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ORIGINAL ARTICLE

Evaluation of Zinc-Alpha-2-Glycoprotein (ZAG) Level as a Risk Factor in Patients with Metabolic Syndrome

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ABSTRACT

Background: raises the risk of cardiovascular disease, type 2 diabetes, and death by a considerable margin. Zinc-alpha-2-glycoprotein (ZAG), which is released by adipocytes, epithelium, and other tissues, is well-known due to its functions in energy expenditure and fat metabolism. Therefore, our aim was to use ZAG in early detection of metabolic syndrome, especially in central obesity, to reduce its risk and improve the outcome.

Methods: 120 subjects were assessed in the Internal Medicine Department's Endocrinology Unit outpatient clinic at Zagazig University hospitals as part of this case-control study. Three groups were formed: Group A: Control group, which consists of 40 individuals in good health. Group B: 40 centrally obese subjects without other problems. Group C: 40 patients with metabolic syndrome. Routine tests and anthropometric measurements were conducted for all participants. Enzyme-linked immunosorbent assay (ELISA) kits were used to measure serum ZAG.

Results: The control group showed the highest ZAG values, but the metabolic group showed the lowest. Serum ZAG ROC curve analysis in differentiating the healthy individuals from the other two groups (central obesity and metabolic syndrome): The ROC curve study of serum ZAG revealed an area under the curve (AUC) of 0.893 in differentiating metabolic syndrome from the central obesity group, and the serum ZAG area under the curve (AUC) was 0.792.

Conclusions: ZAG may play an important role in early detection and management of metabolic syndrome, especially in those with central obesity. Though its moderate specificity indicates the need for combined biomarker strategies to enhance diagnostic accuracy.

Keywords: Zinc-Alpha-2-Glycoprotein; Metabolic Syndrome; Obesity.

INTRODUCTION

A group of risk factors known as the metabolic syndrome includes low HDL-C levels, elevated blood pressure, elevated glucose and triglycerides, and central (abdominal) obesity. Type 2 diabetes mellitus (T2DM), cancer, chronic renal illness, cardiovascular disorders, and nonalcoholic fatty liver disease (NAFLD) are all made more likely by metabolic syndrome **[1].**

When at least three of the following five criteria are met, metabolic syndrome can be diagnosed: Blood pressure of 135/85 mm Hg or higher, fasting blood glucose of 100 mg/dl or more, abdominal fat, triglycerides of 150 mg/dl or higher, HDL cholesterol of 40 mg/dl or less in men and 50 mg/dl in

women, and an abdominal circumference of more than 102 cm in men and 88 cm in women are all indicators of abdominal obesity **[2].**

The intricate pathophysiological processes linked to metabolic syndrome must be thoroughly explained. The significant regional variation in metabolic syndrome prevalence highlights the role that environmental factors and lifestyle choices—such as a high-calorie diet and inactivity—play in the disease. The cytokines released by adipose tissue play a role in insulin resistance and endothelial dysfunction, two disorders that lead to the development of metabolic syndrome **[3].**

Among all the hypothesized pathways, the development of metabolic syndrome and cardiovascular disease seems to be primarily caused by insulin resistance, neurohormonal activation, and chronic inflammation **[4].**

Zinc-alpha-2-glycoprotein (ZAG) is a 41-kDa glycoprotein that belongs to the class I major histocompatibility complex (MHC) protein family. ZAG is released into numerous bodily fluids and found in a range of epithelia. It has been shown that ZAG acts differently from MHC class I molecules and does not bind peptides or beta2-microglobulin like class I MHC proteins do **[5].**

There is mounting evidence that the pathophysiology of obesity and its related consequences, such as metabolic syndrome, are significantly influenced by changes in the synthesis of adipose-derived protein components, such as ZAG. Androgens, progestins, and glucocorticoids are the main factors that control the expression of the ZAG gene in adipocytes **[6].**

The paracrine promotion of adiponectin production, the browning of white adipose tissue, and the modification of enzymes involved in lipogenesis and lipolysis are the mechanisms that underpin the strong correlation between ZAG and obesity to date. ZAG is also essential for regulating the insulin sensitivity of adipose tissue **[7, 8].**

Aim of the work: early detection of metabolic syndrome, especially in central obesity, to reduce its risk and improve the outcome by detecting ZAG serum level.

Research gap: Only a few studies have explored the association between serum zinc-alpha-2-glycoprotein (ZAG) levels and the metabolic syndrome, and the results have remained controversial. Additionally, all of these studies assume the metabolic syndrome criteria approved by the National Cholesterol Education Program-Adult Treatment Panel III (NCEP: ATPIII). Our study was based on the International Diabetes Federation's (IDF) criteria of metabolic syndrome (2006), which requires the presence of central obesity as a key component of metabolic syndrome. ZAG was closely associated with obesity. So, it is significant and necessary to investigate serum ZAG levels in metabolic syndrome patients, especially in metabolic syndrome patients diagnosed by using the IDF criteria.

METHODS

Participants:

The Endocrinology Unit, Internal Medicine Department, and Clinical Pathology Laboratories at Zagazig University Hospitals assessed 120 patients as outpatients between September 2024 and March 2025 as part of this case-control study. Patients were divided into three groups: Group A: Control group, included 40 healthy individuals matched by age and sex. Group B: Included 40 patients having central obesity with only 0-1 other criteria of metabolic syndrome. Group C: Included 40 patients diagnosed with metabolic syndrome based on the IDF criteria (2006). Eligible participants within each group were randomly selected using a computer-

generated simple randomization method to ensure unbiased representation.

