ORIGINAL ARTICLE

Association of fibrinogen-to-albumin ratio in patients with isolated coronary artery ectasia

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Background: Previous research has indicated that chronic inflammation plays a significant role in the development of coronary artery ectasia (CAE). However, there is limited data on the role of plasma fibrinogen-to-albumin ratio (PFAR) in CAE patients. Our study aimed to explore the connection between PFAR and the presence of CAE. **Materials and Methods:** This research used a case–control methodology. We included a total of 108 consecutive patients who had CAE without any stenosis. Among them, there were 65 males and 43 females, with a mean age of 58.2 ± 8.5 years. The control group included 102 consecutive participants with angiographically normal coronary arteries, consisting of 62 males and 40 women, with a mean age of 57.3 ± 8.6 years. Statistical analyses were conducted using Student's-*t*-test, Mann–Whitney *U*-test, Chi-square test, linear regression, logistic regression, and receiver operating characteristic (ROC) curve analysis. **Results:** PFAR in the CAE group was significantly higher compared to the controls (84.8 ± 7.4 vs. 70.1 ± 9.5, $P < 0.001$). Using multiple logistic regression showed a strong link between PFAR and CAE, with an odds ratio for PFAR of 1.818 (95% confidence interval [CI] 1.092–6.201; $P = 0.005$). PFAR was exceeded 72.6, the sensitivity and specificity were 80.2% and 72.6%, respectively. The area under the ROC curve (area under the curve) was 0.731 (95% CI: 0.659–0.803, $P = 0.028$). **Conclusion:** In our study, we found that PFAR levels were notably higher in the CAE group compared to the control group, and we observed a significant correlation between PFAR and CAE.

Key words: Albumin, fibrinogen, inflammation

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INTRODUCTION

Coronary Artery Ectasia (CAE) is when the coronary artery lumen gets bigger, about 1.5–2 times the diameter of the normal artery next to it. Coronary aneurysms are defined as luminal dilatations above 2.0 times the normal diameter. CAE has risen due to the rapid use of coronary angiography. The etiology of this clinical condition is unidentified. Kawasaki disease, collagen abnormalities, and connective tissue disorders also contribute to CAE. CAE is rare with percutaneous coronary intervention (PCI) and trauma. The majority of CAE symptoms manifest as chest pain.[1-6] Studies suggest inflammation may play a role in CAE.[7]

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Liver albumin, the principal plasma protein, is crucial in systemic and local inflammation.[8] Plasma albumin decreases with inflammation. Plasma albumin functions as an antioxidant, mitigating vascular-free radical damage.[9] CAD is more common, gets worse, and kills more people when plasma albumin levels are low.[10] Plasma fibrinogen is associated with extended inflammation.[11] It has been shown that people with stable CAD or ST-segment elevation myocardial infarction (STEMI) are more likely to have atherosclerosis in the CA.[12,13] Elevated plasma fibrinogen levels in patients with CAD are associated with increased cardiovascular (CV) and overall mortality.

Inflammatory indicators are essential for evaluating the severity and course of CV disease (CVD). We

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have used C-reactive protein (CRP), a well-established indicator of systemic inflammation, to assess CV risk. Still, CRP levels can change because of short-term infections, long-term inflammatory diseases, or metabolic problems, which can make it less specific in some situations. The CRP-to-albumin ratio (CAR) was created by researchers as a more accurate measure that takes into account both the amount of inflammation (CRP) and the body's nutritional status (albumin). Despite the prognostic significance of CAR in CAD and other CVD disorders, research on its role in CAE remains inadequate.[14]

The plasma fibrinogen-to-albumin ratio (PFAR) was recently found to be a biomarker for inflammation linked to several CVDs. For example, PFAR is not the same as CRP and CAR because it has albumin, which helps with inflammation and clotting, and fibrinogen, which is an important part of clotting. Compared to CRP alone, this mix makes PFAR a better indicator of both systemic inflammation and the prothrombotic state. PFAR has demonstrated efficacy in predicting the severity of stable CAD.[13] The admission PFAR is a reliable way to tell if a patient with STEMI will have no-reflow and die soon after PCI.[15] Nonetheless, the relationship between the PFAR and CAE remains unreported. As more people realize that CAE is an inflammatory disorder, we think that PFAR could be a new biomarker that helps us figure out what causes it. The aim of our work was to investigate the relationship between CAE and PFAR, potentially offering new insights into its inflammatory mechanisms.

