid arthritis often experience relief or even remission of their symptoms. In inflammatory signaling pathways, proteinases are activated, leading to joint destruction in arthritis [3].

Among the most prominent and frequently reported symptoms experienced by postmenopausal women are vasomotor symptoms, which include hot flashes, night sweats, and flushes. Vasomotor symptoms are often described as sudden, intense sensations of heat that radiate through the body, frequently accompanied by sweating and flushing of the skin [4].

The inability of blood insulin to control glucose uptake and utilization by organs and tissues that are responsive to insulin is the hallmark of insulin resistance. When blood sugar levels increase, the pancreatic B-cells secrete more insulin, and the liver stops making glucose, as is typical under normal circumstances. On the other hand, people who are insulin resistant do not react to this signaling mechanism and, ironically, secrete more insulin and have higher hepatic glucose synthesis, both of which can cause or worsen hyperglycemia [5].

Changes in insulin signaling, elevated insulin levels, high cholesterol, and excess body fat all contribute to the development of insulin resistance. There may be a connection between insulin resistance and the severity of VMS in postmenopausal women, according to recent studies. A higher incidence and severity of VMS may result from insulin resistance interfering with the hypothalamus's regular functioning. This region of the brain is in charge of maintaining an adequate internal body temperature [6].

Only a few studies have evaluated the relationship between VMS and insulin resistance, In order to measure IR, this study utilized the HOMA-IR index. After menopause, women who received medical attention at Zagazig University Hospitals were studied to determine the relationship between IR and the severity of VMS. So, this study aimed to assess the correlation between insulin resistance and the severity of VMS in postmenopausal women because of a lack of attention to the significance of such associations.

METHODS

Between June 2023 and December 2023, this comparative cross-sectional study was carried out at the Endocrinology unit & Clinic of Internal Medicine Department, Zagazig University Hospitals on 74 postmenopausal women (at least 12 consecutive months of amenorrhea) aged 40 years or more.

The study was authorized by the research ethical council of Zagazig University's Faculty of Medicine, and all participants provided written informed permission. The research followed the guidelines laid out in the Declaration of Helsinki, which is part of the World Medical Association's Code of Ethics for Research Involving Humans. This study was carried out after the consent of the Institutional Review Board (IRB#10734/30-5-2023).

Cases with the following criteria were included: those aged 40 years or older who were considered to be postmenopausal if they had gone 12 months without menstruation in consecutive months as long as they agreed to participate (Supplementary Figure 1).

Cases with the following characteristics were excluded: cases who were younger than 40 years, Hysterectomy or bilateral oophorectomy patients, those with a history of chemotherapy or pelvic radiation for cancer or polycystic ovary syndrome, would not be eligible for this study, individuals undergoing surgical menopause, bilateral oophorectomy, or current hormone use for the following conditions: diabetes mellitus, dyslipidemia, overt thyroid, or any history of cardiovascular disease (including but not limited to previous myocardial infarction, angina, stroke, or peripheral arterial diseases), renal failure, or liver cirrhosis.

All patients were subjected to Full history taking involving age, name, sex, history of medical diseases, and family history. Detailed history of Menopausal Vasomotor Symptoms (MVS) (1): Every individual who took part in the study was given the MVS survey, which is a tool for the subjective evaluation of hot flashes.

The MVS survey was done with the intention of being brief, uncomplicated, straightforward, easy to understand, and administered with ease. Hot flushes and related conditions were the subjects of 39

questions spread across one page of the questionnaire [7].

In a mutually exclusive way, questions were asked. There were 35 questions in the survey that were closed-ended and could only be answered with a yes or no (nominal measures). One question asked for the name of the hormonal preparation used to treat hot flashes, and three questions required numerical measures showing the age or year when a specific hot flash-associated event happened. Each of the following aspects of hot flashes was covered in the MVS survey: the length of time these episodes lasted (questions 2–5), how often they occurred (questions 6-8), when they occurred (questions 9, 10), how long an episode lasted (questions 11–13), how intense they were (questions 14–16), and how they affected quality of life (question 17). Hot flashes were measured quantitatively and qualitatively in the questions given to each group.

