

Original Article

Association of adiponectin concentration with acute coronary syndrome types in diabetes mellitus: A cross-sectional study

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Abstract

Diabetes Mellitus (DM) increases the risk of acute coronary syndrome (ACS). Adiponectin has been popular as a potential indicator for DM and ACS. Hypoadiponectinemia has an important role in the pathogenesis of cardiovascular disease associated with metabolic syndrome. A cross-sectional analytical study with simple random sampling was conducted at Dr. Wahidin Hospital. The study samples were diagnosed with DM and ACS. Data analysis was performed using chi-square tests and odds ratios (ORs). This study involved 88 subjects with DM and ACS who fulfilled the study's eligibility criteria. The mean adiponectin concentration was 10.27 µg/ml. The subjects with the highest adiponectin concentration quartile (Q4) were those with STEMI (69.6%), and the difference was not statistically significant (chi-square, $p > 0.05$). There is a significant association between dyslipidemia and adiponectin concentration ($p < 0.05$), with the highest adiponectin concentration in the quartile (Q2) at 28.4%. Subjects with hypoadiponectinemia quartile (Q1–Q2) had 11.6 times higher risk of mortality (OR 11.647, 95% CI: 1.407–96.443, $p = 0.006$). There is no significant association between adiponectin concentration and the type of ACS in DM subjects. Dyslipidemia was identified as the predominant risk factor affecting adiponectin concentration. A significant association was found between adiponectin concentration and mortality from acute coronary syndrome in diabetic subjects.

Keywords: adiponectin, acute coronary syndrome, diabetes mellitus

Introduction

Diabetes Mellitus (DM) is associated with an increased risk of acute coronary syndrome (ACS). It is estimated that between 25% and 30% of patients admitted to hospitals with ACS have DM [1].

The latest International Diabetes Federation (IDF) DM Atlas in 2025 reports that 11.1% or 1 in 9 of the adult population (20–79 years) is living with DM, with over 4 in 10 unaware that they have the condition. According to IDF estimates, 1 in 8 adults, or 853 million people, would have diabetes by 2050, a 46% rise. The number

of adults with DM in South-East Asia (SEA) in 2024 is 106 million. IDF projects that the number of people with DM in the SEA Region will increase by 73%, reaching 185 million by 2050. According to the American Heart Association, adults with DM have a two to four times greater risk of developing heart disease than people who do not have diabetes [2–4].

DM is a chronic disease that occurs when the pancreas does not produce enough insulin. Insulin is a hormone that regulates blood glucose. Hyperglycemia is a common result of uncontrolled DM and, over time, leads to major cardiovascular and neurological



complications. ACS includes STEMI (ST-elevation myocardial infarction, N-STEMI (Non-ST-elevation myocardial infarction), and unstable angina. One of the primary causes of ACS is plaque rupture and thrombus formation, which reduces blood flow to specific areas of the heart muscle [5, 6].

Adiponectin is a homeostatic component that regulates glucose levels, lipid metabolism, and insulin sensitivity through its anti-inflammatory, anti-fibrotic, and antioxidant activities. It is primarily released by adipocytes in adipose tissue. Low adiponectin concentration (hypoadiponectinemia) is a risk factor for ACS, which can lead to atherosclerotic plaque rupture in DM [7–9].

A Cohort study did not find hypoadiponectinemia to be a predictor of mortality in individuals with ACS [10]. However, the majority of research indicates that individuals with hypoadiponectinemia have a lower risk of death compared to those with elevated baseline adiponectin concentrations [11, 12]. This study aims to determine the association of adiponectin concentration with types of ACS, adiponectin concentration with mortality, and to determine risk factors that have the biggest impact on adiponectin concentration in ACS and DM subjects.

Material and methods

Study population

This study was conducted at Dr. Wahidin Sudirohusodo Hospital from August to December 2024 using a cross-sectional design and an analytical observational study methodology. A total of 88 samples were collected, and all met the inclusion and exclusion criteria.

