

Estimation of Omentin-1 and some biochemical parameters in Iraqi patients with Myocardial infarction

Eman Mohamed Ahmed Ali¹, Khalid F. A. Alrawi¹, Hazim Ghazzay²

^{1, 2}Department of Chemistry, College of Science, University of Anbar, Iraq

Email: ema20s3021@uoanbar.edu.iq

Abstract

Background Omentin-1 is a novel adipokine found in visceral adipose tissue that plays a significant role in the progression of chronic inflammatory diseases such as coronary artery disease. **Methods** We enrolled 50 patients with MI and 40 healthy controls. We measure serum omentin-1, high sensitive cardiac Troponin-T (hs-cTnT), creatine phospho kinase-MB (CPK-MB), as well as other parameters. **Results:** Serum omentin-1 levels were significantly decreased in MI patients compared to controls (16.14 vs 37.93) ng/mL. There was a significant increase in serum levels of hs-cTnT (pg/mL) and CPK-MB(IU/L) in MI patients than in control group (1406 vs 3.143) (107.9 vs 11.46). **Conclusions** Serum omentin-1 levels were significantly decreased in MI patient compared to controls.

Keywords: Myocardial infarction, Omentin-1, Adipose tissue

1. Introduction

Myocardial infarction (MI) is one of the most serious vascular diseases, resulting in ischaemia caused by coronary artery occlusion [1]. MI is characterized by myocardial cell death brought on by sustained ischaemia and it is diagnosed by electrocardiographic (ECG) findings and increased levels of biomarkers of myocardial necrosis [2]. Omentin-1, also identified as intelectin-1, was found to be abundantly expressed in human omental adipose tissue [3] It is a novel adipokine discovered in a visceral omental adipose tissue cDNA library in 2003 [4]. Moreover, omentin-1 has been shown to increase insulin-stimulated glucose uptake in adipose tissues as well as to activate the protein kinase Akt/PKB [5]. Omentin-1 levels are positively correlated with insulin sensitivity but negatively correlated with body mass index and fat mass [6]. Additionally, omentin-1 blood levels were found to be lower in individuals with coronary artery disease [7] Omentin-1 is recognized as contributing to the immune system's response, which suggests that it may have anti-inflammatory effects. [8] It has cardiovascular protection and anti-atherosclerotic properties. According to studies, Omentin-1 causes vasodilation by increasing endothelial nitric oxide synthase and inhibiting tumor necrosis factor (TNF) [9]. Omentin-1 is considered to have a protective function in decreasing the risk of atherosclerosis. Reduced omentin-1 levels have been associated with stroke severity and could be an independent indicator of poor stroke outcome [10]. Additionally, omentin-1 activated the AMP-activated protein

kinase (AMPK) signaling pathway, which prevented TNF-induced endothelial cell inflammation and reversed ischemic myocardium damage [11]. Serum omentin-1 levels were decreased in most types of ischemic heart disease compared to healthy controls [12]. While adequate levels of omentin-1 enhance the maintenance of heart health and function, low levels of omentin-1 may contribute to the progression of atherosclerosis. Plasma omentin-1 may therefore be used as a biomarker to assess the progression of coronary artery disease [13]. Creatine kinase-MB fraction is a cardiac biomarker that can be used to help diagnose myocardial infarction. Its levels can be recognized three to eight hours following the onset of chest pain [14]. Increased serum cardiac troponin (cTn) concentration levels are currently the most common diagnostic biomarker for the early diagnosis of myocardial infarction and are considered to be indicative of myocardial injury leading to myocyte necrosis [15].

Present study was aimed to determine the serum levels of omentin-1 in MI Iraqi patients and to study the correlation of omentin-1 and some parameters that help in diagnosing MI.

