Review began 07/27/2021
Review ended 08/17/2021
Published 08/21/2021

© Copyright 2021

Bai et al. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Association Between Coronary Artery Disease and Plasma Omentin-1 Levels

Priya Bai ¹, FNU Abdullah ², Maham Lodi ³, Mayur Sarhadi ⁴, Anum Dilip ⁵, Sibghatullah Shahab ⁵, FNU Yasir ⁵, Maha Jahangir ⁶

1. Internal Medicine, People's University of Medical and Health Sciences for Women, Nawabshah, PAK 2. Neurology, Jinnah Sindh Medical University, Karachi, PAK 3. Internal Medicine, Jinnah Sindh Medical University, Karachi, PAK 4. Internal Medicine, Jinnah Medical and Dental College, Karachi, PAK 5. Internal Medicine, Baqai Medical University, Karachi, PAK 6. Internal Medicine, Dow University of Health Sciences, Karachi, PAK

Corresponding author: Maha Jahangir, jahangirmaha@yahoo.com

Abstract

Introduction: Omentin-1 is secreted from visceral adipose tissue that contributes to chronic inflammatory diseases' pathogenesis, including cardiovascular events. In this case-control study, we will determine the association between plasma omentin and coronary artery disease (CAD).

Methods: This is a case-control study, conducted from June 2020 to April 2021 in the cardiology unit of a tertiary care hospital in Pakistan. Patients diagnosed with CAD (n = 300) within the last six months were included in the study. Another 300 participants without CAD and with similar demographic profiles were included in the control group. A blood sample of 5 ml was drawn from participants of both groups and sent to the laboratory to test for plasma omentin level.

Results: Plasma omentin level was significantly lower in patients with CAD compared to the patients without CAD (61.21 ± 10.21 ng/dL vs. 95.22 ± 12.21 ug/L; p-value: <0.0001). In both genders, the plasma omentin-1 was lower in patients with CAD compared to patients without CAD (p-value: <0.0001).

Conclusion: The present study revealed a negative association between omentin-1 and CAD. We speculate that low levels of omentin-1 might play a role in the pathogenesis of atherosclerosis. Therefore, plasma omentin-1 can be a potential biomarker to predict the development and progression of CAD.

Categories: Cardiology, Internal Medicine, Pathology

Keywords: cardiovascular disease, coronary artery disease, cad, omentin-1, intellectin-1

Introduction

Coronary artery disease (CAD) is a cardiovascular disease (CVD) that is found to be the leading cause of death in both developed and developing worlds [1]. It is characterized by atherosclerotic changes, which are inflammatory in nature. Along with genetic and environmental factors, lifestyle has been found to play an important role in the investigation of the clinical phenotype of CVD. Several risk factors have been associated with the development of CAD, such as hypertension, smoking, diabetes, and hyperlipidemia [2-5].

Measurement of biomarkers has revolutionized the work-up of patients with suspected cardiovascular disease. The most widely used are the natriuretic peptides in the diagnosis and prognosis of heart failure and cardiac troponins in the diagnosis of acute myocardial infarction.

A broad range of biomarkers is associated with cardiovascular risks. The natriuretic peptides and cardiac troponins are among the most widely used contemporary cardiovascular biomarkers that help in the diagnosis and prognosis of CVDs. The scientific advances have led to the discovery of a vast array of novel biomarkers associated with cardiovascular risks, including myeloperoxidase, lipoprotein-associated phospholipase A2, fibrinogen, trimethylamine N-oxide, and cystatin C [6]. Another novel biomarker for CAD is plasma omentin-1 [7].

Like other adipokines, omentin-1 is expressed from visceral adipose tissue that contributes to chronic inflammatory diseases' pathogenesis, including cardiovascular events. Several studies have demonstrated that circulating omentin-1 levels are associated with several metabolic risk factors, such as high blood pressure, dyslipidemia, and glucose intolerance [7]. However, there are contradictory data available. In his study, Saely et al. state that patients within the high omentin group reported significantly more cardiovascular events than patients in the low omentin group [7]. Askani et al. stated that a decrease in the omentin levels is an important predictor of CAD and its severity [8]. Zhou et al. also found a correlation between higher plasma omentin-1 level and better coronary collateral circulation development [9].