Inclusion and exclusion criteria

The study sample criteria are patients DM and ACS who meet the following criteria: (1) Patients agreed to several exams (2) not taking specific drugs that affect increased peroxisome proliferator-activated receptor gamma (PPAR- γ), which can increase insulin sensitivity (e.g. pioglitazone), (3) Subjects with stress hyperglycemia with HbA1c ≤ 6.5 mg/dl.

Clinical data and sample collection

The study carried out simple random sampling and included subjects with DM and ACS who met the criteria. DM is diagnosed based on fasting plasma glu-

cose ≥ 126 mg/dl, non-fasting plasma glucose ≥ 200 mg/dl with classic symptoms, HbA1c $\geq 6.5\%$, or a history of DM treatment. ACS is divided into 3 types: UAP, N-STEMI, and STEMI, which are identified by typical angina symptoms, electrocardiography (ECG) examination changes, and elevated cardiac biomarkers (troponin I/T or CK-MB). The Human Adiponectin ELISA reagent DBC kit (Diagnostics Biochem Canada Inc.) is used to measure the adiponectin concentration. Adiponectin values are divided into 4 quartiles with the following categories: quartiles 1–2 (Q1–Q2) ($\mu\text{g/ml}$) are low, and quartiles 3–4 (Q3–Q4) ($\mu\text{g/ml}$) are high. The risk factors studied that can affect adiponectin concentration are age, gender, obesity, smoking, hypertension, and dyslipidemia. Mortality refers to the number of deaths observed in a population, as reported by DM and ACS, during a specified study period.

Statistical analysis

The data is analyzed with SPSS version 25. The analysis method includes descriptive data and frequency distribution. General study sample information was obtained using descriptive methods. A Chi-Square and odds ratio test was implemented as the statistical analysis. Statistical test results were considered significant if the test *p*-value was < 0.05 .

Results

This study involved 88 Subjects with DM and ACS who fulfilled the inclusion and exclusion criteria. There were 60 male subjects (68.2%) and 28 female subjects (31.8%). Subjects aged ≥ 50 years were (89.8%), obese (61.4%), had hypertension (66%), smoked (48%), and had dyslipidemia (89.8%). The most common ACS type identified was STEMI (58%). The mean adiponectin concentration in this study was $10.27 \mu\text{g/ml}$. The fourth quartile (Q4) showed the highest percentage of subjects with STEMI (69.6%). A total of 10 subjects (11.4%) died during the study (Table 1).

Table 2 shows the distribution of ACS types according to adiponectin concentration quartile. The highest proportion of STEMI subjects was found in the fourth quartile (Q4) of adiponectin concentration (69.6%), and the highest proportion was found in the second quartile (Q2) (48.0%) in N-STEMI types. Statistical analysis revealed no significant differences ($p > 0.05$).

Table 3 shows the distribution of ACS types according to the adiponectin concentration category.

Table 1: The characteristics of the subjects.

No.	Variable	Category	Total		Min	Max	Mean	SD
			n	%				
1.	Gender	Male	60	68.2	38	84	60.98	8.88
		Female	28	31.8				
2.	Age (years)	≥50 years	79	89.8				
		<50 years	9	10.2				
3.	Obesity (BMI)	Yes	34	38.6	16	35	24.71	3.01
		No	54	61.4				
4.	Hypertension	Yes	66	75.0	1.48	75.30	10.27	10.29
		No	22	25.0				
5.	Smoking	Yes	48	54.5				
		No	40	45.5				
6.	Dyslipidemia	Yes	79	89.8				
		No	9	10.2				
		UAP*	7	8				
7.	ACS Type*	NSTEMI*	30	34.1				
		STEMI*	51	58.0				
		Q1 (≤4,273)	18	20.5				
8.	Adiponectin (µg/ml)	Q2 (4,274–7,380)	25	28.4				
		Q3 (7,381–11,949)	22	25				
		Q4 (≥11,950)	23	26.1				
9.	Mortality	Yes	10	11.4				
		No	78	88.6				

Note: UAP – Unstable angina pectoris; N-STEMI – non-ST elevation myocardial infarction; STEMI – ST elevation myocardial infarction; BMI – Body mass index; Q1 – Quartile 1; Q2 – Quartile 2; Q3 – Quartile 3; Q4 – Quartile 4.