All individuals provided written informed consent. The Zagazig University Hospitals' Internal Medicine and Clinical Pathology departments gave their approval for the study after receiving Institutional Review Board (IRB) approval number (575/ 27-Aug-2024).

Inclusion Criteria: The study's participants were between the ages of 25 and 65, of all sexes. Group C participants were diagnosed according to the International Diabetes Federation's (IDF) 2006 standard criteria [having central obesity (waist circumference (WC) ≥ 102 cm for men and ≥ 88 cm for women), plus at least two of the following metabolic abnormalities: HDL-C < 40 mg/dL in men and < 50 mg/dL in women, or having taken lipid-lowering medications; triglycerides (TGs) > 150 mg/dl or drug treatment for elevated triglycerides; systolic blood pressure (SBP) 130 mmHg or diastolic blood pressure (DBP) 85 mmHg, or having taken antihypertensive medications; elevated fasting blood glucose (FBG) levels, FBG 100 mg/dL, or having been diagnosed with type 2 diabetes] **[9].** Group B participants were selected as having increased WC ≥ 102 cm for men and ≥ 88 cm for women, but only with 0–1 other criteria of metabolic syndrome stated above. Matched Age- and sex-matched subjects with normal WC and metabolic components as well as normal liver and kidney functions that assessed by the routine blood tests were used as the healthy controls (Group A).

Exclusion Criteria: We excluded from the study cases with these conditions, e.g., pregnancy, lactation, known cancer, pulmonary, hepatic, or renal diseases, or other issues that influence the patient. Also, a history of transient ischemia episodes, myocardial infarction, or stroke and usage of drugs that alter lipid or blood glucose metabolism (e.g., corticosteroids) were causes of exclusion from this study.

Clinical and Anthropometric Measurements:

Each participant in this study underwent a comprehensive history taking and clinical examination, which included measurements of the patient's height and body mass index (BMI), which was calculated as weight (kg)/height (m^2). Hip circumference (HC), which was measured by wrapping a cloth measuring tape around the hips' maximum circumference; waist circumference (WC), which was measured at the midpoint between the iliac crest and the lowest margin of the ribs; waist-to-hip ratio (WHR) **[10];** and blood pressure.

Blood sampling and biochemical measurements:

All patients' antecubital veins were used to draw venous blood samples following an overnight fast (about 10–12 hours). Following centrifugation at 3,000 rpm for 10 minutes, serum samples were collected and kept in aliquots at -80°C.

Fasting insulin level (FINS) was measured by electro-chemiluminescence immunoassay sandwich (ECLIA) on Cobas 8000 c701 (Cobas, Germany). Glycemic measures include 2-hour postprandial plasma glucose (2HPP) and fasting plasma glucose (FBG) by enzymatic hexokinase and glycated hemoglobin (HbA1c) by turbidimetric inhibition immunoassay (TINIA), which were measured on Cobas 8000 c701 (Roche, Germany). The following formula was used to determine the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR): (Fasting glucose in mg/dL x Fasting insulin in mIU/L) / 405 is HOMA-IR. Low-density lipoprotein (LDL) was measured using the LDLC3 homozygous enzymatic colorimetric assay on Cobas8000 c702 (Roche, Germany), while serum total cholesterol (TC), triglycerides (TG), and high-density lipoprotein (HDL) were

measured using enzymatic reactions that transform these lipids into compounds that can be measured spectrophotometrically. On the Cobas 8000 c701 (Roche, Germany), kidney and liver function tests (AST & ALT) were performed. ELISA (enzyme-linked immunosorbent assay) kits were used to quantify serum ZAG in accordance with the manufacturer's instructions (Catalog No: DL-aZGP1-Hu, Wuxi Donglin Sci & Tech Development Co., Ltd. Wuxi, China), LOT: EDL2025012001 **[11].**

Statistical analysis:

The Statistical Package for the Social Sciences (SPSS) program (version 26.0) was used to import the data and analyze it. Multiple linear regression analysis, Pearson correlation (r), ANOVA (f), the nonparametric Kruskal-Wallis test, the chi-square test ($\chi 2$), and the receiver operating characteristic curve (ROC) were employed.

RESULTS

There were statistically significant variations between the three groups' mean ages ($F = 19.7$, $p = 0.000$). While the distribution of sexes among the groups did not differ statistically significantly ($\chi^2 = 0.394$, $p = 0.941$). Regarding diabetes and hypertension, there was a highly significant difference in the clinical characteristics of the groups that were being studied ($p = 0.000$ for both). No cases of diabetes or hypertension were recorded by either the central obesity group or the control group. The metabolic group, on the other hand, showed a much greater prevalence of both diseases, with 45% of patients having diabetes and 72.5% having hypertension (Table 1).

The metabolic, central obesity, and control groups differed significantly from one another in weight, BMI, waist circumference, hip circumference, and waist-to-hip ratio (WHR) ($p = 0.000$). When compared to the control group, the central obesity and metabolic groups both exhibited greater values in each of these parameters, with the metabolic group typically having the highest values. Although there was no significant difference between the two obese groups ($p = 0.807$), WHR was considerably greater in the central obesity and metabolic groups than in the control group. However, Table 2 shows that there was no discernible variation in height across the groups ($p = 0.264$).