Along with questions on different ways to quantify hot flashes, the MVS survey also asked about conditions that are often linked to them. These included things like hysterectomy (questions 19, 20), hormone therapy (HT) and the type of HT (questions 21-27), how long HT has been used (questions 28-32), how well HT has reduced hot flashes (questions 33-35), the age at which HT was first started (question 36), nonhormonal treatments for hot flashes (question 37), and a history of using hormonal contraceptives (questions 38, 39). Question 23 required women to match the names of their medications with a color chart that matched the hormone formulations now available, taken from the Physician's Desk Reference (PDR). This was done in case they forgot the names of their medications. Questions 24-27 asked for the dosage form of the drug, such as tablet, patch, cream, or injectable, if she did not recognize the hormonal preparation on the chart. In the ANSWER column, respondents indicated their answers to the survey questions. The survey administrator was given space to record supplemental remarks in a different column (NOTES) on the survey.

Laboratory investigations included Hemoglobin A1c, fasting blood glucose (FBG), fasting insulin (FINS), and Kidney function tests with an estimation of the glomerular filtration rate (GFR) and to check for proteinuria (albuminuria).

Insulin resistance index (HOMA-IR): For the HOMA-IR calculation, both groups had their fasting blood glucose (FPG) and insulin (FINS) levels checked. The insulin homeostasis model was used to calculate the IR index, which is calculated as HOMA-IR= FPG (mmol/l) × FINS (mU/L)/22. 5.

Statistical analysis:

We used IBM SPSS software package version 21.0 to examine the data that was fed into the computer. (New York: IBM Corp., 2012) Numbers and percentages were used to describe the qualitative data. To ensure distribution normality, the Kolmogorov-Smirnov test was employed. The quantitative data was presented using the most frequent metrics such as median, interquartile range (IQR), mean, standard deviation, and range (minimum and maximum). When testing categorical variables, researchers turned to the Chi-square test. When comparing group means, they turned to the independent samples t-test. When comparing several groups, they turned to the one-way analysis of variance (one-way ANOVA). The relevant IR-affecting factors were analyzed using multiple linear regression. Statistical analysis was made easier by log-transforming data that did not follow a normal distribution. The $\chi 2$ test was used to compare the rates. A statistically significant difference was defined as $P < 0.05$.

RESULTS:

The evaluated groups did not differ significantly in terms of age or parity. However, there were significant differences in terms of body mass index (BMI), with Group II having a higher BMI than Group I (53.0±2.83 vs 51.0±2.23), with a p-value of less than 0.001. (Table 1).

There were statistically significant differences in studied groups regarding (lipid profile, urea, Creatinine, IR, FBS, PPS, & HBA1C) ($p <0.001$) that were higher in Group II, however, eGFR was lower in Group II ($p <0.001$) (Table 2).

Menopausal symptoms scores according to menopause rating scale (Vasomotor symptoms score, Somato-vegetative symptoms, Uro-genital symptoms) and Overall MRS score(menopause rating scale) all were statistically significantly higher in Group II ($p <0.001$) (Table 3). There were positive Correlations between UACR, HOMA-IR, and VMS severity ($P<0.05$) (Table 4).

Statistically significant positive correlations were revealed between HOMA-IR score and body mass index, Fasting Insulin, HBA1C, FBS, PBS, HDL, TGs, cholesterol, and e FGR to VMS ($P<0.001$). It also shows a statistically significant negative correlation between VMS and age. ($P<0.05$) (Table 5).

The best cut-off value of HOMA-IR as a predictor

test for the severity of VMS was ≥1.67, with a sensitivity of 100%, specificity of 97%, positive predictive value of 98.6%, and negative predictive value of 100% (Table 6 and Supplementary Fig. 1). HOMA-IR at cutoff value = 1.67 was a good predictor test for the severity of VMS among postmenopausal women. HOMA-IR, Age, BMI, and HbAlc were independent predictors for the severity of VMS among postmenopausal women (P<0.05) with Confidence interval (CI): (1.401 – 2.009, 0.564 – 0.917, 0.771 – 2.016, 0.564 – 0.917 respectively) and odd Ratios (OR) of (3.678, 1.719, 9.247, and 7.719 respectively) (Table 7).

Table (1): Distribution of the studied groups regarding demographic data and clinical characteristic..

