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Original article

Chemerin Versus Omentin-1 In Relation to Coronary Artery Disease in Obese Egyptian Patients

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BMI

and CRP.

Abstract

Background: The leading causes of mortality and disability globally are coronary artery disease and myocardial infarction, which is its primary consequence. Their growth is significantly influenced by lifestyle choices and the environment. These complicated illnesses also tend to run in families, which points to a significant hereditary component. Clear evidence of a molecular genetic link with coronary artery disease or myocardial infarction has yet to be found, despite significant investigation of several genes. **Method**: 75 patients were divided into 5 equal groups (control, obese ischemic, overweight ischemic, normal weight ischemic, and Obese noncardiac groups). According to BMI and the results of coronary angiography, patients will be included in one of the study groups mentioned above. **Results:** Omentin-1 is the highest in the control group and is the lowest in the obese cardiac group. Omentin-1 statistically significant differences existed between the groups. Omentin has; a strong negative correlation with BMI and CRP; a weak

negative correlation with Age; a moderate negative correlation with coronary artery disease; no correlation with Lipid profile, Hb A1c, gender, and hypertension. Chemerin is the highest in the obese cardiac group and the lowest in the control group. Chemerin differences between groups were statistically significant, Chemerin has a strong positive correlation with BMI and CRP, a moderate positive correlation with Age and Coronary Artery Disease, a weak positive correlation with HB_A1C, and no correlation with Lipid profile, gender, or hypertension. Omentin-1 & Chemerin have a strong negative correlation between each other. **Conclusion:** plasma chemerin level was increased, while plasma omentin-1 level was decreased in obese patients with CAD, and they can be considered independent predictors of coronary artery disease in obese patients.

1. Introduction:

Over the past 50 years, obesity has become an epidemic in many parts of the world. Because it significantly raises the risk of diseases like type 2 diabetes mellitus, fatty liver disease, hypertension, myocardial infarction, stroke, dementia, osteoarthritis, obstructive sleep apnea, and several cancers, obesity poses a serious threat to one's health and shortens one's life expectancy. **(1).**

The primary causes of mortality and disability globally are coronary artery disease and its principal consequence, myocardial infarction. Their development is heavily influenced by lifestyle and environmental variables. Moreover, these complicated disorders tend to cluster in families, implying a strong hereditary component **(2).**

Through systemic activities in the brain, liver, and muscle, adipokines play significant roles in the control of systemic lipids and glucose metabolism. Because adipokine secretion and blood levels are most affected by adiposity, it has been suggested that a pathogenic connection exists between obesity, the metabolic syndrome (MetS), and cardiovascular disease as a result of the

dysregulation of pro-inflammatory and anti-inflammatory adipokine secretion that is associated with obesity. **(3)**.

Chemerin is a novel adipokine that can control adipocyte development and drive dendritic cell and macrophage chemotaxis. Circulating chemerin has been linked to inflammation, obesity, metabolic syndrome, and coronary artery disease (CAD) in a growing body of research (**4**).

Omentin-1, a new adipokine found in visceral adipose tissue, has been linked to insulin resistance and obesity. Several chronic inflammatory illnesses have been linked to reduced omentin-1 expression. Nevertheless, the role of omentin-1 in coronary artery disease (CAD) remains unknown **(5).**

2. Patients and Methods:

The current study is a case-control study including 75 participants collected from the inpatient unit of the Department of Cardiology, Beni-Suef University Hospital from Feb 2022 to October 2022. the participants aged 30~69Y, 32 females and 43 males and were split up into the following five groups:

- **Group 1**: (control group)15 healthy persons whose BMI is 18.5~24.9kg/m² documented by coronary angiography.
- **Group 2**: (obese ischemic cardiac patients)15 patients whose BMI ≥ 30kg/m² with coronary artery disease documented by coronary angiography.
- **Group 3**: (overweight cardiac ischemic patients)15 patients whose BMI is 25~29.9 kg/m² with coronary artery disease documented by coronary angiography.
- **Group 4**: (normal weight cardiac ischemic patients) 15 patients whose BMI is 18.5~24.9 kg/m² with coronary artery disease documented by coronary angiography.
- **Group 5**: (Obese noncardiac patients) 15 patients whose BMI ≥ 30 kg/m² without coronary artery disease as documented by coronary angiography.

Exclusion Criteria were Patients who decline to take part in the research, Patients <20 years old, BMI<18.5, Patients with significant diseases that may alter our results (Patients with renal impairment, liver disease, and Patients with known malignant disease.

All patients are submitted to a thorough clinical examination and medical history with a focus on age, gender, Body mass index (BMI) calculation, Full History Taking, and Cardiovascular Risk Profile.