When compared to the control and central obesity groups, the metabolic group's fasting blood glucose (FBG), 2-hour postprandial glucose (2HPP), HbA1c, fasting insulin, and HOMA-IR were all markedly elevated. Although there were no significant changes in FBG ($p = 0.448$) or 2HPP ($p = 0.716$) between the central obesity and control groups, both parameters were significantly higher in the metabolic group ($p < 0.001$). Similar to the metabolic group, the central obesity group also showed modest but significant increases in HbA1c and fasting insulin levels as compared to controls. With the metabolic group exhibiting the highest results, HOMA-IR demonstrated a substantial stepwise increase across the groups, indicating growing insulin resistance. These results demonstrate a pronounced decline in metabolism from central obesity to overt metabolic syndrome. Total cholesterol, LDL, and triglycerides (TG) were considerably greater in the metabolic and central obesity groups than in the control group; both LDL and TG were greatest in the metabolic group. The HDL levels of the metabolic group were considerably lower than those of the control and central obesity groups ($p = 0.527$), despite the fact that there was no discernible difference in HDL levels between the two groups (Table 3).

The metabolic group's AST levels were marginally different from the central obesity group's ($p = 0.051$), but they were still

considerably higher than the control group's ($p = 0.001$). Although there was no significant difference between the two obese groups ($p = 0.647$), BUN levels were considerably higher in the central obesity and metabolic groups than in the control group ($p = 0.043$ and $p = 0.013$, respectively). ALT levels ($p = 0.100$) and creatinine levels ($p = 0.178$) did not significantly differ across the groups (Table 3).

The metabolic group had the lowest serum ZAG levels (43.4 ± 19.5 ng/ml), followed by the central obesity group (70.1 ± 25.5 ng/ml), and the highest in the control group (101.6 ± 54.1 ng/ml) (Table 4).

Serum ZAG was shown to be strongly negatively correlated with waist circumference ($r = -0.386$, $p = 0.014$) and BMI ($r = -0.364$, $p = 0.021$), suggesting that lower values of both metrics are linked to elevated blood ZAG levels. Additionally, serum ZAG demonstrated a somewhat negative relationship using weight ($p = 0.054$, $r = -0.306$), but one that was not statistically significant. Other metabolic, lipid, or liver function measures, such as fasting glucose, HbA1c, cholesterol, triglycerides, liver enzymes, or creatinine levels, did not significantly correlate with serum ZAG (all $p > 0.05$). These findings suggest that the central obesity group's blood ZAG levels are mostly related to body composition, particularly waist circumference and BMI (Table 1S).

Serum ZAG and weight ($r = -0.372$, $p = 0.018$) and BMI ($r = -0.361$, $p = 0.022$) showed a strong negative association, suggesting that in this population, lower body weight and BMI are linked to greater blood ZAG levels. Serum ZAG did not, however, substantially correlate with other variables such as waist size, indicators of renal function, liver enzymes, lipid profiles, or fasting blood glucose (all $p > 0.05$). According to these results, serum ZAG is more closely linked to body composition in the metabolic group than to metabolic or organ function metrics (Table 5).

The serum ZAG ROC curve analysis for predicting healthy individuals with all participants having central obesity (the central obesity group and the metabolic syndrome group) revealed that the area under the curve (AUC) for serum ZAG was 0.893, indicating good diagnostic accuracy. Serum ZAG showed 50% specificity and 98% sensitivity with a cutoff limit of 93 ng/ml. AUC and 95% CI ranged from 0.823 to 0.962, highlighting the validity of serum ZAG as a predictor for differentiating between healthy and unhealthy individuals. These findings demonstrate serum ZAG's substantial diagnostic potential for the identification of metabolic syndrome (Table 6).

The area under the curve (AUC) for serum ZAG was 0.792, according to the ROC curve analysis used to differentiate between the central obesity group and metabolic syndrome, suggesting a high degree of diagnostic precision. At a threshold of 58 ng/ml, serum ZAG demonstrated a sensitivity of 80% and a specificity of 60%. Although serum ZAG has a high sensitivity for identifying metabolic syndrome, its specificity should ideally be combined with other clinical or biochemical parameters to enhance specificity and diagnostic accuracy, particularly in populations already exhibiting central adiposity, according to the AUC, which showed a 95% CI of 0.695 to 0.889 (Table 6).

A statistically significant difference was observed between the groups (Kruskal-Wallis test, $p = 0.031$), suggesting that ZAG levels vary with the severity or number of metabolic syndrome criteria. Notably, patients meeting 5 criteria exhibited the lowest mean serum ZAG levels (19 ± 3.6 ng/ml), compared to those with 4 criteria (39.3 ± 20.7 ng/ml) and 3 criteria (48 ± 19.9

ng/ml). Post hoc analysis indicates a significant difference, particularly between the 3-criteria group and the 5-criteria group (P2 = 0.013), suggesting a potential inverse relationship between serum ZAG and metabolic syndrome severity. These findings may support the hypothesis that ZAG levels decline as metabolic syndrome worsens, highlighting its possible role as a biomarker for metabolic risk burden (Table 7).

There was no significant predictive value for serum ZAG from the variables under investigation, according to the results of multivariate linear regression analysis for significant predictors of serum ZAG levels among the study groups (Table 2S).