Variables	GroupI N=37	Group II N=37	Test of Sig	*p*
Age per years Mean ±SD	53.0±2.83	51.0±2.23	χ^2=0.580	0.876NS

Variable	Group I N=37 Mean±SD	Group II N=37 Mean±SD	F test (ANOVA)	P value
Height(m) Range	1.69±.04 1.58-1.75	1.66±.06 1.5-1.75	0.67	0.61 NS
Weight(kg) Range	74.5±6.8 60-85	81.9±12.6 60-110	1.85	0.12 NS
BMI Range	26.3±+1.7 23-28.8	29.6±3.1 25.4-35.9	4.8	<0.001 HS
Waist Range	111.1±6.9 102-125	123.1±4,6 115-131	16.1	<0.001 HS
pulse (beat/min) Range	82.7±6.3 72-92	84.1±6.1 70-92	0.31	0.86 NS
SBP(mmHg) Range	119±7.1 110-130	138.9±27 110-150	8.29	<0.003 HS
DBP(mmHg) Range	74.7±5.1 70-80	87.9±14.7 70-130	5.1	<0.001 HS

BMI: body mass index, SBP: systolic blood pressure, DBP: diastolic blood pressure. HS: high significant, NS: Non significant

Table(2): Biochemical characteristics and glucose homeostasis of the studied subjects.:

Variable	Group I N=37 Mean±SD	Group II N=37 Mean±SD	T test	P value
Cholesterol(mg/dl) Range	226.3±53 114.8-304.7	158.6±19.8 120-188.4	6.29	<.0.003 HS
TG(mg/dl) Range	116.7±44 75.5-267	99.4±24 60.3-163	12.7	<0..001 HS
HDL(mg/dl) Range	43.9±5.7 27-43	43.1±13 23.7-92	3.6	<0.008 HS
LDL(mg/dl) Range	168±53.9 49.6-251-9	95. ±5+20 54-127.5	7.75	<.0.001 HS
Urea (mg/dL) Range	22.74±7.73 20-26.5	20.84±4.78 17-22.5	1.85	0.12 NS
Creatinine (mg/dL) Range	0.850±0.151 0.7-1.35	0.526±0.170 0.6-0.95	10.03	<0.001 HS

Variable	Group I N=37 Mean±SD	Group II N=37 Mean±SD	T test	P value
eGFR (mg/mmol) Range	100.74±7.73 85-95	110±14.78 110-130	16.5	<.0.001 HS
			F test (ANOVA)	
Fasting Insulin(µIU/m) Range	5.6+±1.4 2.92-7.566	9.1±2.1 3.133-9.551	9.38	<0.001 HS
***FBS(mg/dl)* Range**	92.7±14.2 78-120	95.4+±15.4 80-125	60.3	<0.001 HS
***PPG(mg/dl)* Range**	119.1±13.9 99.3-160	122.5±9.4 105-140	71.7	<0.001 HS
HBA1C(%) Range	5.8±0.4 5.32-6.5	6.3±0.4 5.3-6.9	53.7	<0.001 HS
HOMA-IR Range	1.04±0.33 0.62-1.86	5.35±0.39 5.82-7.96	42.19	<0.001 HS

TG: triglycerides, HDL: High-density lipoprotein, LDL: Low-density lipoprotein, e GFR: estimated glomerular filtration rate, FBS: fasting blood sugar, PBG: postprandial glucose, HOMA IR: Homeostatic Model Assessment for Insulin Resistance

Table (3): Menopausal symptoms scores according to the menopause rating scale in in studied Groups.

Menopausal symptoms scores	Group I(n=37) Mean(SD)	Group II(n=37) Mean (SD)	T	p-value
Vasomotor symptoms score		8.03(3.22)		
Somato-vegetative symptoms	5.16(3.01)		9.9	<0.001 HS
Uro-genital symptoms	1.77(2.21)	3.77(2.21)	4.6	<0.001 HS
Overall MRS score(menopause rating scale)	6.93(5.22)	18.99(8.44)	7.8	<0.001 HS

Table (4): Correlation between HOMA-IR and severity of vasomotor symptoms in Group II.

vasomotor symptoms severity	HOMA-IR	R	P
mild (5–8)	2.55±0. 1	0.15	<0.05 (S)
moderate (9–15)	5.85±0.39	0.31	<0.05(S)
severe (more than 16 points)	8.05±0.59	0.29	<0.05(S)

Table (5): Correlation of severity of VMS in group II to other parameters.