Sample collection

A 5 ml blood sample was drawn from patients who agree to participate in this study for obtaining the following investigations:

- plasma chemerin level (An Enzyme-Linked Immunosorbent Assay (ELISA) kit was used to assay human chemerin in the sample according to the manufacturer's instructions (SinoGeneclon Co., Ltd, Product No.: SG-10352) The kit is for the quantitative level of Chemerin in the sample)
- plasma omentin-1 level (ELISA kit (E-EL-H2028, Elabscience) according to its protocol, The kit is for the quantitative level of omentin-1 in the sample.
- Lipid profile (LDL, HDL, TG).
- S.creatinine for measuring kidney function
- Glycated hemoglobin (Hb A 1c).
- CRP

Coronary Angiography Results

Coronary artery stenosis is commonly evaluated by quantitative coronary angiography (QCA). Lumen diameter and stenosis cannot be evaluated objectively by eye inspection. QCA was created as a result for the impartial evaluation of lumen diameter. ***(6).***

Ethical considerations

The Local Research Ethical Committee (REC) of the Beni-Suef University Faculty of Medicine gave the study the ethical approval it required. The research's goals were conveyed to the patients in Arabic, along with their rights to decline participation in the trial. After the participants consented to take part in the trial, they were requested to sign a consent form.

Approval No: FMBSUREC\01022022\Taha

Statistical Analysis

The statistical software SPSS version 23 was used to code and input the data. For quantitative variables, the data were summarized using the mean and standard deviation.. Analysis of variance (ANOVA) with multiple comparisons post hoc test was applied for comparing more than 2 groups. P-values less than 0.05 were considered statistically significant.

3. Results:

Diabatic Profile (HBA1c)

Table (1) and Figure (1) show the differences in HA1c among the studied groups. HBA1c is highest in group 3, and lowest in group 1. There was no statistical significance in HBA1c between the studied groups (p=0.083).

Table (1): differences in HBA1c among the studied groups; (N= 75):

		Control N= 15	Obese Cardiac N=15	Overweight Cardiac N= 15	Normal weight Cardiac N=15	Obese Non-cardiac N= 15	P-Value
HB_A1C	Min	4.5	4.6	4.69	4.53	4.5	0.083
	Max	7.3	7.7	7.910	7.890	7.860	
	Mean ± SD	5.27±0.816	5.854±1.103	6.362±1.13	5.775±1.085	6.088±1.17	

** P-value ≤ 0.05 is considered significant by ((Kruskal-Wallis one-way ANOVA).*

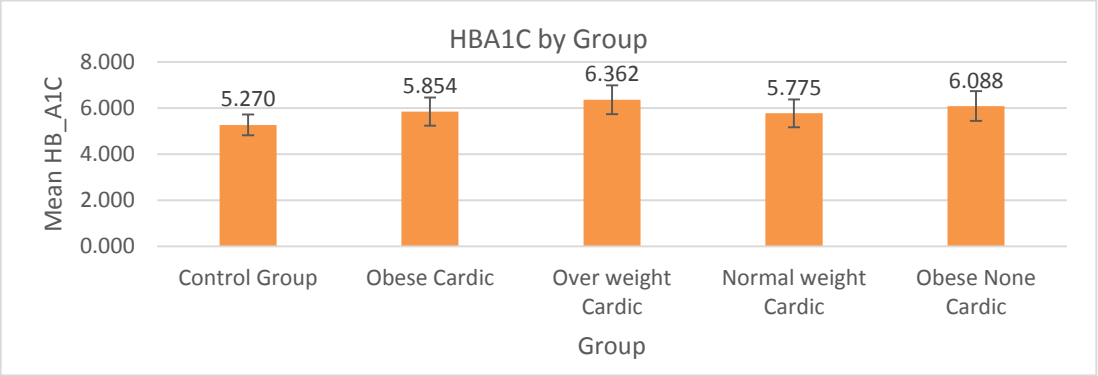

Figure (1): differences in HBA1c among the studied groups

Inflammatory Profile

Table (2) and Figure (2) show the differences in CRP among the studied groups. CRP level is highest in group 2 and lowest in group 1.

CRP showed a statistical significance(p-value<0.001) increase in group 2 by (+736%), group 3 by (+570%), group 4 by (+391%), and group 5 by (+197%) in comparison to group 1.

Moreover, CRP was statistically significantly lower in group 3 by (-25%), in group 4 by (-70.4%), and in group 5 by (-184%) in comparison to group 2.

in addition, CRP was statistically significantly lower in group 4 by (-36.4%) and in group 5 by (-127.4%) in comparison to group 3. and CRP was statistically significantly lower in group 5 by (-66.7%) in comparison to group 4.