Table (1): Comparison of the baseline data of the studied groups:

Variable	Control (n=40)		C.Obesity (n=40)		Metabolic (n=40)		F-test	P-value
Age (years): • ***Mean ± SD*** • ***Range***	37.2±10.8 25-62		41.9±8.9 26-60		50.4±8.8 52-64		19.7	**0.000**** **(HS)**
Variable	**N**	**%**	**N**	**%**	**N**	**%**	**χ^2**	**P-value**
Sex: • ***Males*** • ***Females***	20 20	50 50	11 29	27.5 72.5	20 20	50 50	0.394	0.941 (NS)
Hypertension: • **No** • **Yes**	40 0	100 0	40 0	100 0	11 29	27.5 72.5	76.5	**0.000**** **(HS)**
Diabetes: • **No** • **Yes**	40 0	100 0	40 0	100 0	22 18	55 45	42.4	**0.000**** **(HS)**

Anova (F- test) and chi square (χ 2) tests were used, NS: Non- significant, C.: Central

HS: Highly significant

Table (2): Comparison of the anthropometric measures among the studied groups:

Variable	Control (n=40)	C.Obesity (n=40)	Metabolic (n=40)	F-test	P-value	LSD
Weight (Kg): • **Mean ± SD** • **Range**	65.8± 8.4 48-85	89.7±10.5 70-115	98.3±13.9 76-131	90.6	**0.000 (HS)**	**P1,0.000**** **P2,0.000**** **P3,0.001****
Height (cm): • **Mean ± SD** • **Range**	169.4±7.9 155-187	167.3±4.5 160-178	169.2±6.2 155-180	1.3	0.264 (NS)	--------
BMI (Kg/m^2): • **Mean ± SD** • **Range**	22.8±1.3 18.7-24.7	32±2.8 26.7-38.1	34.3±4 28.7-42.8	171.2	**0.000 (HS)**	**P1,0.000**** **P2,0.000**** **P3,0.001****
Waist C. (cm): • **Mean ± SD**	86.4±6.8 70-100	112.6±11.9 92-132	117.8±10.2 99-140	115.7	**0.000 (HS)**	**P1,0.000**** **P2,0.000**** **P3,0.000****

Variable	Control (n=40)	C.Obesity (n=40)	Metabolic (n=40)	F-test	P-value	LSD
• **Range**						
Hip C. (cm): • **Mean ± SD** • **Range**	103.8±6.6 90-1116	109.6±10.6 87-130	115.6±15.5 85-146	10.5	**0.000 (HS)**	**P1,0.026*** **P2,0.000**** **P3,0.021***
WHR: • **Mean ± SD** • **Range**	0.84±0.08 0.71-0.97	1±0.1 0.86-1.28	1±0.12 0.84-1.30	45.7	**0.000 (HS)**	**P1,0.000**** **P2,0.000**** P3,0.807

P1: control vs central obesity, P2: control vs metabolic, P3: central obesity vs metabolic, LSD: Least significant difference, S: Significant, HS : Highly significant, BMI: Body mass index, WHR: waist hip ratio, C.: circumference

Table (3): Comparison of the glycemic, lipid profile and liver and renal function parameters among the studied groups:

Variable	Control (n=40)	C.Obesity (n=40)	Metabolic (n=40)	F-test	P-value	LSD
FBG (mg/dl): • **Mean ± SD** • **Range**	79.9±5 71-90	82.6 ±5.7 73-97	102.2±26.6 124-194	13.1	**0.000 (HS)**	P1,0.448 **P2,0.000**** **P3,0.000****
2HPP (mg/dl): • **Mean ± SD** • **Range**	117.7±10.2 100-135	119.6±16.4 36-138	153.4±35.4 108-220	29.8	**0.000 (HS)**	P1,0.716 **P2,0.000**** **P3,0.000****
HbA1C (%): • **Mean ± SD** • **Range**	4.8±0.52 3.5-5.8	5.3±0.42 4.5-6	6.2±1.3 4.5-8.3	29.5	**0.000 (HS)**	**P1,0.017*** **P2,0.000**** **P3,0.000****
Fasting insulin (µIU/mL): • **Mean ± SD** • **Range**	6.9±0.96 4.3-9.3	8.2±0.78 6.7-10	8.9±1.9 6-13	23.7	**0.000 (HS)**	**P1,0.001**** **P2,0.000**** **P3,0.030***
HOMA-IR: • **Mean ± SD** • **Median** • **Range**	1.4±0.19 1.4 0.81-1.8	1.7±0.18 1.7 1.2-1.9	2.3±1 1.75 1.2-4.4	26.3 (K)	**0.000 (HS)**	**P1,0.019*** **P2,0.000**** **P3,0.000****

Variable	Control (n=40)	C.Obesity (n=40)	Metabolic (n=40)	F-test	P-value	LSD

Total. Cholesterol(mg/dl): • **Mean ± SD** • **Range**	156.9 ±11.5 134-182	173.9±13.2 150-210	167.3±13.6 140-200	17.7	**0.000 (HS)**	**P1,0.000**** **P2,0.000**** **P3,0.025***
HDL (mg/dl): • **Mean ± SD** • **Range**	59.7±8.3 42-77	61±6.5 46-73	40.1±12.6 19-66	60.3	**0.000 (HS)**	P1,0.527 **P2,0.000**** **P3,0.000****
LDL (mg/dl): • **Mean ± SD** • **Range**	78.6±11.6 49-121	89.9±12.5 67-128	96±14.4 79-135	18.8	**0.000 (HS)**	**P1,0.000**** **P2,0.000**** **P3,0.037***
TG (mg/dl): • **Mean ± SD** • **Range**	99.2±15.5 60.8-129	118.1±19.9 77-146	155.9±25.8 90-210	77.1	**0.000 (HS)**	**P1,0.000**** **P2,0.000**** **P3,0.000***