	R	P	Sig.
Age	- 0.068	0.373	S
BMI	+ 0.527	0.0001	HS
HOMA-IR score	+ 0.329	0.0001	HS
FBS	+ 0.758	0.046	HS
PPG	+ 0.908	0.003	HS
Cholesterol	+ 0.288	0.0001	HS
TG	+ 0.329	0.0001	HS

	R	P	Sig.
HDL	- 0.462	0.0001	HS
LDL	+ 0.758	0.05	HS
Urea	+ 0.261	0.0001	S
Creatinine	+ 0.252	0.001	HS
eGFR	- 0.364	0.0001	HS
Fasting Insulin	+ 0.001	0.08	HS
HBA1C	+ 0.323	0.0001	HS

TG: triglycerides, HDL: High-density lipoprotein, LDL: Low-density lipoprotein, e GFR: estimated glomerular filtration rate, FBS: fasting blood sugar, PBG: postprandial glucose, HOMA IR: Homeostatic Model Assessment for Insulin Resistance, HBA1c: Hemoglobin A1c

Table (6): ROC Curve Analysis of HOMA-IR as predictor test for the severity of VMS.

Cut-off values	Sensitivity % (95% CI)	Specificity % (95% CI)	PPV % (95% CI)	NPV % (95% CI)	AUC (95% CI)
> **1.67**	100 %	97 %	98.6 %	100 %	0.658

Table (7): Logistic regression analysis for predictors for the severity of VMS in postmenopausal women.

	Regression Coefficient	SE	OR	95% CI	P
HOMA-IR	+ 0.517	0.092	3.678	1.401 – 2.009	0.0001
Age	- 0.330	0.124	1.719	0.564 – 0.917	0.008
BMI	+ 0.221	0.245	9.247	0.771 – 2.016	0.004
HbAlc	+ 0.485	0.761	7.719	0.564 – 0.917	0.008