2. Materials and Methods

Fifty MI patients who were admitted to Al-Ramadi Teaching Hospital (Al-Anbar Governorate) between November 2021 and April 2022 were included in this study. aged between 41 and 68. Positive troponin assessments, along with myocardial infarction-related ECG findings and clinical signs, were used to diagnose the patients. Our test group included The forty healthy controls were chosen from people

without ischemic heart disease who also had no history of smoking or drinking alcohol. They were the same gender and age as the patient groups. Each subject had a venous blood sample taken, which was centrifuged for five minutes at 4000 rpm. Eppendorf tubes were used to separate the samples into different parts, which were then kept at -20 °C pending biochemical assessment. Anthropometric measurements were performed for all participants in the study. BMI was calculated by dividing weight (kg) by height squared (m²). The following parameters were measured using commercial kits from Roche, Switzerland: hs-cTnT, CPK-MB, Urea, and Creatinine, while serum levels of omentin-1 were determined by ELISA technique (Melson Company, China).

Statistics

We used GraphPad Prism 7 to analyze our data. The

data were presented using the mean, standard error of the mean (SEM), and standard deviation (SD) values. To assess how different means differed from one another, a students' t-test was employed. Pearson correlation coefficients were used to analyze bivariate associations. The accuracy of the investigation was evaluated using the area under the ROC curve. The significance level was set at P 0.05

3. Results

our results showed that the mean of age has no significant difference in patients group (53.98 vs 51.33) as compared with control group. As shown in table 1 and (figure 1) While in the patients group, the mean BMI significantly increased (31.1 vs 23.38) kg/m² more than in the healthy controls.as shown in table 1 (Figure 2).

Parameter	Healthy controls			MI patients			p-value
	Mean	SD	SEM	Mean	SD	SEM	
Age years	51.33	8.395	1.327	53.98	8.22	1.163	0.1351
BMI kg/m ²	23.38	1.155	0.1826	31.1	4.442	0.6282	<0.0001
hs-cTnT pg/mL	3.143	0.8289	0.1311	1406	472.5	66.82	<0.0001
CPK-MB IU/L	11.46	3.24	0.5123	107.9	45.82	6.48	<0.0001
Urea mg/dL	24.85	6.351	1.004	41.84	20.88	2.953	<0.0001
Creatinine mg/dL	0.7275	0.1281	0.02025	0.92	0.3597	0.05087	0.0018
Omentin-1 ng/mL	37.93	8.004	1.266	16.14	4.587	0.6487	<0.0001

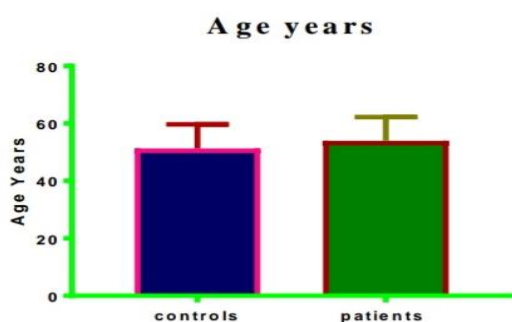


Figure 1. Mean+S.D. for Age in control and patients.

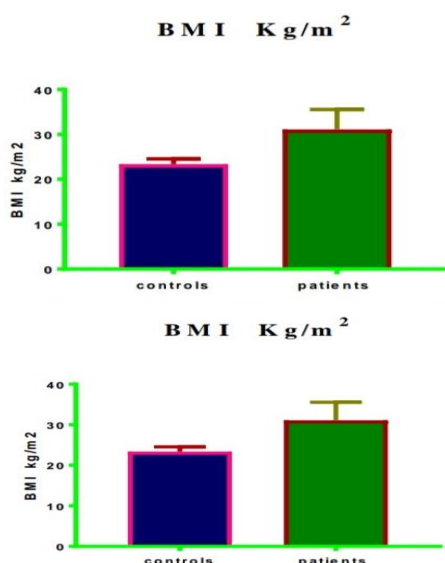


Figure 2. Mean+S.D. for BMI in control and patients.