Table (2): the differences in CRP among the studied groups; (N= 75):

		Control N= 15	Obese Cardiac N=15 a,c,d,e	Overweight Cardiac N= 15 a,b,d,e	Normal weight Cardiac N=15 a,b,e,d	Obese Non-cardiac N= 15 a,b,c,d	P-Value
CRP	**Min**	0.1	2	2	1.5	0.9	<0.001
	Max	1.2	6.5	5.7	5	3	
	Mean ± SD	0.57±0.31	4.77±1.36	3.82±1.17	2.8±0.98	1.68±0.7	

** P-value ≤ 0.05 is considered significant by ((Kruskal-Wallis one-way ANOVA).*

a: significance from control group at p-value <0.05.

b: significance from Obese Cardiac group at p-value <0.05

c: significance from Overweight Cardiac group at p-value <0.05

d: significance from Normal weight Cardiac group at p-value <0.05

e.: significance from Obese Non-Cardiac group at p-value <0.05

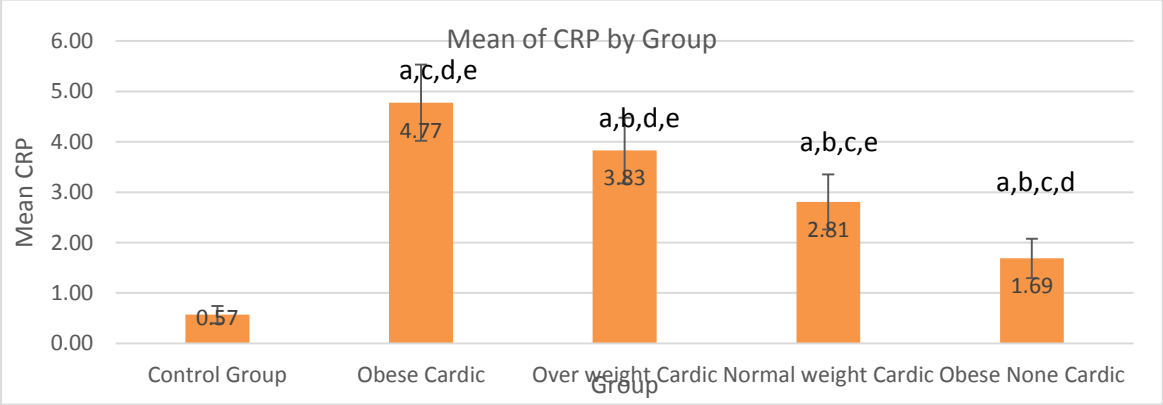

Figure (2): differences in CRP among the studied groups

Adipokines Profile

Table (3) and Figure (3) show the differences in chemerin among the studied groups. Chemerin is highest in group 2 and lowest in group 1.

Chemerin showed a statistical significance(p-value<0.001) increase in group 2 by (+461%), group 3 by (+284%), group 4 by (+193%), and group 5 by(+326%) in comparison to group 1.

Moreover, Chemerin was statistically significantly lower in group 5 by (-24.2%), in group 3 by (-31.6%), and in group 4 by (-47.8%) in comparison to group 2. Furthermore, Chemerin was statistically significantly higher in group 5 by (+45.2%) and in group 3 by (+31.6%) in comparison to group 4. And there is no statistically significant difference between group 3 and group 5.

Table (7) and Figure (17) show the differences in omentin-1 among the studied groups. Omentin-1 is highest in group 1 and lowest in group 2.

Omentin-1 showed a statistical significant (p-value<0.001) decrease in group 2 by (-65%), group 3 by (-41%), and group 5 by (-10%) in comparison to group 1.

Furthermore, Omentin-1 levels were statistically significantly higher in group 4 by (+187%), in group 5 by (+158%), and in group 3 by (+68%) in comparison to group 2. And it was statistically significantly higher in group 4 by (+70%) and in group 5 by (+53%) in comparison to group 3. And there is no statistically significant between group 1 and group 4.

Table (3): the differences in adipokines profile among the studied groups (N= 75):

		G #1 N=15	G #2 N=15	G #3 N=15	G #4 N=15	G #5 N=15	p-Value
Chemerin	**Min**	1.01	7.4	4.88	3.2	5.2	<0.001
	Max	2.3	10.7	8.69	7.06	9.02	
	Mean ± SD	1.65±0.42	9.27±1.12 a,c,d,e	6.34±1.21 a,b,d	4.84±1.14 a,b,e	7.03±1.18 a,b,d	
Omentin-1	**Min**	253.4	85.4	132.1	229.1	213.7	<0.001
	Max	318.6	112.1	205.6	312.6	302.4	
	Mean ± SD	284±29	98±8.9 a,c,d,e	165±23.3 a,b,d,e	281±19.38 b,c,e	253±31 a,b,c,d	

** P-value ≤ 0.05 is considered significant by ((Kruskal-Wallis one-way ANOVA).*

a: significance from control group at p-value <0.05.