Table 2: Association of adiponectin concentration quartiles and ACS types in DM.

No.	Adiponectin Quartile	n (%)	ACS types			Total	p
			UAP	N-STEMI	STEMI		
1.	Q1	n	3	4	11	18	0.392*
		%	16.7	22.2	61.1	100	
2.	Q2	n	1	12	12	25	
		%	4.0	48.0	48.0	100	
3.	Q3	n	2	8	12	22	
		%	9.1	36.4	54.5	100	
4.	Q4	n	1	6	16	23	
		%	4.3	26.1	69.6	100	
Total		n	7	30	51	88	
		%	8.0	34.1	58.0	100	

Note: * – Chi-Square test; UAP – Unstable angina pectoris; N-STEMI – non-ST elevation myocardial infarction; STEMI – ST elevation myocardial infarction; Q1 – Quartile 1; Q2 – Quartile 2; Q3 – Quartile 3; Q4 – Quartile 4.

Table 3: Association of adiponectin concentration category and ACS types in DM.

No.	Adiponectin Quartile	n (%)	ACS types			Total	p
			UAP	N-STEMI	STEMI		
1.	Q1-Q2 (low)	n	4	16	23	43	0.697*
		%	9.3	37.2	53.5	100	
2.	Q3-Q4 (high)	n	3	14	28	45	
		%	6.7	31.1	62.2	100	
Total		n	7	30	51	88	
		%	8.0	34.1	58.0	100	

Note: * – Chi-Square test; UAP – Unstable angina pectoris; N-STEMI – non-ST elevation myocardial infarction; STEMI – ST elevation myocardial infarction; Q1 – Quartile 1; Q2 – Quartile 2; Q3 – Quartile 3; Q4 – Quartile 4.

The proportion of STEMI ACS was higher in the high adiponectin concentration category (quartiles Q3–Q4), at 62.2%. The proportion of N-STEMI ACS was higher in the low adiponectin concentration category in quartile (Q1–Q2) at (37.2%). Statistical analysis revealed no significant differences ($p > 0.05$). Therefore, there was no significant association between adiponectin concentration category and the types of ACS in DM subjects.

Table 4 shows the association of adiponectin concentration category and mortality from ACS in DM subjects. There is a significant association between adiponectin concentration and mortality ($p < 0.05$), where the proportion of subjects who died was found to be higher in the low adiponectin concentration category (quartiles Q1–Q2) at 20.9%. Hypoadiponectinemia (Q1–Q2) had a risk of death 11.6 times greater than subjects with high adiponectin (Q3–Q4) (OR 11.647, 95% CI: 1.407–96.443, $p = 0.006$).

Table 5 shows the association between the adiponectin concentration quartile and the risk factor

ACS in DM subjects. There is a significant association between risk factor dyslipidemia and the adiponectin concentration quartile ($p < 0.05$), with the highest proportion of subjects with dyslipidemia in the second quartile of adiponectin (Q2) at 28.4% compared to other risk factors, including gender, age, obesity, hypertension, and smoking.

Discussion

This study involved 88 DM and ACS subjects. The majority of subjects in this study were 60 male subjects (68.2%) with an age of ≥ 50 years (89.8%), dyslipidemia (89.8%), hypertension (75%), and smoking (54.5%). In several studies, the prevalence rate of dyslipidemia was 75.7% in females and 72.6% in males with T2DM (Diabetes mellitus type 2) subjects; this finding is also similar to other studies in different countries that agreed with the high prevalence [13]. This suggests that

Table 4: Association of adiponectin concentration category and mortality ACS in DM.

No.	Adiponectin Quartile	n (%)	Mortality		Total	p*	OR**	95% CI	
			Yes	No				Lower	Upper
1.	Q1-Q2 (low)	n	9	34	43	0.006	11.647	1.407	96.443
		%	20.9	79.1	100				
2.	Q3-Q4 (high)	n	1	44	45				
		%	2.2	97.8	100				
Total		n	10	78	88				
		%	11.4	88.6	100				

Note: * – Chi-Square test; ** – Odds Ratio test; Q1 – Quartile 1, Q2 – Quartile 2, Q3 – Quartile 3, Q4 – Quartile 4.