How to cite this article

Bai P, Abdullah F, Lodi M, et al. (August 21, 2021) Association Between Coronary Artery Disease and Plasma Omentin-1 Levels. Cureus 13(8): e17347. DOI 10.7759/cureus.17347

Currently, very little data are available globally and regionally related to the correlation between plasma omentin and CAD. In this case-control study, we will determine the association between the two.

Materials And Methods

This is a case-control study, conducted from June 2020 to April 2021 in the cardiology unit of a tertiary care hospital in Pakistan. CAD was defined as damage to the major blood vessels of the heart secondary to atherosclerotic changes and inflammation. Patients diagnosed with CAD (n = 300) within the last six months were included in the study. Diagnosis of CAD was made by an interventional cardiologist via angiography. Another 300 participants without CAD and with similar demographic profiles were included in the control group. Informed consent was taken from each participant after the entire process was explained to them. Case and control groups both were enrolled using a consecutive convenient non-probability sampling technique. The process of enrollment was started only after the approval from the ethical review board of the institute.

After the process of enrollment, participants were asked about their age, gender, body mass index (BMI), and relevant history. A blood sample of 5 ml was drawn from participants of both case and control groups from the cubital vein and sent to the laboratory to test for plasma omentin level. The blood sample was collected in the morning to avoid diurnal fluctuation. Plasma omentin was assessed using an enzyme-linked immune-sorbent assay.

Data were analyzed using the statistical software Statistical Packages for Social Sciences (SPSS) v. 23.0 (IBM Corporation, Armonk, New York). Chi-square and unpaired t-test were applied to compare the parameters of two groups. A p-value of less than 0.05 meant that there is a significant difference in the value between the two groups and the null hypothesis is void.

Results

In this study, no significant difference in age and gender distribution was found between the two groups. Risk factors, including diabetes, hypertension, smoking, and BMI more than 25 kg/m², were comparable in both groups and no significant difference was found between them (Table 1).

Characteristics	Patients with CAD (n = 300)	Patients without CAD (n = 300)	p-value
Age in years (mean ± SD)	52 ± 10	53 ± 10	NS
Male	181 (60.33%)	186 (62.0%)	NS
Hypertension	281 (93.66%)	283 (94.3%)	NS
Diabetes	154 (51.3%)	160 (53.3%)	NS
Smoking	112 (37.3%)	116 (38.6%)	NS
BMI more than 25 kg/m ²	131 (43.6%)	128 (42.6%)	NS

TABLE 1: Characteristics of the study participants

BMI: body mass index; CAD: coronary artery disease; kg/m²: kilogram per square meter; NS: nonsignificant; SD: standard deviation.

Plasma omentin level was significantly lower in patients with CAD compared to the patients without CAD (61.21 ± 10.21 ng/dL vs. 95.22 ± 12.21 ng/dL; p-value: <0.0001). For males, plasma omentin-1 was lower in patients with CAD compared to patients without CAD (61.78 ± 10.12 ng/dL vs. 95.23 ± 12.17 ng/dL; p-value: <0.0001). Similarly, plasma omentin-1 was lower in female patients with CAD compared to their counterparts (60.98 ± 10.02 ng/dL vs. 94.80 ± 12.04 ng/dL; p-value: <0.0001) (Table 2).

Omentin-1 levels (mean ± SD ng/dL)	Patients with CAD (n = 300)	Patients without CAD (n = 300)	p-value
Total omentin-1	61.21 ± 10.21	95.22 ± 12.21	<0.0001
Male	61.78 ± 10.12	95.23 ± 12.17	<0.0001
Female	60.98 ± 10.02	94.80 ± 12.04	<0.0001

TABLE 2: Gender-wise comparison of omentin-1 levels in patients with and without CAD

CAD: coronary artery disease; ng/dL: nanogram per decilitre; SD: standard deviation.