b: significance from Obese Cardiac group at p-value <0.05

c: significance from Overweight Cardiac group at p-value <0.05

d: significance from Normal weight Cardiac group at p-value <0.05

e.: significance from Obese Non-Cardiac group at p-value <0.05

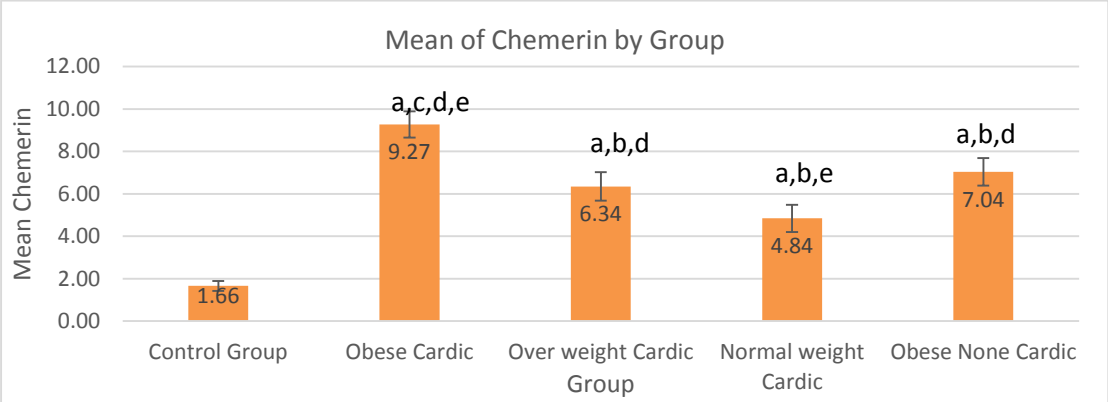

Figure (3): differences in chemerin among the studied groups.

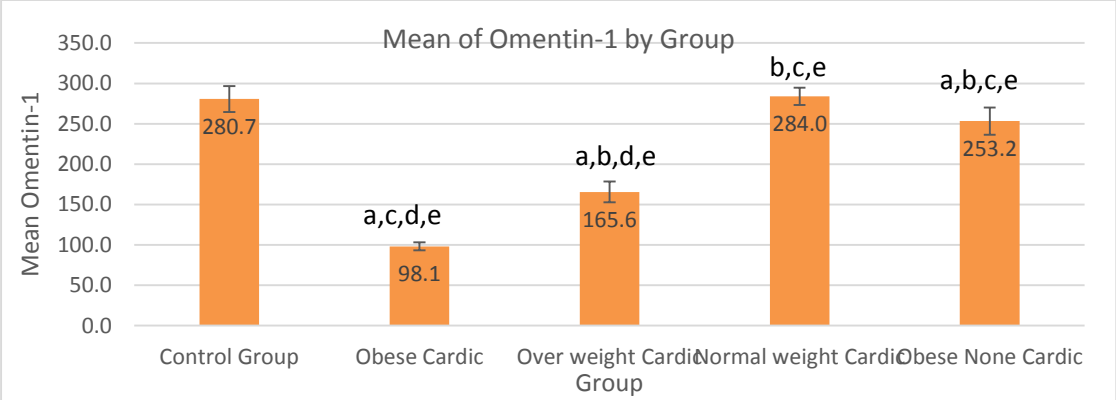

Figure (4): differences in Omentin-1 among the studied groups.

Serum Chemerin and Omentin-1 correlation with BMI:

Table (4) and figures (5) (6) demonstrate the correlation with BMI among each group and in the total population. There was a statistically significant positive linear correlation between Chemerin and BMI (r= 0.664, p=0.001) in all studied populations, however, there was a non-statistically significant correlation in each group separately (p-values >0.05). There was a statistically significant negative linear correlation between Omentin-1 and BMI (r= -0.487, p=0.001) in all studied population, however there was non-statistically significant correlation in each group separately (p-values >0.05).

Table (4): Serum Chemerin and Omentin-1 correlation with BMI:

		G #1 N= 15	G #2 N= 15	G #3 N= 15	G #4 N= 15	G #5 N= 15	Total Population N= 75
Chemerin							
BMI	**R**	0.353	0.036	0.309	-0.206	-0.012	0.664
	p-value	**0.196**	**0.898**	**0.262**	**0.462**	**0.967**	**<0.001***
Omentin-1							
BMI	**R**	-0.331	-0.239	-0.024	-0.059	0.023	-0.487
	p-value	**0.228**	**0.391**	**0.933**	**0.835**	**0.936**	**<0.001***

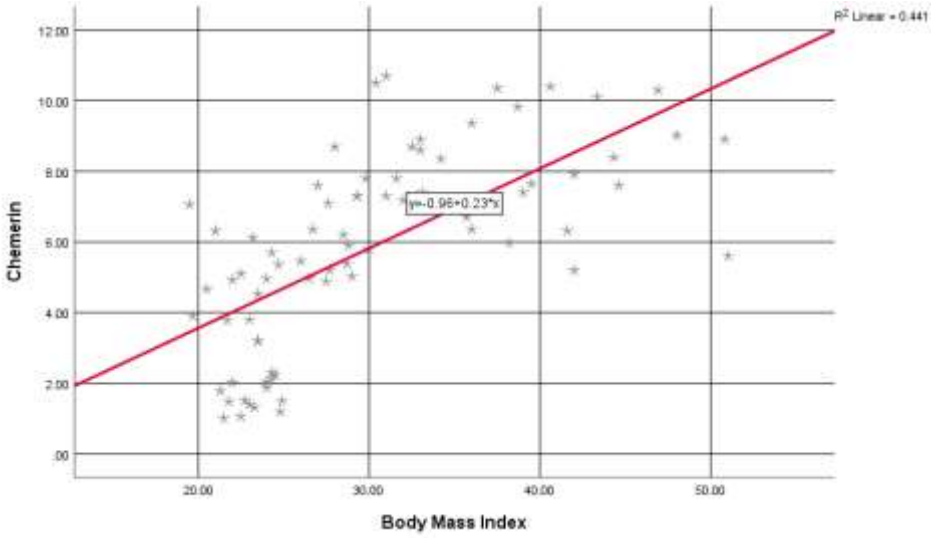

Figure (5): Omentin-1 level & BMI among the Studied Population.