METHODS

The Ethics Committee of Adiyaman University Hospital approved a case-control study. We included patients who had myocardial ischemia and chest pain confirmed by noninvasive ischaemic testing. We exclusively included patients who received coronary angiography. During angiography, the medical team evaluated each patient's digital data and quantitative coronary measurements. We assessed the coronary artery lumen diameter using a catheter. We characterized patients with normal coronary angiography and ecstatic coronary segments by measuring the proximal, middle, and distal coronary arteries twice each. Isolated CAE patients had extensive or regional CA lumen dilation 1.5–2.0 times the adjacent normal diameter. In normal coronaries, plaques and ectasia were absent.

We included a total of 108 consecutive patients who had CAE without any stenosis. For the control group, patients with normal coronary arteries were selected through random sampling from all eligible individuals within the same study timeframe, ensuring that the selection process was unbiased and reflective of the broader population. This methodology helps mitigate selection bias and improves the comparability between groups. The recruitment of cases and controls occurred simultaneously. We gathered study participants' medical histories from medical records and reported them in patient-specific forms. Diagnosed hypertension (HT) with systolic blood pressure >140 mmHg, diastolic blood pressure >90 mmHg, or antihypertensive medication usage. Diabetes is diagnosed when fasting plasma glucose is ≥126 mg/dL (7.0 mmol/L), A1C is ≥6.5% (48 mmol/mol), 2-h plasma glucose is ≥200 mg/dL (11.1 mmol/L) during a 75-g OGTT, or random plasma glucose is ≥200 mg/dL (11.1 mmol/L) in individuals with classic symptoms of hyperglycemia or hyperglycaemic crisis. The use of antidiabetic medications also indicates a diagnosis of diabetes. Dyslipidaemia is defined based on abnormal lipid profile results, including elevated total cholesterol, Low density lipoprotein-cholesterol, or triglycerides, or low High density lipoprotein-cholesterol, as per CV risk assessment. A history of dyslipidaemia or the use of antilipidemic drugs also confirms the condition. Patients were smokers if they smoked for a year. Using echocardiography software, the modified Simpson's approach computed the left ventricular ejection fraction (LVEF).[4] The estimated glomerular filtration rate (eGFR) was determined with the Cockcroft-Gault formula.[4] The exclusion criteria of our study are: (1) Left ventricular hypertrophy and heart valve disease (2) HT and renal failure (3) Neurovascular disease, hepatic dysfunction, autoimmune illness, neoplastic disease, and osteoporosis. (4) Myocardial infarction and left ventricular dysfunction.

Laboratory measurements

We performed central laboratory tests 1 h after the venipuncture. An autobiochemical analyzer (AU5400; Olympus, Tokyo, Japan) conducted a routine biochemical test. Our automated coagulation analyzer (STA Compact Max; Stago, Paris, France) measured plasma fibrinogen. For PFAR, divide plasma fibrinogen by albumin concentration and multiply by 1000. We clearly articulate the protocols for measuring fibrinogen and albumin. To make sure everything was consistent and reliable, all blood samples were collected using standard venipuncture methods, kept at −80°C, and transported in a way that followed strict rules to protect the integrity of the samples.

Coronary angiography

During coronary angiography, interventional cardiologists used the Judkins method. The femoral approach, along with the cranial and caudal angles in the right and left inclined planes, were measured at 30 frames per second. In this study, interventional cardiologists analyzed coronary angiography images. Falsetti and Carroll's definition of CAE guided our work. The coronary angiography classified

normal segments as stenosis-free. We did not study CAE with coronary stenosis.