Also Results of this study showed a significant increase (p < 0.05) in serum levels of hs-cTnT (pg/mL) and CPK-MB(IU/L) in MI patients than in control group (1406 vs 3.143) (107.9 vs 11.46) respectively, as shown in Figures (3,4).

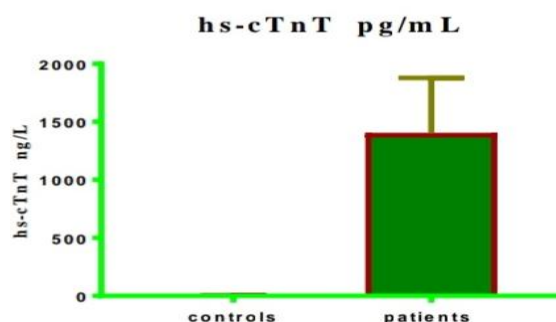


Figure 3. Mean+S.D. for hs-cTnT in control and patients.

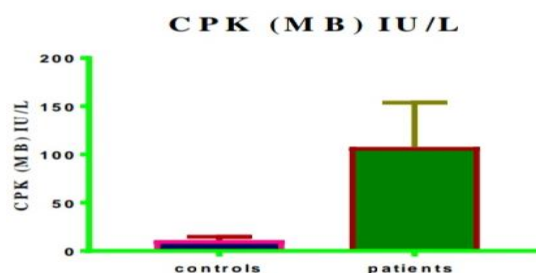


Figure 4. Mean+S.D. for CPK-MB in control and patients.

Our statistical results showed that serum omentin-1 levels significantly ($p < 0.05$) lower in patients' group (16.14 vs 37.93) ng/mL compared to the control group as shown in table 1 and Figure 5.

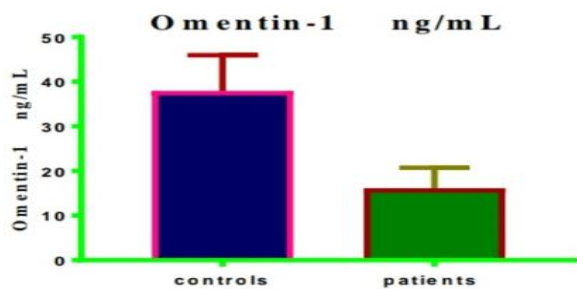


Figure 5. Mean+S.D. for Omentin-1 in control and patients.

The findings of this study showed that there was significant difference ($p < 0.05$) in level of urea in MI patients (41.84 vs 24.85) mg/dL compared with control group. as shown in Figure 6
The current study also showed there was significant difference ($p < 0.05$) in level of creatinine between MI patients and control group (0.92 vs 0.7275) IU/L as shown in Figure 7.

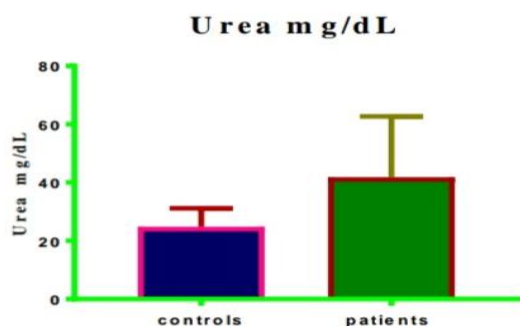


Figure 6. Mean+S.D. for Urea in control and patients.

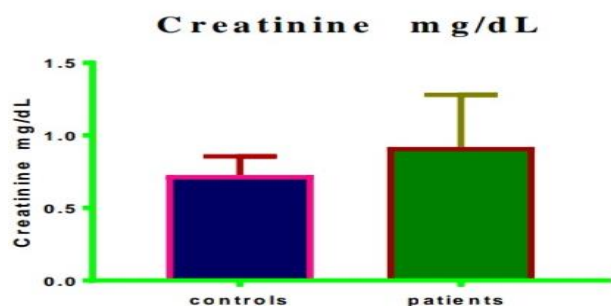


Figure 7. Mean+S.D. for Omentin-1 in control and patients

Parameter	r (Omentin-1 ng/mL)	p-value
hs-cTnT pg/mL	-0.794	<0.0001
CPK-MB IU/L	-0.687	<0.0001
Urea mg/dL	-0.339	0.001
Creatinine mg/dL	-0.262	0.012
BMI kg/m ²	-0.653	<0.0001
Age years	-0.134	0.210