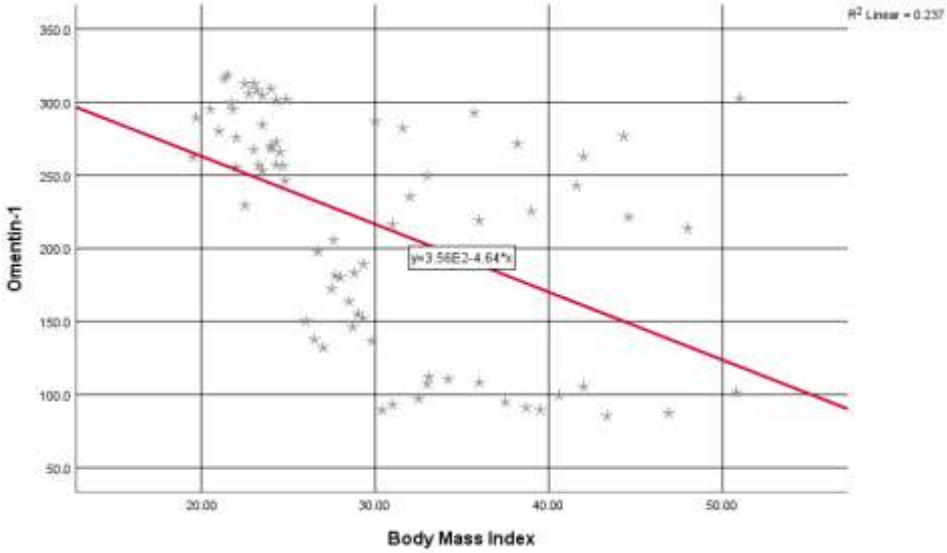

Figure (6): Serum Omentin-1 correlation with BMI among the Studied Population.

Association between Serum Chemerin and Omentin-1 with participants' CAD:

Table (5) shows that The Omentin-1 and Chemerin and coronary artery disease have a moderate negative correlation ($p < 0.001$).

Table (5): Association between Serum Chemerin and Omentin-1 with participants' CAD:

		Total Population N= 75
Chemerin		
CAD	**Normal**	4.3477± 2.87
	Abnormal	6.81± 2.17
	p-value	<0.001
Omentin-1		
CAD	**Normal**	266.9± 32.53
	Abnormal	182.575± 79.7
	p-value	<0.001

All studied participants in group-2 and group-3 and group-4 have abnormal CAD, All studied participants in group-1 and group-5 has normal CAD. So, no statistics are computed.

Data presented as mean ±SD

** P-value ≤ 0.05 is considered significant by ((Kruskal-Wallis one-way ANOVA).*

Serum Chemerin and Omentin-1 as indicators for coronary artery disease:

The patients were classified into two groups based on the ischemic cardiac disease to determine the cut-off diagnostic value of serum chemerin and omentin-1 levels.

Using ROC curve analysis, the sensitivity and specificity of serum chemerin and omentin-1 levels for prediction of ischemic cardiac disease among studied population was evaluated as demonstrated in table (١٦).

Table (6) and figure (7) the area under the curve (AUC) of chemerin for prediction of ischemic cardiac disease was (AUC = 0.720, SE = 0.063, 95% CI: 0.597–0.843). A chemerin of ≥3.4 could predict ischemic cardiac disease with a sensitivity of 95% (true positive cases) and a specificity of 50% (true negative cases).

Table (6) and figure (8) demonstrate the area under the curve (AUC) of omentin-1 for prediction of ischemic cardiac disease was (AUC = 0.784, SE = 0.053, 95% CI: 0.681–0.888). An omentin-1 of ≤220 could predict ischemic cardiac disease with a sensitivity of 90% (true positive cases) and a specificity of 66.7% (true negative cases).

Table (6): results of ROC curve analysis for sensitivity and specificity of Chemerin and Omentin-1 for prediction of ischemic cardiac disease among studied population:

	AUC	SE	p-value	95% CI of AUC		Cut-off	Sensitivity	Specificity
				Lower Bound	Upper Bound			
Chemerin	0.72	0.063	<0.001*	0.597	0.843	≥3.4	95%	50%
Omentin-1	0.784	0.053	<0.001*	0.681	0.888	≤220	90%	66.70%

AUC: *Area under the curve,* ***CI:*** *Asymptotic 95% Confidence Interval of AUC.*