Variable	Control (n=40)	C.Obesity (n=40)	Metabolic (n=40)	F-test	P-value	LSD
AST (U/L): • **Mean ± SD** • **Range**	23.5 ±5.8 14-34	25.5±6.6 15-39	28.3±6.6 14-40	5.7	**0.004*** **(S)**	P1,0.167 **P2,0.001** P3,0.051
ALT (U/L): • **Mean ± SD** • **Range**	33.9±7.4 20-52	32.7±7.2 18-50	36.4±8.6 17-51	2.4	0.100 (NS)	———
BUN (mg/dl): • **Mean ± SD** • **Range**	11.4±2.2 7.9-16.1	12.4±1.8 9-15.2	12.6±2.3 8-16	3.6	**0.031*** **(S)**	**P1,0.043** **P2,0.013** P3,0.647
Creat (mg/dl): • **Mean ± SD** • **Range**	0.87±0.08 0.73-1.07	0.87±0.08 0.74-1.04	0.89±0.08 0.75-1.09	1.8	0.178 (NS)	———

K: Kruskal-Wallis's test, TG: triglycerides, LDL: Low density lipoprotein, HDL: High density lipoprotein BUN: Blood urea nitrogen

Table (4): Comparison of Serum Zinc-Alpha-2-Glycoprotein (ZAG) levels among the studied groups:

Variable	Control (n=40)	C.Obesity (n=40)	Metabolic (n=40)	K-test	P-value	LSD
Serum ZAG (ng/ml): • **Mean ± SD** • **Median** • **Range**	101.6 ±54.1 93.7 33.1-299.2	70.1±25.5 68.4 26.4-115.8	43.4±19.5 41.6 26.3-94.5	25.7	**0.000**** **(HS)**	**P1,0.000** **P2,0.000** **P3,0.001**

Table (5): Correlation between Serum ZAG and the other clinical data and biochemical parameters in the metabolic group:

Variable	Serum ZAG (ng\ml)	
	R	p-value
Age (years)	-0.115	0.480
Weight (Kg)	**-0.372***	**0.018***
Height (cm)	-0.142	0.382
BMI (Kg/m2)	**-0.361***	**0.022***
Waist C (cm)	-0.029	0.857
Hip C (cm)	-0.012	0.941
WHR	0.012	0.941
FBG (mg\dl)	-0.053	0.747
2HPP (mg\dl)	-0.095	0.558
HbA1c (%)	-0.022	0.891
F. Insulin (µIU/mL)	0.110	0.499
HOMA- IR	0.044	0.788
T. Cholesterol (mg/dl)	-0.146	0.477
HDL (mg/dl)	0.159	0.327
LDL (mg/dl)	-0.228	0.157
TG (mg/dl)	-0.138	0.394
AST (IU/L)	-0.121	0.459
ALT (IU/L)	-0.125	0.442
BUN (mg/dl)	-0.038	0.817
Creatinine (mg/dl)	0.028	0.864

Table (6): ROC curve analysis of serum ZAG in differentiating control group from the other 2 groups and in differentiating metabolic syndrome from the central obesity group.:

Variable	AUC	Cutoff point	P-value	Sensitivity	Specificity	CI
In differentiating control group from the other 2 groups.						
Serum Zag	**0.893**	**93**	**0.000**	**98%**	**50%**	**(0.823-0.962)**
In differentiating metabolic syndrome from the central obesity group.						
Serum Zag	**0.792**	**58**	**0.000**	**80%**	**60%**	**(0.695-0.889)**

Table (7): Serum Zinc-Alpha-2-Glycoprotein (ZAG) of different criteria groups among

Variable	3 criteria (n=26)	4 criteria (n=11)	5 criteria (n=3)	K-test	P-value	LSD
Serum ZAG (ng/ml): • ***Mean ± SD*** • ***Median*** • ***Range***	 48 ±19.9 44.2 16.8-94.5	 39.3±20.7 33.3 19.5-82.6	 19±3.6 17.8 16.3-23.2	3.8	**0.031 (S)**	P1,0.189 **P2,0.013** P3,0.098

DISCUSSION

In the current study, the mean ages of the study groups varied significantly, with the central obesity and control groups coming in second and third, respectively, to the metabolic syndrome group. This implies that the development of metabolic syndrome may be influenced by aging. This could be brought on by a decline in physical activity as people age, which increases oxidative stress and causes fat to accumulate. However, there was no discernible variation in the distribution of sexes, suggesting that sex might not be a decisive issue in this investigation.

These results are in line with those of Alenad et al. **[6]**, who found that the mean age of patients with metabolic syndrome was considerably higher than that of controls ($p < 0.001$) in a case-control comparison. Similarly, Yao et al. **[12]** discovered that older age (OR = 1.037; 95% CI: 1.036–1.039) and female sex (OR = 1.773; 95% CI: 1.709–1.840) were significant risk variables for metabolic syndrome.

Significant differences ($p < 0.001$) were seen in the study groups' weight, BMI, waist-to-hip ratio (WHR), and waist and hip circumferences based on anthropometric measurements. The groups with central obesity and metabolic syndrome had significantly higher values on these measures than the control group, with the metabolic group usually showing the highest means, indicating more advanced adiposity. Both the central obesity and metabolic groups did not significantly differ in WHR, and none of the groups differed significantly in height. These results demonstrate the clinical use of these anthropometric markers in identifying those at heightened risk for metabolic syndrome and the strong association between metabolic risk and central obesity.