Statistical analysis

We conducted the statistical analysis using SPSS v25 (SPSS Inc., Chicago, IL, USA). After testing continuous variables, the Kolmogorov–Smirnov test assessed the normality of data distribution and provided the mean, standard deviation, median, and interquartile range. Given the sample size exceeds 100, the assumption of normality for the *t*-test can be waived. Therefore, the Student's-*t*-test was used for all continuous variables, regardless of distribution, while the Mann–Whitney *U*-test was not required. The Student's-*t*-test was applied to datasets that lacked pairing. Chi-squared compared categorical data. In univariate linear regression analysis, factors with $P < 0.25$ were identified as risk markers and included in the full-variable model. We used logistic regression to find CAE-independent determinants. An receiver operating characteristic (ROC) curve analysis assessed PFAR's accuracy, sensitivity, and specificity in differentiating CAE. We judged the results significant when the $P < 0.05$ on both sides. We performed variance inflation factor diagnostics before logistic regression to search for any potential collinearity between variables. This made sure that the model interpretation was solid.

RESULTS

Out of 228 patients, we eliminated 7 for myocardial infarction and LV dysfunction. 5 for LV hypertrophy and heart valve disease, and 4 for HT and renal failure. Neurovascular disease, hepatic dysfunction, autoimmune illness, neoplastic disease, and osteoporosis disqualified two people. Exclusions allowed 210 patients to register. In this study, 102 people with coronary arteries that were angiographically normal (62 men and 40 women; mean age 57.3 ± 8.6 years) and 108 people with isolated CAE and no coronary artery stenosis were looked at. The mean age of the men and women was 58.2 ± 8.5 years.

Table 1 displays patient data. The prevalence of smoking, HT, DL, and family history were substantially greater in the CAE group compared to the control group ($P < 0.001$, $P < 0.001$, $P = 0.005$, and $P = 0.022$, respectively). Treatment regimens remained consistent across groups. The groups did not vary in age, gender, body mass index, or LVEF ($P < 0.005$).

As shown in Table 2, white blood cell counts, creatinine, eGFR, plasma D-dimer, fasting glucose, lipid panels, and uric acid were similar in CAE and controls ($P > 0.05$). The levels of fibrinogen and PFAR were much higher in the CAE group (3.1 ± 0.5 vs. 2.9 ± 0.7, $P = 0.007$), but the levels of albumin were much lower (38.1 ± 2.4 vs. 40.2 ± 3.8, $P < 0.001$).

Linear regression analysis was conducted to assess the appropriateness of multivariate logistic analysis for PFAR. Using multiple logistic regression showed a strong link between PFAR and CAE, with an odds ratio for PFAR of 1.818 (95% confidence interval [CI] 1.092–6.201; $P = 0.005$) [Table 3].

Table 1: Baseline characteristics and medication of the two groups

	Patients with CAE (*n*=108)	Control group (*n*=102)	*P*
Age (years)	58.2±8.5	57.3±8.6	0.756
Gender, male, *n* (%)	65 (60)	62 (60)	0.758
BMI (kg/m²)	28.2±0.2	27.4±0.7	0.656
LVEF (%)	57.1±1.5	59.1±1.8	0.302
Smoking, *n* (%)	42 (38)	20 (19)	<0.001
Family history of CAD	17	9	0.022
Diabetes mellitus	18	14	0.316
HT	27	16	<0.001
Dyslipidemia	22	10	0.005
ACEI/ARB/ARNI	16	12	0.609
Beta-blocker	21	16	0.452
Calcium canal blocker	13	9	0.425
Antiplatelet	20	15	0.112
Statin	12	7	0.102

BMI=Body mass index; CAD=Coronary artery disease; ACEI=Angiotensin-converting enzyme inhibitor; ARB=Angiotensin II receptor blocker; ARNI=Angiotensin receptor enkephalinase inhibitör; LVEF=Left ventricular ejection fraction; CAE=Coronary artery ectasia; HT=Hypertension