As shown in table 2. Omentin-1 showed a strong negative correlation with hs-cTnT (Figure 8) and CPK-MB (Figure 9) ($r = -0.794$ at $p < 0.0001$) ($r = -0.687$ at $p < 0.0001$) respectively. We also observed a negative correlation between Omentin-1 with BMI (Figure 10) ($r = -0.653$ at $p < 0.0001$). there was a weak negative correlation between Omentin-1 with Urea ($r = -0.339$ at $p < 0.0001$). However, no correlation was observed between Omentin-1 with Age and Creatinine.

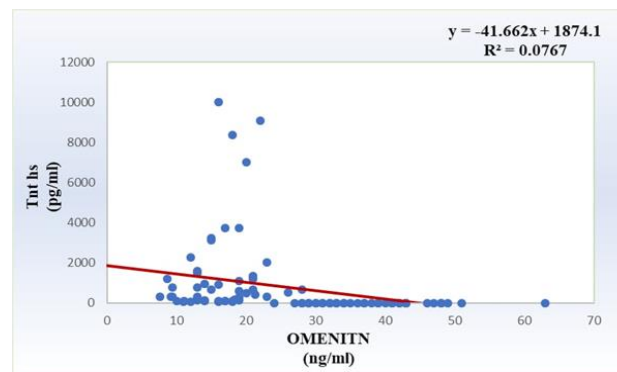


Fig. 8: Correlation between omentine-1 and hs-cTnT

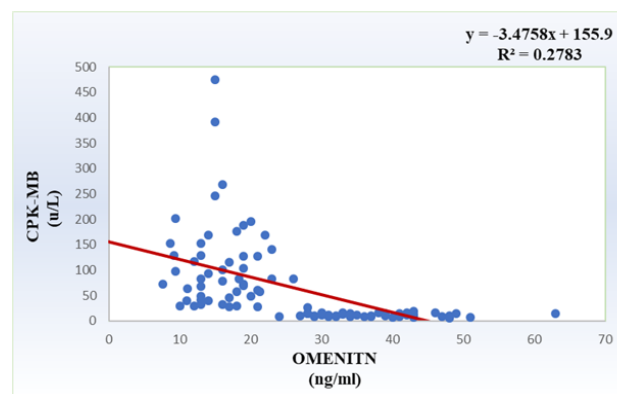


Fig. 9: Correlation between omentine-1 and CPK-MB

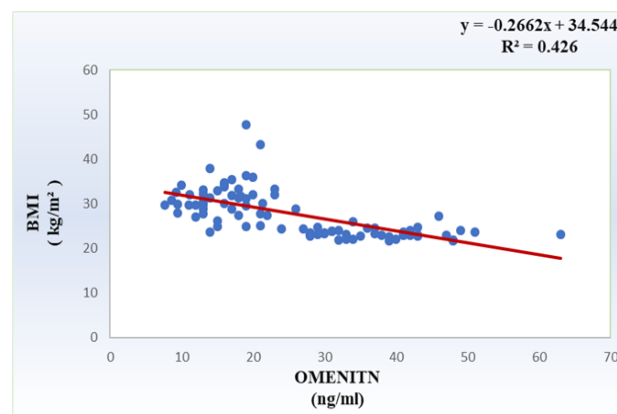


Fig. 10: Correlation between omentine-1 and BMI

Receiver Operating Characteristic Curve Analysis

The receiver operating characteristic (ROC) curve and the area under the curve (AUC) were used as an efficient measurement of accuracy in this study. This curve is critical in selecting the most appropriate cut-off values for evaluating test diagnostic ability to recognize subjects' true state [16].