Discussion

Our study demonstrated that patients with CAD reported lower levels of omentin-1 than those who did not have CAD. Literature has enough evidence in concordance with our results. A cross-sectional study conducted by Shang et al. included metabolic syndrome (MetS) patients to analyze the association between omentin-1 level and CAD [10]. It was suggested that MetS patients with CAD were reported to have lower levels of omentin-1 compared to those who did not have CAD. Additionally, omentin-1 showed a negative association with the presence and angiographic severity of CAD [10]. In recent years, studies support the evidence that omentin-1 could potentially be the reason behind the pathology of atherosclerosis. Liu et al. also established a negative link between omentin-1 level and carotid atherosclerosis in MetS patients [11].

Adipose tissues primarily help in conserving energy and also demonstrate endocrine functions in producing and secreting several bioactive adipokines that further lead to CVDs [12]. Omentin-1, also known as intelectin-1, is abundantly expressed in intra-abdominal fat [13]. There is evidence that obese patients demonstrate low levels of plasma omentin-1, and its levels are negatively related to BMI, waist circumference, leptin levels, and insulin resistance [14]. Furthermore, omentin-1 is not positively linked with the presence and angiographic severity of CAD in MetS patients [10]. It also imposes neutralizing effects on atherosclerosis and ischemia-induced revascularization [15-17].

In light of the above findings, future studies including a larger sample size should be carried out to confirm our results. Our study suggests that plasma omentin-1 levels should be assessed at regular intervals in at-risk patients or those who have underlying cardiac issues. This would help in better management and monitoring of heart-related problems.

To the best of our knowledge, this was the first study that explored the association between plasma omentin-1 and CAD in a local setting. However, since the study was conducted in a single institute, sample size diversity was limited. Similarly, since it was a case-control study, the causal relationship between the two could not be confirmed. Therefore, more studies, including large and diverse sample sizes, should be conducted to explore and establish the association between plasma omentin-1 and CAD.

Conclusions

In conclusion, the present study revealed a negative association between omentin-1 and CAD. We speculate that low levels of omentin-1 might play a role in the pathogenesis of atherosclerosis and adequate levels of omentin-1 are beneficial in maintaining a healthy state of the heart and its function. Therefore, plasma omentin-1 can be a potential biomarker to predict the development and progression of CAD.

Additional Information

Disclosures

Human subjects: Consent was obtained or waived by all participants in this study. Jinnah Sindh Medical University issued approval JSMU/IRB/2020/13. **Animal subjects:** All authors have confirmed that this study did not involve animal subjects or tissue. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. GBD 2013 Mortality and Causes of Death Collaborators: Global, regional, and national age-sex specific all-cause and cause-specific mortality for 240 causes of death, 1990-2013: a systematic analysis for the Global