Table 2: Laboratory parameters of the two groups

	Patients with CAE (*n*=108)	Control group (*n*=102)	*P*
White blood cell (10^3/mL)	8.2±1.9	8.4±1.2	0.658
Platelet count (10^3/mL)	230.3±75.4	234.8±74.2	0.754
Fasting plasma glucose (mmol/L)	26.1±40.8	122.1±41.2	0.346
Creatinine (mg/dL)	0.80 (0.75–0.99)	0.81 (0.75–0.97)	0.782
EGFR (mL/min)	91.3 (68.9–105.6)	92.8 (74.9–107.9)	0.696
Total cholesterol (mg/dL)	164.0±32.0	158.7±39.4	0.432
HDL (mg/dL)	33.6±6.0	35.8±7.0	0.412
LDL (mg/dL)	124.2±30.6	107.4±29.6	0.112
Triglyceride (mg/dL)	128.2±28.6	122.4±23.6	0.188
Uric acid (umol/L)	403.0±47.8	378.3±44.1	0.212
Albumin (g/L)	34.1±2.4	42.2±3.8	<0.001
Total protein (g/L)	622±2.4	65±2.1	0.313
Fibrinogen (g/L)	3.4±0.3	2.7±0.4	<0.001
D-Dimer (mg/L)	0.4±0.2	0.3±0.2	0.819
PFAR	84.8±7.4	70.1±9.5	<0.001

EGFR=Estimated glomerular filtration rate; PFAR=Plasma fibrinogen to albumin ratio; HDL=High density lipoprotein; LDL=Low density lipoprotein; CAE=Coronary artery ectasia

Table 3: Factors associated with CAE

	Univariate analysis			Multivariate analysis		
	Coefficients	95% CI	*P*	OR	95% CI	*P*
FAR/10	1.962	1.083–5.624	0.010	1.818	1.092–6.201	0.005

Variable with *P*<0.25 in univariate regression were included into multivariate regression. CI=Confidence interval; OR=Odds ratio; FAR=Fibrinogen to albumin ratio

The ROC curve showed that when PFAR exceeded 72.6, the sensitivity and specificity were 80.2% and 72.6%, respectively. The area under the ROC curve (area under the curve [AUC]) was 0.731 (95% CI: 0.659–0.803, P = 0.028). Similarly, when the fibrinogen level exceeded 2.99, the sensitivity and specificity were 78.3% and 71.2%, respectively. The AUC was 0.692 (95% CI: 0.615–0.770, P = 0.001). Finally, when the albumin level was <38.6, the sensitivity and specificity were 77.2% and 70.9%, respectively. The AUC was 0.691 (95% CI: 0.609–0.774, P = 0.004) [Figure 1].

DISCUSSION

This research indicates that the PFAR was notably greater in the CAE group compared to the control group. As far as we know, we are the first to demonstrate a tight association between the PFAR and CAE.

Reports suggest that CAE stems from a widespread abnormality in the vascular wall that affects multiple segments and is characterized primarily by saccular ectasia rather than fusiform ectasia. Limited research has been conducted on the prognosis of CAE patients.[16] Three decades ago, a significant study revealed a 5-year mortality rate of 26% among individuals with aneurysmal CAE.[17] In a study by Kajinami *et al*.,[18] plasma cells, macrophages, and lymphocytes were found in large numbers in the intimal and medial layers of the coronary arteries of a person who died of an acute myocardial infarction in the 20th century. This person had CAE and familial hypercholesterolemia.