Table 3. Standard of ROC Curves for Tested Variables in MI Patients				
Parameter	AUC	Std. Error	95% confidence interval	P-value
Age years	0.594	0.0606	0.4752 to 0.7128	0.1269
BMI Kg/m ²	0.9795	0.01276	0.9545 to 1.005	<0.0001
Troponin-T pg/mL	1	0	1 to 1	<0.0001
CPK-MB IU/L	1	0	1 to 1	<0.0001
Urea mg/dL	0.827	0.04351	0.7417 to 0.9123	<0.0001
Creatinine mg/dL	0.6443	0.05803	0.5305 to 0.758	0.0191
Omentin-1 ng/mL	1	0	1 to 1	<0.0001

Table 3. shows the result of ROC curve analysis showing which biomarkers were most effective at distinguishing patients with MI from the control group. The parameters Omentin-1, hs-cTnT and CPK-MB were among the standards with the highest validity and displayed an excellent strategy for discriminating between healthy people and patients with MI, [AUC= 1 P < 0.0001, 95% Confidence Interval (CI): 1 to 1 and SE: 0] figures (11,12,13) Additionally, it can be suggested that the BMI index value is evidence on the risk of obtaining diseases with [AUC = 0.9795, p <0.0001 95% Confidence Interval (CI): 0.9545 to 1.005 and SE: 0.01276] Figure 14

While Urea value is a very important parameter with a value of [AUC = 0.827, p <0.0001, 95% Confidence Interval (CI):0.7417 to 0.9123 and SE:0.04351] Figure (15). Finally, age and creatinine were among the factors with the lowest predictive validity for MI. [AUC = 0.594, p = 0.1269 95% Confidence Interval (CI) 0.4752 to 0.7128 and SE: 0.0606], Creatinine [AUC = 0.6443, p = 0.0191 95% Confidence Interval (CI): 0.5305 to 0.758, and SE: 0.05803]. Figures (16,17)

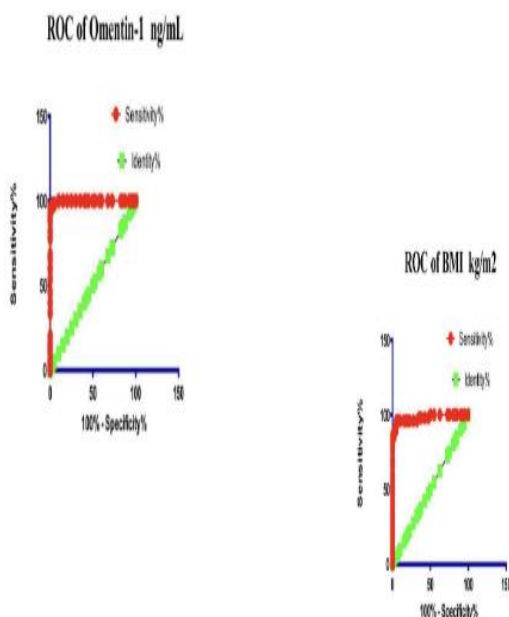


Figure 11. Area under Curve of omentine-1 in MI Patients.

ROC of CPK (MB) IU/L

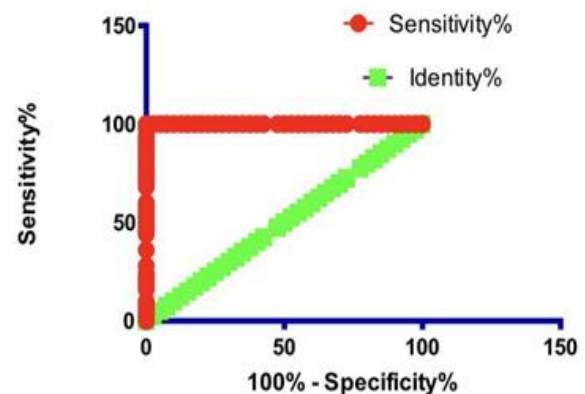


Figure 13. Area under Curve of CPK-MB in MI Patients.