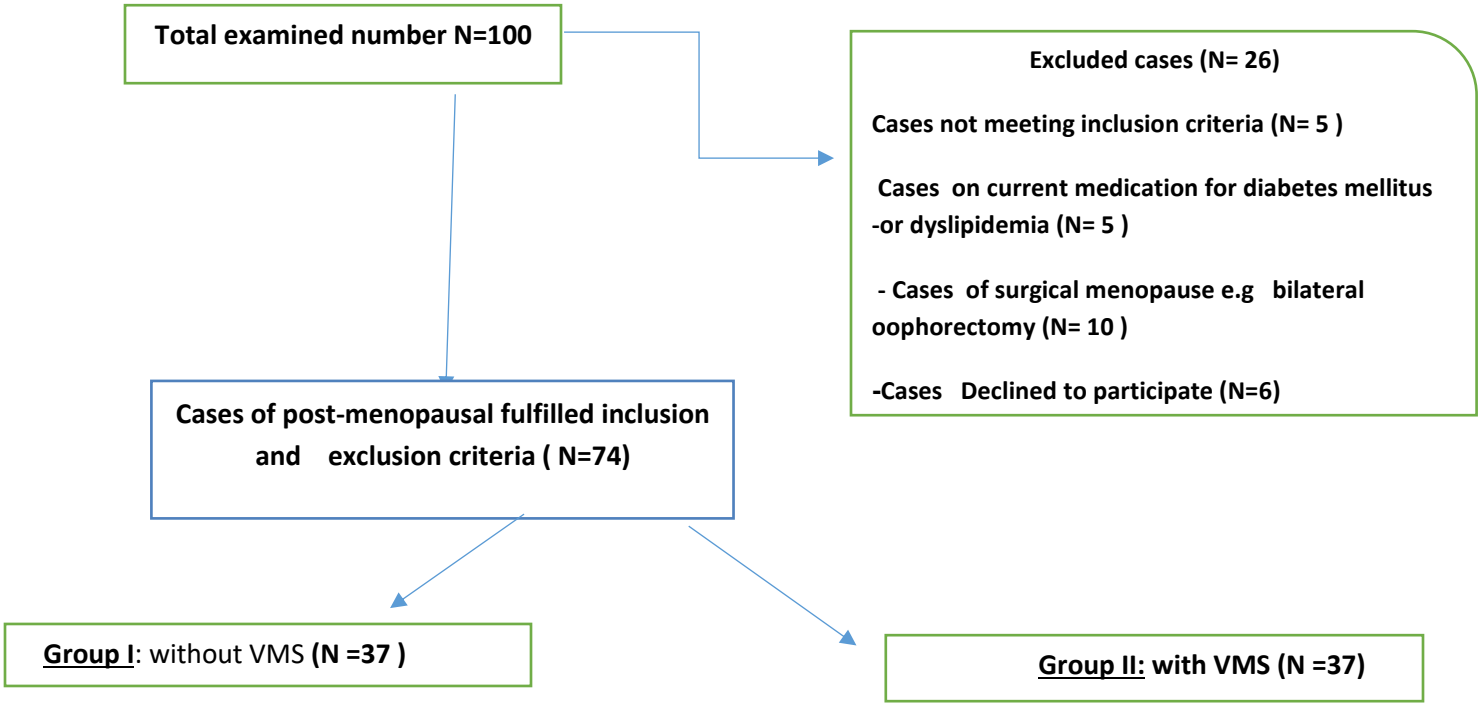

Figure 1: Flowchart of the recruited patients

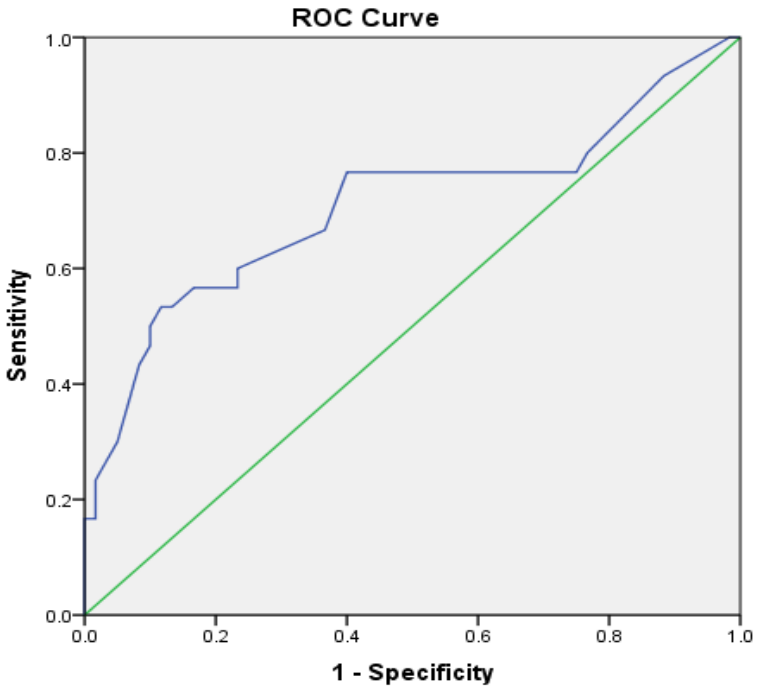

Fig. (2): ROC Curve of HOMA-IR as predictor test for the severity of VMS in postmenopausal women.

DISCUSSION:

Androgen bioavailability and the relative androgenicity of the body after menopause hypothalamus sensitivity decreases, and the hypothalamus thermoregulatory system narrows with age and estrogen deprivation [8].

The decline in estrogen levels that occurs after menopause can cause insulin resistance. On the other hand, progesterone has a more nuanced relationship with insulin, but there is evidence that it can impact insulin sensitivity as well. Lastly, the decline in progesterone after menopause impacts blood sugar levels [9].

Weight gain, one of the metabolic changes linked to menopause, appears to be more of a result of aging than menopause itself. In addition, Impairment in glucose metabolism was a result of changes in body composition, including an increase in fat mass, a rise in abdominal fat, and a decrease in lean body mass [9].

The HOMA-IR is a clinical research tool, but it is also one of the most popular IR index validation tools. It uses fasting insulin and glucose determinations. Other readily applicable broad population metrics are required for everyday clinical practice [10].