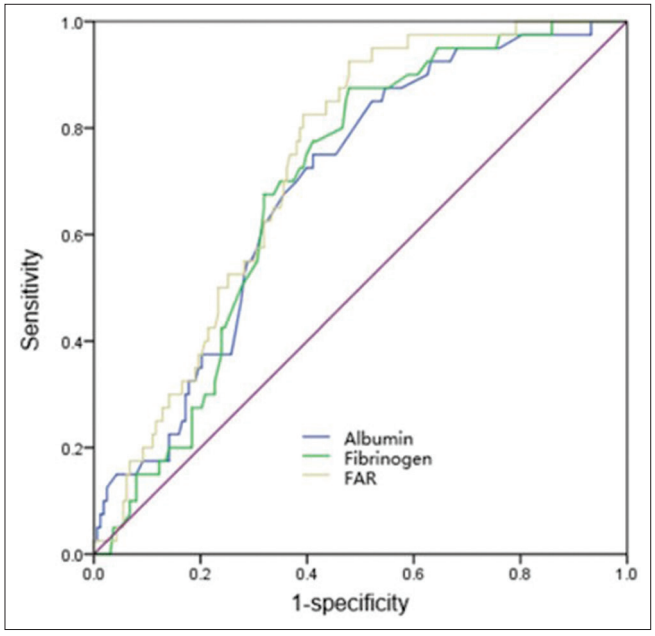
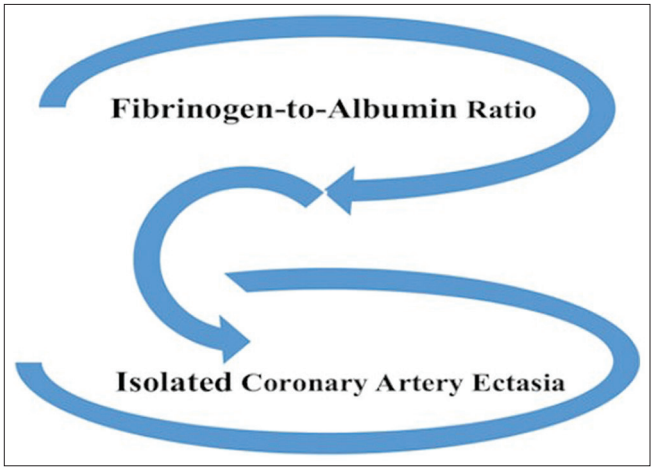


Figure 1: Receiver operating characteristic curve showing the predicting value of risk factors for the presence of coronary artery ectasia

Nitric oxide (NO) is a potential contributor to the onset of CAE. It induces coronary dilation by excessively stimulating the endothelium. Many people receive long-term glyceryl trinitrate for angina, which may exacerbate ectasia by activating NO. Such patients may also experience CAD, a condition where atherosclerosis triggers the inappropriate release of endothelial NO.[19] The exact cause of CAE remains unclear. While a direct link between atherosclerosis and CAE has not been established definitively, CAE is often viewed as a type of CAD, with atherosclerosis being its primary driver.[20-22] Inflammation plays a crucial role in the progression of atherosclerosis.[21-23] This condition can cause aneurysms to form in the tunica media during an inflammatory process, which in turn causes the arterial wall to break down.[24]

Cells that are deprived of oxygen (ischaemic) or are dying (necrotic) trigger an inflammatory reaction throughout the body by releasing substances that promote inflammation in both tissue and blood. The outlook of the condition may vary depending on how quickly this inflammation progresses.[25]

Inflammation plays a significant role in the development of CAE. Markers like CRP and the systemic immune-inflammatory index are linked to CAE and can predict its occurrence.[26] We examined easily accessible inflammatory markers to understand their connection to CAE. The liver primarily produces fibrinogen, which plays a crucial role in inflammation, platelet activation, and the progression of atherosclerotic plaque. When there is inflammation, high levels of fibrinogen can make platelets stick together more and change the viscosity of the blood, which could damage the endothelium and make it harder for blood vessels to work. Studies have shown a positive relationship between plasma fibrinogen levels and coronary atherosclerosis.[27-29]



Graphical Abstract

A study by Lupi *et al*. found that people with an ST-elevation myocardial infarction who had primary PCI were more likely to develop in-stent restenosis if they had higher levels of plasma fibrinogen.[30] In our study, plasma fibrinogen levels were much higher in the CAE group than in the control group. High fibrinogen levels were able to predict CAE on their own. We think that fibrinogen affects CAE by causing inflammatory reactions in the heart or throughout the body, which speeds up atherosclerosis and makes endothelial cells stop working properly.