ROC of hs-cTnT pg/mL

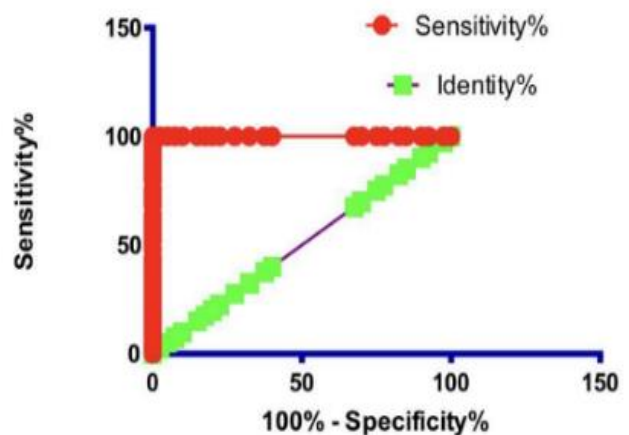


Figure 14. Area under Curve of BMI in MI Patients.

ROC of Urea mg/dL

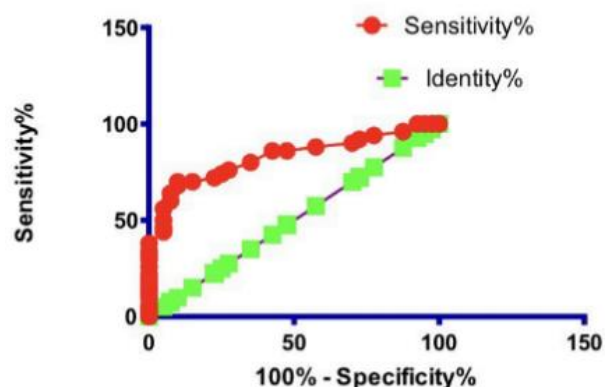


Figure 15. Area under Curve of Urea in MI Patients.

ROC of Age Years

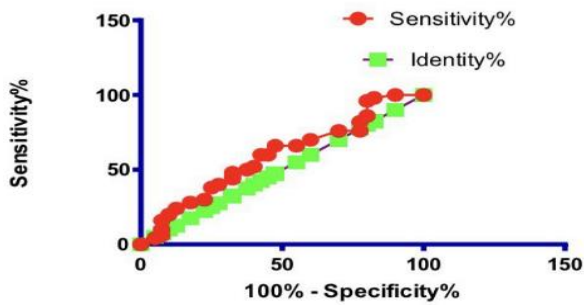


Figure 16. Area under Curve of Age in MI Patients.

ROC of Creatinine mg/dL

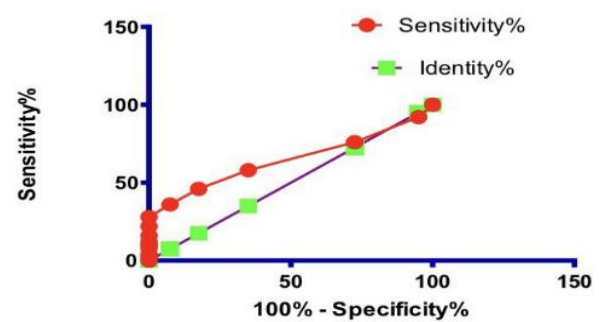


Figure 17. Area under Curve of creatinine in MI Patients.