In line with these findings, according to Alenad et al. **[6]**, there was no discernible variation in hip circumference or height; however, individuals with metabolic syndrome had significantly higher waist circumferences, weights, BMIs, and WHRs than controls. In a similar vein, Wang et al. **[13]** showed that the groups with central obesity and metabolic syndrome had significantly higher values of lean muscle mass, body weight, BMI, WC, HC, WHR, body fat rate, and fat-to-muscle ratio in comparison to the group with metabolic syndrome and healthy controls, as well as the central obesity group. Subjects with metabolic syndrome had significantly higher BMI, WC, HC, and WHR values ($p < 0.01$), according to Kheirouri and Alizadeh **[14].**

Comparing the clinical data from the study groups demonstrated a highly strong correlation between the prevalence of diabetes and hypertension and metabolic syndrome ($p = 0.000$ for both). A significant percentage of people in the metabolic syndrome group were afflicted; 72.5% had hypertension and 45% had diabetes, whereas neither condition was recorded in the control or central obesity groups. These results emphasize the clinical burden linked to metabolic syndrome by showing how the

condition is strongly correlated with an increased risk of cardiometabolic comorbidities.

In line with these findings, Bamekhlah and Saeed **[15]** found that diabetes was the most common metabolic syndrome component (79.2%), and all patients had hypertension. The significant comorbidity load linked to metabolic syndrome was further supported by Behera et al. **[16],** who discovered that patients with the syndrome also had high rates of diabetes (53.7%) and hypertension (55.4%).

A comparison of glycemic markers between the study groups showed that compared to the control and central obesity groups, the metabolic syndrome group had significantly higher levels of fasting blood glucose (FBG), 2-hour postprandial glucose (2HPP), HbA1c, fasting insulin, and HOMA-IR ($p < 0.001$ for all). The central obesity group and the control group did not differ significantly, but both FBG and 2HPPG were significantly higher in the metabolic group, indicating impaired glucose regulation. While the central obesity group also displayed slight increases, the metabolic group's HbA1c and fasting insulin were much greater. With the greatest median values observed among those with metabolic syndrome, HOMA-IR values gradually increased across the groups, indicating rising levels of insulin resistance. These results highlight the necessity of early intervention in at-risk patients and the continuum of metabolic dysregulation from obesity to metabolic syndrome.

Similarly, FBG, HbA1c, fasting insulin, and HOMA-IR, according to Elshourbagy et al. [17], were significantly greater in those with metabolic syndrome than in people without the condition. Patients with metabolic syndrome had significantly higher HbA1c values, according to research by Alenad et al. [6] and Alkhatib et al. [18].

All measured parameters, including total cholesterol, LDL, HDL, and triglycerides (TG), showed statistically significant differences when the lipid profiles of the groups under inquiry were compared ($p = 0.000$ for all). Total cholesterol, LDL, and TG values were considerably higher in the central obesity and metabolic syndrome groups than in the control group. With the highest mean TG and LDL readings, the metabolic group appeared to have a more severe dyslipidemic profile. Although there was no discernible difference between the HDL levels of the central obesity and control groups ($p = 0.527$), the metabolic group's HDL values were significantly lower than those of the control and central obesity groups. These results highlight the distinctive atherogenic dyslipidemia linked to metabolic syndrome and show a cumulative deterioration of lipid metabolism from central obesity to metabolic syndrome.

These results are consistent with Wang et al.'s study [13], which showed that TG levels rose sequentially and that HDL-C progressively dropped from the control group to the group with central obesity to the group with metabolic syndrome ($p<0.05$). Furthermore, they discovered that compared to the controls, the central obesity group's LDL-C values were noticeably greater, whereas the metabolic syndrome group had TC and LDL-C readings that were noticeably greater than those of the control and central obesity groups.

Similarly, Elshourbagy et al. [17] found that people with metabolic syndrome had higher levels of TC, TG, and LDL and significantly lower levels of HDL. Alenad et al. [6] discovered that people with metabolic syndrome had considerably higher TG and lower LDL when compared to matched controls, but no appreciable changes in TC or HDL.

The dyslipidemic shift that occurs when central obesity gives way to metabolic

syndrome is highlighted by these findings taken together, and they also emphasize how crucial early lipid profile monitoring and intervention are in preventing cardiovascular problems in at-risk patients.

AST levels were substantially higher ($p = 0.004$) than ALT levels. It showed no statistically significant difference ($p = 0.100$) when the liver function parameters of the study groups were compared. The group with metabolic syndrome had considerably greater AST than the control group ($p = 0.001$) and displayed a somewhat different pattern from the group with central obesity ($p = 0.051$). This pattern may indicate mild hepatocellular stress or early hepatic involvement associated with metabolic syndrome. Conversely, ALT levels were similar in all groups, indicating that overt hepatocellular damage might not be visible in this investigation just yet. These findings support the idea that AST may be used as a more precise early marker of liver stress in individuals who are developing metabolic syndrome.

Similarly, Elshourbagy et al. [17] discovered that people with metabolic syndrome had considerably higher levels of ALT and AST than matched controls. However, there were no observed variations in the levels of direct bilirubin, total bilirubin, or serum albumin.

Additionally, the effect of obesity on liver enzyme levels has been emphasized in a number of studies. BMI was found to be strongly correlated with both ALT ($\beta = 0.16$, $p = 0.002$) and GGT ($\beta = 0.19$, $p = 0.01$) by Jalili et al. [19]. Furthermore, Noor et al. [20] discovered that individuals with higher BMIs were more likely to have abnormal elevations in at least one liver enzyme. As a result, Huynh et al. [21] found that the prevalence of high AST and ALT increased progressively with BMI: between 9.0% and 24.0% for individuals with normal BMI and 16.4% for obese individuals, respectively.