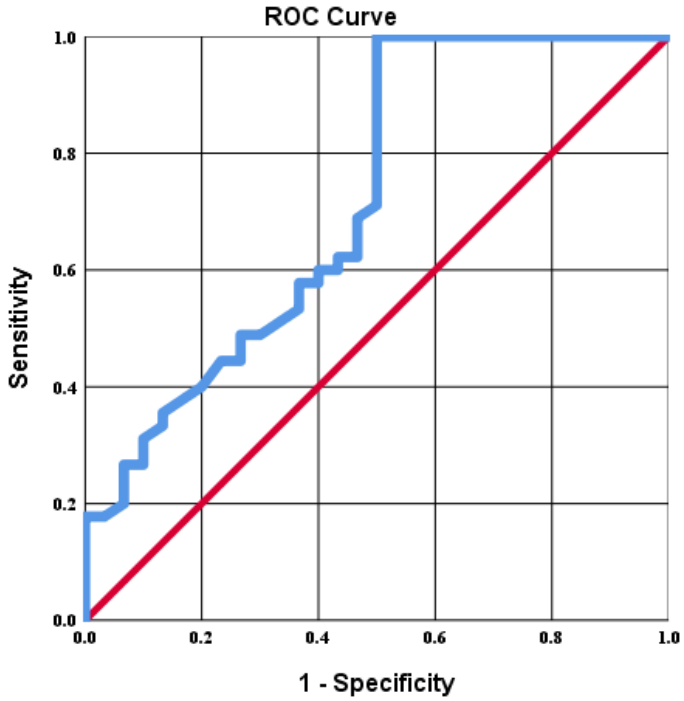

Figure (7): results of ROC curve analysis for sensitivity and specificity of Chemerin for prediction of ischemic cardiac disease among studied population

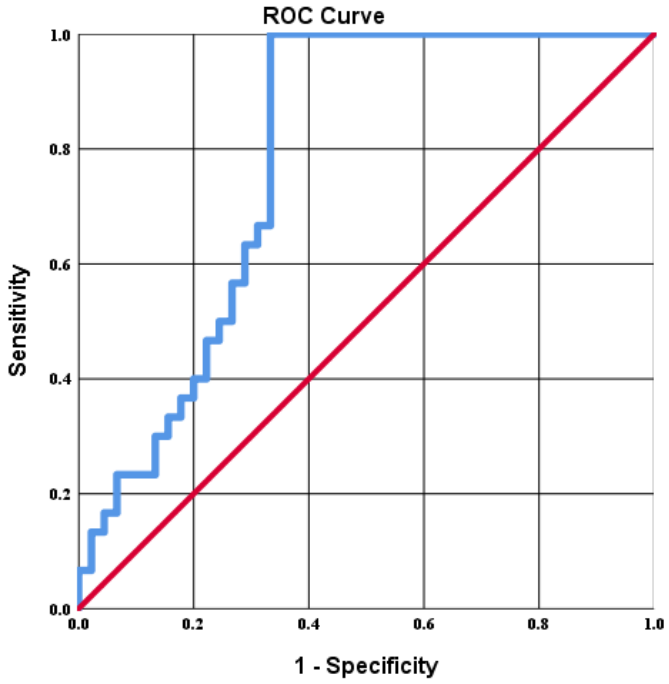

Figure (8): results of ROC curve analysis for sensitivity and specificity of Omentin-1 for prediction of ischemic cardiac disease among studied population

4. Discussion:

Cardiovascular diseases (CVDs) are one of the leading causes of mortality for millions of people worldwide. Controlling the risk factors such as lifestyle modification, glycemic control, BP, and BMI as they have a more significant impact on the CVDs may prevent the CVDs. By 2030, the number of deaths from CVDs might reach 23.3 million if this is disregarded and no preventative measures are done. **(7)**.

Patients with carotid atherosclerosis and CAD had reduced levels of the adipokine omentin-1, which points to its cardioprotective function. **(8)**.

An adipokine called chemerin is generated by fully developed adipocytes. It is a pro-inflammatory cytokine that has been linked to insulin resistance, metabolic disorders, and cardiovascular disease (CVD) since it is elevated in the blood of people who have obesity and cardiovascular disease. **(9)**.

The present study was designed to examine whether serum levels of Chemerin and Omentin-1 are associated with metabolic parameters and coronary artery disease in obese Egyptian patients and to determine

whether serum Chemerin and Omentin-1 can be considered independent predictors of coronary artery disease in obese patients.

Results of the present study revealed that HBA1C is highest in group 3, and lowest in group 1. There was no statistically significant in HBA1C between the studied groups.

This is in agreement with the study performed by Siegrist et al. **(10),** who found that at baseline, children with either overweight or obese had higher glucose levels and insulin, compared to children with normal weight. Also, *Baig et al.* **(11),** in studying the association of serum omentin-1, chemerin, and leptin with acute myocardial infarction and its risk factors found that FPG and HbA1c were higher in the patients.

This can be explained by that Type 2 diabetes poses a risk factor for CAD, CAD has been observed to be higher in patients with diabetes than in non- diabetes **(12)**.