Regarding the Clinical characteristics of the studied groups in the present study, statistically significant differences were found between both studied groups regarding BMI, waist, SBP, and DBP that were higher in Group II ($p < 0.05$); the current study findings were inconsistent with the study results of Na et al. [11] who examined the correlation between insulin resistance and the ratio of urine microalbumin to creatinine in women who had gone through menopause. There were statistically significant differences in the study groups related to age, body mass index, systolic blood pressure (SBP), and diastolic blood pressure (DBP). Postmenopausal women exhibited greater levels of these variables, this could be attributed to difference in exclusion criteria study of Na et al. [11] had history of DM (type 2) but in the studied group were not so there was a slight difference in the results but +ve correlation between IR , and VMS.

These results were also correlated with studies of Jui-Kun Chiang et al. [12] and Antonio Gonzalez-Chavez et al. [13], who revealed that Obesity and intra-abdominal fat deposition are linked to increased

inflammatory cytokines and decreased adiponectin concentrations, which VMS exhibited with a high body mass index and a large waist circumference. Obesity may be a significant shared risk factor of both visceral muscle wasting (VMS) and insulin resistance since it raises core body temperature and may enhance the frequency and severity of VMS.

Regarding biochemical characteristics of the studied subjects in the current study, there were statistically significant differences in the studied groups regarding (lipid profile, urea, & Creatinine) ($p < 0.001$) that were higher in Group II. However, eGFR was lower in Group II ($p < 0.001$).

The present study findings were in line with Thurston et al. [14], who examined 3,201 women who were 42–52 years old when they entered the study. They were asked about their hot flush and night sweat frequency, as well as their height, weight, blood pressure, triglycerides, follicle-stimulating hormone, apolipoprotein A-1, apolipoprotein B, lipoprotein[a], and other biomarkers. The study followed these subjects for 8 years. After controlling for hormones, medicines, and cardiovascular risk factors, linear mixed models were used to assess the relationship between symptoms and lipids. They found that increased levels of triglycerides, apolipoprotein A-1, apolipoprotein B, and low-density lipoprotein were linked to vasomotor symptoms. Connections between hot flashes and the risk of cardiovascular disease should take lipids into account.

Consistent with previous population-based studies, the current investigation found elevated levels of LDL-C, apoB, and triglycerides. Vasomotor symptoms were linked to higher total cholesterol in a single large cross-sectional research [15].

Hot flashes and lipids were not linked in another trial with 150 women in excellent health [16]. Disagreements may arise for reasons that aren't immediately obvious, such as variations in sample size or the fact that some studies choose women who are low on cardiovascular risk factors but have severe symptoms. Lipids may have a role in vasomotor symptoms, but the exact mechanism is unclear.

The relationships were unchanged when controlling for the menopausal stage and E2, and the only linkages whose associations were diminished when controlling for FSH were those involving vasomotor symptoms and LDL-C. Hormones, then, accounted for just a fraction of the correlations seen here. Quite a few additional systems could be pertinent. Endothelial dysfunction and an unfavorable hemostatic profile are both suggested in females experiencing vasomotor symptoms. Additionally, vasomotor symptoms are associated with shifts in the balance of the autonomic nervous system, which favor an increase in sympathetic tone and a decrease in parasympathetic tone (Vasomotor symptoms) [14]. A profile related to cardiovascular risk. One last point: some have linked vasomotor symptoms to altered hypothalamic-pituitary-adrenal axis function. [17]. Thus, many mechanisms may link vasomotor symptoms to altered lipid profiles.

In terms of the glucose homeostasis parameter for the participants analyzed, the present study found that Group II had significantly higher insulin, IR, FBS, PPS, and HOMA-IR & HBA1C values ($p < 0.001$). Statistically significant differences were observed between the groups when looking at the postmenopausal women's data, which revealed that UACR levels increased considerably with rising HOMA-IR in women with VMS. a discovery that has the potential to form a solid foundation for therapeutic prevention and therapy.

Results from the current study did not match up with those from Na et al. [11] A study involving 104 postmenopausal women with type 2 diabetes examined the correlation between insulin resistance and urinary micro albumin creatinine ratio. The results showed that the study group had higher levels of HOMA-IR and UACR compared to the control group, and this difference was statistically significant ($P < 0.05$), this could be attributed to that the study of Na et al. [11] involved 104 postmenopausal women with type 2 DM examined, but our studied group had no history of medical disease (as exclusion criteria) so, the study didn't match up with Na et al. [11].