In relation to renal function, Between groups, The difference in BUN levels was statistically significant ($p = 0.031$). Serum creatinine levels, however, ($p = 0.178$) were not statistically significant. Although there was no significant difference between the two obese groups ($p = 0.647$), BUN was considerably higher in the central obesity and metabolic syndrome groups than in the control group ($p = 0.043$ and $p = 0.013$, respectively). The raised BUN may be a sign of early functional problems in renal perfusion or increased protein metabolism in relation to metabolic syndrome and central obesity. Nonetheless, the absence of variation in creatinine levels indicates that glomerular filtration is unaffected and that there isn't any obvious renal impairment at this time. These results could point to mild renal stress in the early phases of metabolic decline, highlighting the significance of regular monitoring in high-risk patients.

According to Elshourbagy et al. [17], those with metabolic syndrome exhibited noticeably greater levels of both creatinine and urea than controls, which could be a sign of an early decline in renal function. Furthermore, in people 45 years of age and older, Ahmed et al. [22] discovered a favorable relationship between serum creatinine and BMI, identifying BMI as an independent predictor of renal function decline in this age group. This emphasizes how crucial it is to prevent obesity in order to preserve kidney health.

There was a substantial, progressive drop in Zinc-Alpha-2-Glycoprotein (ZAG) blood levels from the central obesity and metabolic syndrome groups to the control group ($p = 0.000$) when the study groups were compared. By validating substantial differences across all groups, the inverse correlation between ZAG levels and the degree of metabolic dysfunction was validated by post hoc analysis. These results imply that ZAG might regulate metabolism

and be a useful early indicator of metabolic deterioration.

Additionally, according to Liu et al. [23], those with metabolic syndrome had significantly lower serum ZAG levels than healthy controls, and there was a substantial correlation between serum ZAG and obesity. The results of Tan et al. [24] and Lei et al. [25], who discovered that people with metabolic syndrome had considerably lower ZAG levels than healthy controls, further reinforce the notion that ZAG is inversely associated with metabolic risk. Additionally, Liu et al. [26] showed that in Chinese participants, ZAG level had an inverse relationship with body weight and BMI.

Its possible use as a biomarker for evaluating metabolic risk early is demonstrated by the observed decrease in ZAG across deteriorating metabolic states. Further research is needed to determine how age, ethnicity, and other confounding factors affect ZAG levels in different groups.

The current study found that in the central obesity group, serum Zinc-Alpha-2-Glycoprotein (ZAG) levels were significantly inversely linked with both waist circumference ($r = -0.386$, $p = 0.014$) and BMI ($r = -0.364$, $p = 0.021$). This implies that decreased levels of ZAG in the blood are linked to higher levels of central and overall adiposity. There was also a somewhat negative correlation with body weight, but it was not statistically significant ($r = -0.306$, $p = 0.054$).

Conversely, serum ZAG levels and glycemic markers (fasting glucose, 2-hour postprandial glucose, and HbA1c) were found to be negatively but not significantly correlated. Serum ZAG levels also did not significantly correlate with insulin resistance (HOMA-IR), renal function markers (BUN, creatinine), liver function tests (AST, ALT), or lipid profiles (total cholesterol, HDL, LDL, and triglycerides). These findings imply that serum ZAG has a stronger correlation with anthropometric measurements in centrally obese persons than with metabolic or biochemical indicators.

Even in the absence of evident metabolic problems, ZAG's significance as a biomarker for metabolic risk associated with obesity is demonstrated by the observed associations, which support its position as a possible adipokine connected to fat mass and central fat distribution.

These results are in line with earlier research. In Chinese participants, Liu et al. [26] found that serum ZAG levels were inversely correlated with body weight and BMI. In a similar vein, Selva et al. [27] found that ZAG expression was decreased in both adipose tissue and the liver, and that blood ZAG concentrations were considerably lower in obese people than in controls ($p = .002$). Furthermore, ZAG was inversely correlated with visceral and subcutaneous fat BMI ($r = -0.65$, $p < .001$) and mRNA ZAG levels ($r = -0.68$, $p < .001$). Additionally, Gong et al. [28] showed inverse relationships between ZAG levels and body fat percentage ($r = -0.59$, $p = .01$), waist and hip circumferences ($r = -0.60$, $p < .001$), and BMI. Furthermore, according to Qu et al. [29], serum ZAG levels are inversely correlated with body fat percentage, lipids, fasting insulin, fasting plasma glucose, waist-to-hip ratio, BMI, and HOMA-IR, indicating more extensive links to metabolic disorders and obesity.

In the group with metabolic syndrome, Zinc-Alpha-2-Glycoprotein (ZAG) levels in the serum were found to substantially correlate adversely with BMI ($r = -0.361$, $p = 0.022$) and body weight ($r = -0.372$, $p = 0.018$). These results suggest that in people with metabolic syndrome, lower serum ZAG levels are linked to higher levels of total adiposity. It's noteworthy that serum ZAG levels did not significantly correlate with any of the following: lipid profile, liver

enzymes, renal function markers, insulin resistance (HOMA-IR), glycemic indices (HbA1c, 2-hour postprandial glucose, and fasting blood glucose), or waist circumference (all $p > 0.05$).