The amount of serum albumin has an inverse correlation with both local and systemic inflammation. High levels of plasma albumin may also make it harder for platelets to activate and group together, whereas low levels can make the blood thicker and make the endothelium less effective. Albumin, functioning as an antioxidant, may mitigate vascular endothelial damage by capturing free radicals.[31] A study by Kayapinar *et al*.[32] found that people with slow coronary flow had lower levels of albumin in their plasma than healthy people. Our study also found lower levels of albumin in CAE patients. In addition, higher plasma albumin levels independently predicted CAE, suggesting that lower albumin levels may play a significant role in CAE pathogenesis by promoting inflammation, reducing antioxidant defenses, and enhancing platelet aggregation.

The novel inflammatory biomarker PFAR uses fibrinogen and albumin to detect inflammation, oxidative stress, and coronary atherosclerosis. Recent research has linked plasma PFAR closely to CVD. For example, PFAR has been linked to severe coronary stenosis in people with stable angina, bad outcomes after PCI, in-stent restenosis, and acute kidney injury after PCI. PFAR also predicts coronary no-reflow and short-term prognosis in STEMI patients with primary PCI. In our study, we investigated the relationship between PFAR and CAE, finding a strong connection. PFAR levels increased with higher thrombolysis in myocardial infarction scores and greater vessel involvement in SCFP. High levels of PFAR in the plasma could independently predict SCFP. This might be because of how they affect inflammation, oxidative stress, endothelial dysfunction, and CV risk factors.[33-35]

CAE is a complex condition with numerous unidentified risk factors. Combining albumin and fibrinogen levels lets us measure inflammation and oxidative stress. Although not significant, PFAR improved CAE prediction over albumin or fibrinogen levels. Given its widespread clinical use and easy accessibility, PFAR holds promise for CAE assessments. However, our study had limitations. We conducted it at a single center with a small sample size, potentially introducing selection bias. Despite conducting multivariate analyses, residual factors might still impact PFAR's predictive value. In addition, we could not include all inflammatory markers like CRP. Finally, our study focused on a specific patient group; larger, multi-center studies are necessary to confirm our findings.

Inflammatory cytokines, including interleukin-6 and tumor necrosis factor-alpha, are crucial in initiating endothelial damage and facilitating vascular remodeling. These cytokines activate oxidative stress pathways, which leads to an imbalance in the production of NO and higher levels of reactive oxygen species, which make vascular damage worse. In addition, fibrinogen plays a role in endothelial dysfunction by making it easier for platelets to stick together and increasing blood viscosity, which makes vascular inflammation worse. Albumin mitigates oxidative stress due to its antioxidant characteristics; nonetheless, its reduction during systemic inflammation may jeopardize vascular protection. By combining these two pathways, PFAR acts as a marker that shows both the amount of inflammation and the activity of the coagulation system in CAE. Focussing on these pathways makes our results more biologically sound and shows how useful PFAR could be as a tool for assessing risk in clinical practice.[36,37]

Potential clinical implications

If PFAR proves to be an independent predictor of CAE, it could significantly impact clinical outcomes. Clinicians may use PFAR as an inexpensive and accessible inflammatory marker to identify patients at elevated risk for CAE. Setting PFAR levels for "high-risk" patients may facilitate early detection, stratification, and treatment optimization. Patients with high PFAR levels may need more intensive CV risk management, which may include changes to their lifestyle, anti-inflammatory medications, and more frequent imaging exams to see how the disease is progressing. Future research should concentrate on pinpointing the precise PFAR levels associated with favorable clinical results. This will make it easier to use this information in everyday clinical practice.

CONCLUSION

Our study revealed elevated PFAR levels in CAE patients compared to those with normal coronary arteries. This suggests that high PFAR levels might be associated with CAE. Our investigation revealed a substantial association between elevated PFAR levels and CAE.

Data availability

The dataset examined in this study is available on reasonable request from the corresponding author.

Ethics committee approval

All methods were carried out in accordance with the Declaration of Helsinki. The Ethics Review Committee

of Adiyaman University approved the study and written informed consents were obtained (2021/03–15). The informed consent to participate was obtained from all of the participants in this study.

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Nil.

Conflicts of interest
There are no conflicts of interest.

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