Research has demonstrated that ovarian shrinkage causes postmenopausal women to have much-decreased estrogen levels. Another finding that suggests a role for estrogen decline in the development of IR in postmenopausal women is that their IR levels are 44% [18].

We found a positive correlation between HOMA-IR and the intensity of vasomotor symptoms in Group II. The urine albumin-creatinine ratio (UACR) was the endpoint of the analysis. After menopause, the insulin resistance index (HOMR-IR) was determined, and the relationship between IR and UACR was examined. Results from the Pearson correlation study demonstrated a positive relationship between UACR and HOMA-IR and HbA, and they also discovered that the postmenopausal group had higher HOMA-IR and UACR levels than the control group. A more in-depth examination using multiple linear stepwise

regression revealed a favorable correlation between HOMA-IR, age, and UACR.

Regarding HOMA-IR as a predictor test for the severity of VMS in the present work: the best cut-off value of HOMA-IR was ≥1.67, with a sensitivity of 100%, specificity of 97%, positive predictive value of 98.6%, a negative predictive value of 100%. This was in agreement with Kwon et al. [19]; they used the homeostatic model assessment (HOMA) index to hypothesize a link between insulin resistance and vasomotor symptoms (VMS) in a cross-sectional investigation. The participants in the study were 1,547 postmenopausal women from Korea, ranging in age from 45 to 65. A menopause rating scale questionnaire was used to measure the intensity of VMS. A total of 885 individuals (57.2%) reported experiencing VMS to varying degrees, with an average age of 55.22±4.8 years. They found that the mean HOMA index went up as the severity of VMS went up, and they concluded that insulin resistance has to be taken into account to comprehend the connection between VMS and cardiometabolic diseases.

Concerning the distribution of cases in Group II) according to the degree of HOMA-IR, 35% of the cases were IR-L(Low), and 45 % of the cases were IR-M(medium). In comparison, 30 % of the cases were IR-H(higher) in Group II.

This was in agreement with Lee et al. [5]; they included 4,314 people (65+) without diabetes in a prospective community-based cohort and divided them into three groups according to their baseline HOMA-IR tertiles: low (n=1454), moderate (n=1414), and high (n=1446). Initial diagnosis of type 2 diabetes mellitus was the main result. A composite of macrovascular events, such as coronary artery disease, myocardial infarction, and stroke, as well as chronic kidney disease (CKD), were included as secondary outcomes. Hypertension, elevated cholesterol, and elevated hemoglobin A1c were all shown to be more common in the high HOMA-IR group. Compared to the other groups, those with a high HOMA-IR had a greater incidence of new-onset type 2 diabetes (5.8%) and chronic kidney disease (12.2%). No group had a lower or higher frequency of macrovascular events. A high HOMA-IR independently increased the probability of developing type 2 diabetes mellitus (odds ratio 1.86 [1.17-2.96]; p = 0.01) as well as chronic kidney disease (1.49 [1.12-1.98]; p = 0.01). Regardless of HbA1c, they found that High HOMA-IR was a strong predictor of new-onset type 2 diabetes and chronic kidney disease in non-diabetic people.

Limitations:

Since this study is a small-scale, Comparative cross-sectional study involving only 74 cases, there is bound to be some selection bias in the results. Adjusting for all potential confounders, such as the unknown intervention outside of Zagazig University Hospitals, is challenging. Second, our findings only apply to the Egyptian population. Further studies are needed with a large sample size that could produce significant results. Also, additional further research, such as prospective cohort studies or randomized controlled trials, is needed to establish causality and evaluate potential interventions to mitigate the severity of vasomotor symptoms in postmenopausal women with insulin resistance.

Author contribution: All authors contributed to the study. MSSS was responsible for selecting the subject, AMAA, AHA was accountable for laboratory revisions and analysis, IAE was responsible for data collection, statistical analysis, and initial writing, and AMAA was responsible for collecting the data of the studied cases and all shared for the formulation of the study design, editing, revision, and preparation of the final manuscript.

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CONCLUSIONS:

In postmenopausal women, there was a correlation between IR and VMS providing a strong basis for clinical diagnosis and treatment to improve the quality of life of postmenopausal Female patients.

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