4. Discussion

Omentin-1 is a strong anti-inflammatory and adipocyte that is released from the visceral adipose tissue and contributes to the homeostasis of energy state, glucose metabolism, and anti-atherogenic impacts [17]. Omentin-1 levels have been observed to significantly decrease in people with obesity, insulin resistance, and diabetes mellitus [18] [19]. Our findings are in agreement with those of another study on MI patients, which found a significant decrease in omentin-1 levels [20]. Omentin-1 levels were found to be significantly lower in patients with cardiovascular diseases, according to a recent study. Omentin-1 may therefore be a good predictor of cardiovascular diseases (CVD) [21]. As atherosclerosis develops and progresses, adipocytokines produced by adipose tissue are known to be crucial. Patients with severe coronary artery disease (CAD) had significantly higher levels of pro-inflammatory cytokines in their epicardial adipose tissue [22]. Additionally, study findings showed that serum IL-6 levels were an independent risk factor which affects omentin-1 levels. This observation demonstrates that omentin-1 is regulated by inflammation [5]. Omentin-1 prevents the release of pro-inflammatory cytokines by endothelial cells in blood vessels, which are important in the development of endothelial dysfunction and arterial calcification [23]. In our study omentin-1 correlates negatively with BMI, hs-cTnT and CPK-MB. our results are in agreement with other studies have suggested a negative correlation between omentin-1 levels with BMI [24] [19].

5. Conclusions

According to this study, omentin-1 and myocardial infarction have a negative correlation. We hypothesize that low levels of omentin-1 may contribute to the pathogenesis of atherosclerosis. As a result, this finding explains the importance of this

biomarker. Serum omentin-1 may be a useful biomarker for predicting the onset and course of MI. Furthermore, levels of hs-cTnT and CPK-MB showed a strong positive correlation with blood levels of omentin-1, indicating that these levels of omentin-1 could be used as novel therapeutic targets for atherosclerosis and myocardial infarction.

References

- [1] V. Nikolic-Heitzler et al., "Persistent oxidative stress after myocardial infarction treated by percutaneous coronary intervention," *Tohoku J. Exp. Med.*, vol. 210, no. 3, pp. 247–255, 2006.
- [2] E. S. C. C. for P. G. (CPG) et al., "Third universal definition of myocardial infarction," *J. Am. Coll. Cardiol.*, vol. 60, no. 16, pp. 1581–1598, 2012.
- [3] M. Nishimura et al., "Plasma omentin levels are inversely associated with atherosclerosis in type 2 diabetes patients with increased plasma adiponectin levels: a cross-sectional study," *Cardiovasc. Diabetol.*, vol. 18, no. 1, pp. 1–10, 2019.
- [4] R. Z. Yang et al., "Cloning of omentin, a new adipocytokine from omental fat tissue in humans," in *Diabetes*, 2003, vol. 52, pp. A1–A1.
- [5] A. Schäffler, M. Neumeier, H. Herfarth, A. Fürst, J. Schölmerich, and C. Büchler, "Genomic structure of human omentin, a new adipocytokine expressed in omental adipose tissue," *Biochim. Biophys. Acta (BBA)-Gene Struct. Expr.*, vol. 1732, no. 1–3, pp. 96–102, 2005.
- [6] S. Greulich et al., "Cardioprotective Properties of Omentin-1 in Type 2 Diabetes: Evidence from Clinical and In Vitro Studies," *PLoS One*, vol. 8, no. 3, 2013, doi: 10.1371/journal.pone.0059697.
- [7] R. Shibata et al., "Circulating omentin is associated with coronary artery disease in men," *Atherosclerosis*, vol. 219, no. 2, pp. 811–814, 2011.
- [8] J. Lesná et al., "Omentin-1 plasma levels and cholesterol metabolism in obese patients with diabetes mellitus type 1: Impact of weight reduction," *Nutr. Diabetes*, vol. 5, no. November, 2015, doi: 10.1038/nutd.2015.33.
- [9] M. Çelik, R. Nar, G. Nar, E. Sökmen, and G. Günver, "Serum omentin-1 levels in hypertensive