The result of the present study revealed that the CRP level is highest in group 2 and lowest in group 1.CRP showed a statistically significant increase in group 2, group 3, group 4, and group 5 in comparison to group1. Moreover, CRP was statistically significantly lower in group 3, group 4, and group 5 in comparison to group 2.

in addition, CRP was statistically significantly lower in group 4 and in group 5 in comparison to group 3. and CRP was statistically significantly lower in group 5 in comparison to group 4.

This is in agreement with the study performed by Siegrist et al. **(10),** who found that at baseline, children with either overweight or obese had significantly higher hs-CRP compared to children with normal weight.

The results of the present study were also similar to the findings in a study performed by Mohammad and his colleagues. **(13),** who found that CRP level increased in both the SA and ACS groups.

The possible explanation is that CAD is a pathologic condition recognized as a chronic inflammatory process with elevated markers of systemic inflammation ***(14)***. Also, low-grade inflammation induced by obesity is likely to increase CRP.

Results of the present study revealed that Chemerin is highest in group 2 and lowest in group 1. Chemerin showed a statistically significant increase in group 2, group 3, group 4, and group 5 in comparison to group 1. Moreover, Chemerin was

statistically significantly lower in group 5, group 3, and group 4 in comparison to group 2. Furthermore, Chemerin was statistically significantly higher in group 5 and in group 3 in comparison to group 4. And there is no statistically significant between group 3 and group 5.

This is in agreement with the study performed by Baig et al. **(11),** who found that plasma chemerin level was markedly raised among acute myocardial infarction patients.

Also, Mohammad and his colleagues. **(13),** concluded that; plasma Chemerin level showed a significant increase in its level in the SA group and ACS groups compared to the control group, there was also a significant increase between SA and ACS groups.

In addition, Sell et al. **(15),** discovered that the release of chemerin is directly linked with BMI, waist-hip ratio, and fat cell volume and is greater in obese persons' adipose tissue explants than in normal-weight controls..

On the other hand, a study that disagrees with our results done by Szpakowicz et al. **(16),** found that There was no significant difference in chemerin levels between patients with carotid atherosclerosis and patients with normal carotid arteries.

The results of this study revealed that Omentin-1 is highest in group 1 and lowest in group 2. Omentin-1 showed a statistically significant decrease in group 2, group 3, and group 5 in comparison to group 1. Furthermore, Omentin-1 levels were statistically significantly higher in group 4, group 5, and group 3 in comparison to group 2. And it was statistically significantly higher in group 4 and in group 5 in comparison to group 3. And there is no statistically significant between group 1 and group 4.

This is in agreement with the study performed by Baig et al. **(11),** who found that serum omentin-1 was significantly lowered among myocardial infarction patients.

The present study results are also similar to the study performed by Siegrist et al. **(10),** who found that at baseline, children with either overweight or obese had significantly lower omentin-1 levels compared to children with normal weight.

This can be explained by the possibility that the endothelial cell dysfunction in the blood arteries of the visceral abdominal tissues is caused by the inflammatory state associated with obesity and CVD, which reduces omentin expression and release into the circulation. **(11).**

In the present study; There was a statistically significant negative linear correlation between Omentin-1 and BMI in all studied populations.

This may be because obesity adversely regulates the expression and release of the omentin gene **(11).**

This is in agreement with the study performed by Baig et al. **(11),** who found that Serum omentin-1 had a negative correlation with body weight, BMI, and waist circumference in MI patients and in the control group.

On the other hand, a study that disagrees with the results of the present study was done by Özkan et al. **(17),** who found that plasma omentin-1 levels were significantly higher in the O/O group compared to the control group. this may be due to the presence of non-alcoholic fatty liver disease subjects in their study and elevated adipokine levels are closely linked with hepatosteatosis.

The results of the present study revealed that There was a statistically significant positive linear correlation between Chemerin and BMI in all studied populations.

This is in agreement with the study performed by *Mirmajidi et al.* **(18),** who found evidence of a strong positive correlation between plasma concentrations of chemerin and BMI. Also, Baig et al. **(11),** found that Serum chemerin significantly increased with body weight, BMI, and waist circumference in MI patients and control subjects.

This can be explained by the fact that the expression and secretion of chemerin are elevated with adipogenesis **(18)**.

In the present study; the results of the Omentin-1 ROC curve analysis showed that a plasma omentin-1 level of ≤220 could predict ischemic cardiac disease with a sensitivity of 90% and a specificity of 66.7%.

In this study; the Chemerin ROC curve analysis showed that A chemerin of ≥3.4 could predict ischemic cardiac disease with a sensitivity of 95% and a specificity of 50%.

5. References:

1. **Blüher, M. (2019):** Obesity: global epidemiology and pathogenesis. *Nature Reviews Endocrinology*, *15*(5), 288-298.
2. M**alakar AK, Choudhury D, Halder B, Paul P, Uddin A, Chakraborty S. A (2019):** review on coronary artery disease, its risk factors, and therapeutics. J Cell Physiol;234(10):16812-16823.
3. **Farkhondeh, T., Folgado, S. L., Pourbagher-Shahri, A. M.,**

Ashrafizadeh, M., & Samarghandian, S. (2020): The therapeutic effect of resveratrol: Focusing on the Nrf2 signaling pathway. *Biomedicine & Pharmacotherapy*, *127*, 110234.