Table 5: Association of adiponectin concentration quartile and risk factor ACS in DM.

No.	Variable	Category	Adiponectin Quartile				p
			Q1 n (%)	Q2 n (%)	Q3 n (%)	Q4 n (%)	
1.	Gender	Male	14 (15.9)	19 (21.6)	15 (17)	12 (13.6)	0.242
		Female	4 (4.5)	6 (6.8)	7 (8)	11 (12.5)	
2.	Age (years)	≥50 years	17 (19.3)	23 (26.1)	19 (21.6)	20 (22.7)	0.792
		<50 years	1 (1)	2 (2.3)	3 (3.4)	3 (3.4)	
3.	Obesity (BMI)	Yes	6 (6.8)	9 (10.2)	12 (13.6)	7 (8)	0.349
		No	12 (13.6)	16 (18.2)	10 (11.4)	16 (18.2)	
4.	Hypertension	Yes	13 (14.8)	18 (20.5)	16 (18.2)	19 (21.6)	0.810
		No	5 (5.7)	7 (8)	6 (6.8)	4 (4.5)	
5.	Smoking	Yes	11 (12.5)	15 (17)	10 (11.4)	12 (13.6)	0.706
		No	7 (8)	10 (11.4)	12 (13.6)	11 (12.5)	
6.	Dyslipidemia	Yes	14 (15.9)	25 (28.4)	18 (20.5)	22 (25)	0.045*
		No	4 (4.5)	0 (0)	4 (4.5)	1 (1)	

Note: * – Chi-Square test; ** – Odds Ratio test; Q1 – Quartile 1; Q2 – Quartile 2; Q3 – Quartile 3; Q4 – Quartile 4.

the incidence of dyslipidemia is still high in DM and ACS subjects, as found in this study. Another study, prevalence of hypertension in T2DM was detected in 70.5% and 73% patients were male, with the dominating age 55–64 years, and 52% of patients had a history of smoking was detected in ACS subjects [14, 15].

There is no significant difference in adiponectin concentration between ACS types in DM subjects ($p > 0.05$). STEMI had the highest adiponectin concentration in the fourth quartile (Q4), at 69.6%, and the category in quartiles (Q3–Q4), at 62.2%. The results of this study align with those of Pooja Parashar et al., who conducted research on 72 patients with acute myocardial infarction, comprising 42 subjects with STEMI and 32 with N-STEMI, in India. The study revealed that ACS events, particularly STEMI, exhibited the highest concentrations of adiponectin. However, overall, this study found no significant association between adiponectin concentration and the types of ACS in DM subjects. Another study by Takahashi et al. explains that although adiponectin has a protective effect on atherosclerosis and inflammation, some subjects with STEMI and heart failure have high concentrations of adiponectin. This phenomenon is a result of the body's compensatory response to vascular damage. Sattar et al. reported in their research that high concentrations of adiponectin can be associated with the progression of cardiovascular disease in DM subjects,

making this phenomenon a subject of debate. However, several studies reveal the paradox of adiponectin, where high concentrations of it are observed in severe cases of cardiovascular disease, including STEMI, suggesting that the body is attempting to protect itself from cardiovascular stress [16–18].

One of the risk factors is the role of pharmacological therapy, which can affect adiponectin concentrations. In this study, most subjects with DM, hypertension, and dyslipidemia were taking medications, including anti-diabetic drugs, anti-hypertensive drugs, and statins, which can increase adiponectin concentrations. An article written by Susan A. Phillips states that high concentrations of adiponectin can occur with the use of medications such as statins, angiotensin-converting enzyme (ACE) inhibitors, and thiazolidinediones (TZD) [19].