- patients," *J. Hum. Hypertens.*, vol. 35, no. 3, pp. 290–295, 2021, doi: 10.1038/s41371-020-00420-4.
- [10] D. M. Wu et al., "Impact of serum omentin-1 levels on functional prognosis in nondiabetic patients with ischemic stroke," *Am. J. Transl. Res.*, vol. 11, no. 3, pp. 1854–1863, 2019.
- [11] Y. Chen, F. Liu, F. Han, L. Lv, C. E. Tang, and F. Luo, "Omentin-1 Ameliorated Free Fatty Acid-Induced Impairment in Proliferation, Migration, and Inflammatory States of HUVECs," *Cardiol. Res. Pract.*, vol. 2020, 2020, doi: 10.1155/2020/3054379.
- [12] H. M. Alkuraishy and A. I. Al-Gareeb, "New Insights into the Role of Metformin Effects on Serum Omentin-1 Levels in Acute Myocardial Infarction: Cross-Sectional Study," *Emerg. Med. Int.*, vol. 2015, pp. 1–8, 2015, doi: 10.1155/2015/283021.
- [13] P. Bai et al., "Association Between Coronary Artery Disease and Plasma Omentin-1 Levels," *Cureus*, vol. 13, no. 8, pp. 8–11, 2021, doi: 10.7759/cureus.17347.
- [14] D. A. D. Goel, "Study on Correlation between High Sensitivity C-Reactive Protein Levels and Left Ventricular Function in Patients with Acute Coronary Syndrome," *J. Med. Sci. Clin. Res.*, vol. 08, no. 04, pp. 102–108, 2020, doi: 10.18535/jmscr/v8i4.19.
- [15] B. R. Weil et al., "Brief myocardial ischemia produces cardiac troponin I release and focal myocyte apoptosis in the absence of pathological infarction in swine," *Basic to Transl. Sci.*, vol. 2, no. 2, pp. 105–114, 2017.
- [16] K. Hajian-Tilaki, "Receiver operating characteristic (ROC) curve analysis for medical diagnostic test evaluation," *Casp. J. Intern. Med.*, vol. 4, no. 2, p. 627, 2013.
- [17] M. Yasir, G. P. Senthilkumar, K. Jayashree, K. Ramesh Babu, M. Vadivelan, and C. Palanivel, "Association of serum omentin-1, apelin and chemerin concentrations with the presence and severity of diabetic retinopathy in type 2 diabetes mellitus patients," *Arch. Physiol. Biochem.*, vol. 128, no. 2, pp. 313–320, 2022.
- [18] I. Castan-Laurell, C. Dray, C. Knauf, O. Kunduzova, and P. Valet, "Apelin, a promising target for type 2 diabetes treatment?," *Trends Endocrinol. Metab.*, vol. 23, no. 5, pp. 234–241, 2012.
- [19] B. K. Tan et al., "Omentin-1, a novel adipokine, is decreased in overweight insulin-resistant women with polycystic ovary syndrome: ex vivo and in vivo regulation of omentin-1 by insulin and glucose," *Diabetes*, vol. 57, no. 4, pp. 801–808, 2008.
- [20] M. Baig, K. W. Alghalayini, Z. J. Gazzaz, and H. Atta, "Association of serum omentin-1, chemerin, and leptin with acute myocardial infarction and its risk factors," *Pakistan J. Med. Sci.*, vol. 36, no. 6, p. 1183, 2020.
- [21] J. Wang, X. Zhuo, and Z. Jiang, "Omentin-1 circulating levels as predictor of heart diseases: a systematic review and meta-analysis," *Rev. Assoc. Med. Bras.*, vol. 68, pp. 542–548, 2022.
- [22] T. Mazurek et al., "Human epicardial adipose tissue is a source of inflammatory mediators," *Circulation*, vol. 108, no. 20, pp. 2460–2466, 2003.
- [23] F. Zabetian-Targhi, K. Mirzaei, S. A. Keshavarz, and A. Hossein-Nezhad, "Modulatory role of omentin-1 in inflammation: cytokines and dietary intake," *J. Am. Coll. Nutr.*, vol. 35, no. 8, pp. 670–678, 2016.
- [24] C. M. de Souza Batista et al., "Omentin plasma levels and gene expression are decreased in obesity," *Diabetes*, vol. 56, no. 6, pp. 1655–1661, 2007.