- Burden of Disease Study 2013. *Lancet*. 2015, 385:117-71. [10.1016/S0140-6736\(14\)61682-2](https://doi.org/10.1016/S0140-6736(14)61682-2)
2. Yusuf S, Hawken S, Ounpuu S, et al.: Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. *Lancet*. 2004, 364:937-52. [10.1016/S0140-6736\(04\)17018-9](https://doi.org/10.1016/S0140-6736(04)17018-9)
3. Siasos G, Tsigkou V, Kokkou E, et al.: Smoking and atherosclerosis: mechanisms of disease and new therapeutic approaches. *Curr Med Chem*. 2014, 21:3936-48. [10.2174/092986732134141015161539](https://doi.org/10.2174/092986732134141015161539)
4. Henning RJ: Type-2 diabetes mellitus and cardiovascular disease . *Future Cardiol*. 2018, 14:491-509. [10.2217/fca-2018-0045](https://doi.org/10.2217/fca-2018-0045)
5. Cushman WC, Evans GW, Byington RP, et al.: Effects of intensive blood-pressure control in type 2 diabetes mellitus. *N Engl J Med*. 2010, 362:1575-85. [10.1056/NEJMoa1001286](https://doi.org/10.1056/NEJMoa1001286)
6. Huang Y, Gulshan K, Nguyen T, Wu Y: Biomarkers of cardiovascular disease . *Dis Markers*. 2017, 2017:8208609. [10.1155/2017/8208609](https://doi.org/10.1155/2017/8208609)
7. Saelly CH, Leihner A, Muendlein A, et al.: Coronary patients with high plasma omentin are at a higher cardiovascular risk. *Data Brief*. 2016, 6:158-61. [10.1016/j.dib.2015.11.065](https://doi.org/10.1016/j.dib.2015.11.065)
8. Askin L, Duman H, Ozyildiz A, Tanriverdi O, Turkmen S: Association between omentin-1 and coronary artery disease: pathogenesis and clinical research. *Curr Cardiol Rev*. 2020, 16:198-201. [10.2174/1573403X16666200511085304](https://doi.org/10.2174/1573403X16666200511085304)
9. Zhou JP, Tong XY, Zhu LP, et al.: Plasma omentin-1 level as a predictor of good coronary collateral circulation. *J Atheroscler Thromb*. 2017, 24:940-8. [10.5551/jat.37440](https://doi.org/10.5551/jat.37440)
10. Shang FJ, Wang JP, Liu XT, Zheng QS, Xue YS, Wang B, Zhao LY: Serum omentin-1 levels are inversely associated with the presence and severity of coronary artery disease in patients with metabolic syndrome. *Biomarkers*. 2011, 16:657-62. [10.3109/1354750X.2011.622789](https://doi.org/10.3109/1354750X.2011.622789)
11. Liu R, Wang X, Bu P: Omentin-1 is associated with carotid atherosclerosis in patients with metabolic syndrome. *Diabetes Res Clin Pract*. 2011, 93:21-5. [10.1016/j.diabres.2011.03.001](https://doi.org/10.1016/j.diabres.2011.03.001)
12. Li ZY, Wang P, Miao CY: Adipokines in inflammation, insulin resistance and cardiovascular disease . *Clin Exp Pharmacol Physiol*. 2011, 38:888-96. [10.1111/j.1440-1681.2011.05602.x](https://doi.org/10.1111/j.1440-1681.2011.05602.x)
13. Schäffler A, Neumeier M, Herfarth H, Fürst A, Schölmerich J, Büchler C: Genomic structure of human omentin, a new adipocytokine expressed in omental adipose tissue. *Biochim Biophys Acta*. 2005, 1732:96-102. [10.1016/j.bbaexp.2005.11.005](https://doi.org/10.1016/j.bbaexp.2005.11.005)
14. de Souza Batista CM, Yang RZ, Lee MJ, et al.: Omentin plasma levels and gene expression are decreased in obesity. *Diabetes*. 2007, 56:1655-61. [10.2337/db06-1506](https://doi.org/10.2337/db06-1506)
15. Watanabe K, Watanabe R, Konii H, et al.: Counteractive effects of omentin-1 against atherogenesis . *Cardiovasc Res*. 2016, 110:118-28. [10.1093/cvr/cvw016](https://doi.org/10.1093/cvr/cvw016)
16. De Jager SC, Pasterkamp G: Atheroprotective properties of human omentin-1 in experimental atherosclerosis. *Cardiovasc Res*. 2016, 110:1-3. [10.1093/cvr/cvw040](https://doi.org/10.1093/cvr/cvw040)
17. Maruyama S, Shibata R, Kikuchi R, et al.: Fat-derived factor omentin stimulates endothelial cell function and ischemia-induced revascularization via endothelial nitric oxide synthase-dependent mechanism. *J Biol Chem*. 2012, 287:408-17. [10.1074/jbc.M111.261818](https://doi.org/10.1074/jbc.M111.261818)