This trend implies that rather than reflecting central fat distribution or particular metabolic problems, serum ZAG continues to reflect overall adiposity in people with established metabolic syndrome. The lack of correlation with important metabolic indicators would suggest that ZAG's impact is more directly related to the load of adipose tissue than it is to the downstream metabolic abnormalities that define metabolic syndrome.

These findings are consistent with those of Liu et al. [26], who found that in patients with newly diagnosed metabolic syndrome, serum ZAG was negatively correlated with fat percentage, BMI, waist-to-hip ratio, blood pressure, triglycerides, fasting glucose, postprandial glucose, insulin levels, HbA1c, and HOMA-IR (all $p < 0.01$). Tan et al. [24] also discovered a high correlation between ZAG and a number of metabolic syndrome indicators, including fasting blood glucose, waist circumference, and triglycerides.

In the current study, according to receiver operating characteristic (ROC) curve analysis, blood zinc-alpha-2-glycoprotein (ZAG) offers a strong diagnostic potential for identifying individuals with metabolic syndrome. When comparing individuals with metabolic syndrome to healthy controls, the diagnosis accuracy was very good, with an area under the curve (AUC) of 0.893 (95% CI: 0.823–0.962, $p = 0.000$). Serum ZAG had a sensitivity of 98% and a specificity of 50%, with a threshold value of 93 ng/mL. These findings imply that serum ZAG is a valuable noninvasive biomarker for metabolic syndrome early detection, which may lead to better clinical management and prompt intervention.

Subsequent investigation assessed serum ZAG's diagnostic capacity to differentiate between those with central obesity and those with metabolic syndrome. This comparison's AUC, which showed good diagnostic performance, 95% CI: 0.695–0.889, $p = 0.000$, was 0.792. Using a cutoff value of 58 ng/mL, serum ZAG demonstrated a high sensitivity of 80% and a low specificity of 60%. This pattern indicates that although ZAG is useful in identifying metabolic syndrome in people who are centrally obese, its lower specificity might be brought on by early metabolic dysregulation or overlapping metabolic characteristics in this population. These results suggest that, even if serum ZAG is sensitive, it is best to complement it with additional clinical or biochemical measures to improve diagnostic accuracy and specificity, especially in people that already have central adiposity.

These findings are consistent with earlier studies. Serum ZAG by itself has a low diagnostic usefulness for metabolic syndrome (AUC = 0.655), but when combined with fat mass ratio, it significantly increased diagnostic accuracy (AUC = 0.951), with 90.7% sensitivity and 88.6% specificity, according to Wang et al. [13]. Similarly, Lei et al. [25] showed that serum ZAG had a 92% sensitivity and 59% specificity, making it a viable predictor for metabolic syndrome with a threshold value of 45.2 mg/L and an AUC of 0.80.

Conclusion: ZAG may play an important role in early diagnosis and management of metabolic syndrome, especially in those with central obesity, as they are the most susceptible to it. And since zinc is the main regulator of ZAG, zinc may be used as a supportive treatment for metabolic syndrome to decrease its occurrence and improve its prognosis, but we need further studies to confirm this.

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Table 1S: Correlation between Serum ZAG and the other clinical and biochemical parameters in central obesity group:

Variable	Serum ZAG (ng\ml)	
	R	**p-value**
Age (years)	-0.261	0.104
Weight (Kg)	-0.306	0.054
Height (cm)	-0.091	0.577
BMI (Kg/m2)	**-0.364***	**0.021***
Waist C (cm)	**-0.386***	**0.014***
Hip C (cm)	-0.133	0.421
WHR	-0.214	0.148
FBG (mg\dl)	0.090	0.518
2HPP (mg\dl)	0.160	0.323
HbA1c (%)	-0.001	0.995
F. Insulin (µIU/mL)	0.016	0.921
HOMA- IR	0.101	0.536
T.Cholesterol (mg/dl)	-0.096	0.566
HDL (mg/dl)	-0.028	0.862
LDL (mg/dl)	-0.110	0.499
TG (mg/dl)	-0.183	0.257
AST (IU/L)	-0.304	0.506
ALT (IU/L)	-0.205	0.205
BUN (mg/dl)	-0.131	0.419
Creatinine (mg/dl)	0.092	0.572

Table 2S: Multivariate linear regression analysis for significant predictors of Serum Zag level among the studied groups:

Variables	Standardized Coefficients Beta	SE	P-value
Age (years)	-0.108	0.370	0.250
Weight (Kg)	-1.02	2.8	0.375
Height (cm)	0.50	2.7	0.211
BMI (Kg/m2)	0.528	8.2	0.632
Waist C (cm)	-0.438	1.6	0.492
Hip C (cm)	0.329	1.6	0.466
WHR	0.437	167.9	0.419
FBG (mg\dl)	0.346	1.5	0.590
2HPP (mg\dl)	-0.304	0.277	0.097
HbA1c (%)	0.066	7.8	0.718
F. Insulin (µIU/mL)	0.128	13.7	0.791
HOMA- IR	-0.184	58.3	0.849
T. Cholesterol (mg/dl)	0.245	0.804	0.366
HDL (mg/dl)	-0.106	0.805	0.690
LDL (mg/dl)	-0.146	0.719	0.551
TG (mg/dl)	-0.331	0.255	0.078
AST (IU/L)	-0.080	1.1	0.635
ALT (IU/L)	0.037	0.904	0.822
BUN (mg/dl)	-0.018	1.9	0.855
Creatinine (mg/dl)	0.70	50.2	0.461

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