4. ***Macvanin, M. T., Rizzo, M., Radovanovic, J., Sonmez, A., Paneni, F., & Isenovic, E. R. (2022):*** Role of Chemerin in Cardiovascular Diseases. *Biomedicines*, *10*(11), 2970.
5. **Radzik-Zając, J., Wytrychowski, K., Wiśniewski, A., & Barg, W. (2022):** The role of the novel adipokines vaspin and omentin in chronic inflammatory diseases. *Pediatric Endocrinology Diabetes and Metabolism*, *28*(1).
6. **Garcia-Labbé D, Ruka E, Bertrand OF, Voisine P, Costerousse O, Poirier P. (2015):** Obesity and coronary artery disease: evaluation and treatment. Can J Cardiol ;31(2):184-94.
7. **Yusuf, S., Joseph, P., Rangarajan, S., Islam, S., Mente, A., Hystad, P.,& Dagenais, G. (2020):** Modifiable risk factors, cardiovascular disease, and mortality in 155 722 individuals from 21 high-income, middle-income, and low-income countries (PURE): a prospective cohort study. The Lancet, 395(10226), 795-808.
8. **Zhao, A., Xiao, H., Zhu, Y., Liu, S., Zhang, S., Yang, Z.,& Tian, Y. (2022):** Omentin-1: a newly discovered warrior against metabolic related diseases. Expert Opinion on Therapeutic Targets, 26(3), 275-289. **10- El-Gaphar, O. A. M. A., Abo-Youssef, A. M., & Abo-Saif, A. A. (2018).** Effect of losartan in complete freund's adjuvant–induced arthritis in rats. Iranian Journal of Pharmaceutical Research: IJPR, 17(4), 1420.
9. **Léniz, A., González, M., Besné, I., Carr-Ugarte, H., Gómez-García, I., & Portillo, M. P. (2022):** Role of chemerin in the control of glucose homeostasis. Molecular and Cellular Endocrinology, 541, 111504.
10. **Siegrist, M., Heitkamp, M., Braun, I., Vogg, N., Haller, B., Langhof, H., & Halle, M. (2021):** Changes of omentin-1 and chemerin during 4 weeks of lifestyle intervention and 1-year follow-up in children with obesity. Clinical Nutrition, 40(11), 5648-5654.
11. **Baig, M., Alghalayini, K. W., Gazzaz, Z. J., & Atta, H. (2020):** Association of serum omentin-1, chemerin, and leptin with acute myocardial infarction and its risk

factors. Pakistan Journal of Medical Sciences, 36(6), 1183.

12. M**alakar AK, Choudhury D, Halder B, Paul P, Uddin A, Chakraborty S. A (2019):** review on coronary artery disease, its risk factors, and therapeutics. J Cell Physiol;234(10):16812-16823.
13. **Mohammad, M. B., Sallam, A. S., Fekry, A. A., & Amin, M. I. (2021):** Study of Omentin-1 and Chemerin as Predictors of Coronary Artery Disease in Elderly Patients, with Retrospective Studying of their Relation to Diabetes Mellitus Type 2. The Egyptian Journal of Hospital Medicine, 83(1), 995-1001.
14. **Quispe, R., Michos, E. D., Martin, S. S., Puri, R., Toth, P. P., Al Suwaidi, J.,& Elshazly, M. B. (2020):** High- Sensitivity C-Reactive Protein Discordance With Atherogenic Lipid Measures and Incidence of Atherosclerotic Cardiovascular Disease in Primary Prevention: The ARIC Study. Journal of the American Heart Association, 9(3), e013600.
15. **Sell H, Laurencikiene J, Taube A, Eckardt K, Cramer A, Horrighs A, Arner P & Eckel J (2009):** Che merin is a novel adipocyte-derived factor inducing insulin resistance in primary human skeletal muscle cells. Diabetes 58 2731–2740.
16. **Szpakowicz, A., Kaminski, K. A., Szpakowicz, M., Lapinska, M., Paniczko, M., Lawicki, S., & Kowalska, I. (2020):** Chemerin in coronary artery disease patients. *European Heart Journal*, *41*(Supplement_2), ehaa946-1263.
17. **Özkan, E. A., Sadigov, A., & Öztürk, O. (2022):** Evaluation of serum omentin-1, vaspin, leptin, adiponectin levels in obese/overweight children and their relationship with non-alcoholic fatty liver disease. Clinical Nutrition Research, 11(3), 194.
18. **Mirmajidi, S., Izadi, A., Saghafi-Asl, M., Vahid, F., Karamzad, N., Amiri, P., & Hébert, J. R. (2019):** Inflammatory potential of diet: association with chemerin, omentin, lipopolysaccharide-binding protein, and insulin resistance in the apparently healthy obese. Journal of the American College of Nutrition, 38(4), 302-310.