Adiponectin concentration was significantly associated with mortality from ACS in DM subjects ($p < 0.05$). The percentage of subjects who died was higher in the low adiponectin concentration groups in quartiles 1 and 2 (Q1–Q2), at 20.9%, with a risk estimate 11.6 times higher than in the high adiponectin concentration groups (RR 11.6; 95% CI, 1.407–96.443; $P = 0.006$). The results of this study are consistent with Quan Li et al., who found that low concentrations of adiponectin are a reliable indicator of MACE in CAD patients (RR 1.75; 95% CI, 1.066–2.865; $P = 0.027$). Consequently, the survival

ratio decreases for patients with low adiponectin concentrations, as indicated by the Kaplan-Meier survival analysis curve (log-rank $\chi^2=4.592$, $P=.032$) [18]. A cohort study conducted by Pradnyana at the Harapan Kita National Heart Center found that hypoadiponectinemia (adiponectin levels $<5.34 \mu\text{g/ml}$) was associated with major adverse cardiovascular events (MACE), including mortality, but it was not statistically significant (RR 4.33; 95% CI: 0.86–21.8; $p=0.075$) [20].

According to the different results in several studies, Brian A. Bergmark showed that subjects with the highest baseline adiponectin concentration quartile (Q4) are at a significantly higher risk of mortality and cardiovascular events (8.4% versus 1.7%; $P<.0001$) after adjusting for age, sex, estimated glomerular filtration rate, and hypertension [12].

The Difference in condition shows that high adiponectin concentrations in ACS subjects are a compensatory response that occurs to acute inflammatory lesions and hypoxia-reoxygenation. High concentrations of adiponectin independently predict the risk of all-cause mortality, cardiac death, and myocardial infarction in CAD patients. In ACS subjects, adiponectin is associated with a higher risk of MACE [21].

A significant association was found between risk factors, dyslipidemia and adiponectin concentration ($p<0.05$), with the highest proportion of dyslipidemia subjects found in the second quartile of adiponectin (Q2) at 28.4%. Dyslipidemia is a major risk factor for ACS in DM subjects. Adiponectin plays a role in reducing critical processes that contribute to atherosclerosis, including the adhesion of inflammatory cells in vascular tissue, the proliferation of smooth muscle cells, and the formation of foam cells. The ability to reduce inflammation is derived from activation of AdipoR1/AdipoR2 receptors on specific immune and vascular cells. This process reduces the quantity of inflammatory cells at injury sites, increases the availability of nitric oxide in vascular cells, and mitigates oxidative stress. So their role in fat processing and vascular inflammation suggests that hypoadiponectinemia is involved in the development of ACS in DM subjects [22–25].

Adiponectin has both direct and indirect effects on the protection against cardiovascular disease. Adiponectin inhibits monocyte adhesion by suppressing the expression of adhesion molecules (VCAM-1), inhibiting the transformation of macrophages into foam cells by inhibiting the expression of scavenger receptors, and reducing the proliferation of migrating smooth muscle. This phenomenon occurs as a result of the growth factor's response. In addition, adiponectin tends to lower

TNF- α levels. It also increases the production of nitric oxide (NO) in endothelial cells and stimulates angiogenesis. Hypoadiponectinemia is a risk factor for myocardial infarction [10]. There is no literature explaining the reasons for these different results. The phenomenon observed is that high levels of inflammation decrease adiponectin concentrations; however, there is still no clarity on whether this is a cause-and-effect relationship.

This study has a limitation because it does not separate patients with type 1 DM and type 2 DM; it should exclude patients taking insulin-sensitizing therapy, anti-hypertension drugs, statins, and oral hypoglycemic drugs, as well as those with heart and kidney problems, including heart failure and kidney disease. Additionally, in this study to establish a predictor, the sample used should be the first onset event sample. This study uses the ACS samples, regardless of their prior infarction status, whether they had a previous infarction or not.

Conclusion

There is no significant association between adiponectin concentration and the type of ACS in DM subjects. Dyslipidemia was identified as the predominant risk factor affecting adiponectin concentration. A significant association was found between adiponectin concentration and mortality from acute coronary syndrome in diabetic subjects.

Conflict of interest

The authors declare no conflict of interest.

Ethics approval

The research ethics committee of Hasanuddin University's medical faculty issued an ethical approval recommendation for this study on July 10, 2024, with Number 712/UN4.6.4.31/PP36/2024, and the research authorization at Dr. Wahidin Sudirohusodo Hospital, Makassar, was issued on July 25, 2024, with Number DP.04.03/D.XIX.2/21025/2024.

Consent to participate

Written informed consent was obtained from all the participants.

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