Angiology
1–9

DOI: 10.1177/00033197251398366
journals.sagepub.com/home/ang
Sage

Comparison of Plasma Postprandial Versus Fasting Atherogenic Index in Predicting Contrast-Induced Nephropathy After Acute Coronary Syndrome

Muhammet Salih Ateş, MD[1], Erdoğan Sökmen, MD[1], Fikret Keleş, MD[1], Alp Yıldırım , MD[1], Mustafa Çelik, MD[2], and Muhammed Fatih Kaleli, MD[2]

Abstract

Contrast-induced nephropathy (CIN) is a common complication in patients undergoing percutaneous coronary intervention (PCI) for acute coronary syndrome (ACS). The atherogenic index of plasma (AIP; log [triglyceride/high-density lipoprotein cholesterol (HDL-C)]), is linked to cardiovascular risk, but the value of postprandial AIP (PAIP) versus fasting AIP (FAIP) for predicting CIN is unclear. The present study compared the predictive ability of PAIP and FAIP for CIN in ACS patients undergoing PCI. ACS patients (n = 882) were analyzed, with 512 in the FAIP group and 370 in the PAIP group, based on the timing of lipid panel collection. CIN occurred in 15.6% of the FAIP group and 16.2% of the PAIP group ($P = .813$). AIP was significantly higher in the PAIP group (0.643 ± 0.264) compared with the FAIP group (0.493 ± 0.262, $P < .001$). AIP was an independent predictor of CIN in both groups, with stronger predictive power in the PAIP group (OR 46.57, $P < .001$) versus the FAIP group (OR 6.33, $P = .003$) Receiver operating characteristic (ROC) analysis showed a higher area under the curve (AUC) for PAIP (0.712) than FAIP (0.670, $P < .001$). PAIP is a superior predictor of CIN compared with FAIP, emphasizing the importance of the postprandial lipid profile in ACS patients.

Keywords

contrast-induced nephropathy, atherogenic index of plasma, postprandial atherogenic index, fasting atherogenic index, acute coronary syndrome

Introduction

Contrast-induced nephropathy (CIN) is a renal complication of contrast agent after intravenous administration.[1] It is defined as at least 0.5 mg/dl or 25% increase in baseline serum creatinine levels within 3 days following contrast medium administration.[2] Despite an incidence as low as 2% to 5% in the general population, CIN may be observed in up to 55% of high-risk patients, such as those with diabetes mellitus, chronic renal failure, hearth failure, and older age, after coronary angiography and percutaneous coronary interventions (PCI) in patients with acute coronary syndrome (ACS).[3] The exact underlying etiology of CIN is still yet to be totally understood. However, a few mechanisms have been proposed such as renal vasoconstriction injury, inflammation and oxidative stress.[4-6] Beyond renal impairment, CIN is also associated with adverse cardiovascular outcomes, including higher rates of major adverse cardiac events (MACE), acute heart failure, arrhythmias, reinfarction, and both in-hospital and long-term mortality.[7-10] In severe cases, CIN can necessitate renal replacement therapy and markedly worsen short- and long-term prognosis.

The atherogenic index of plasma (AIP) is a novel index and computed by Log [triglyceride (TG)/high-density lipoprotein cholesterol (HDL-C)]. It is suggested to be a sensitive marker of lipoprotein sizes and esterification rates, hence a good marker of plasma atherogenicity.[11] Previous studies reported that AIP had an independent association with coronary artery disease (CAD). AIP also has prognostic significance in patients with such as CAD, ACS, ischemic stroke and arterial stiffness.[12-21] Recently, fasting PAI was also reported be to associated with the occurrence of CIN.[22]

Many investigations only assessed the fasting state lipid profile in their study cohorts and did not take into consideration the fact that TG levels increase after food ingestion.[23]

[1]Department of Cardiology, Ahi Evran University Faculty of Medicine, Kırsehir, Turkey
[2]Department of Cardiology, Necmettin Erbakan University, Konya, Turkey

Corresponding Author:
Muhammet Salih Ateş, Department of Cardiology, Ahi Evran University Faculty of Medicine, Kervansaray Mahallesi, 2019. Sokak. No:1, Merkez, Kırsehir 40200, Turkey.
Email: m.salih.ates@gmail.com

On the other hand, the postprandial plasma TG concentration following a meal has been of paramount importance in determining the actual lipid profile.[24] It has been shown that patients with CAD exhibit an exaggerated postprandial TG response after fat consumption, and that postprandial TG concentration is an independent predictor of CAD.[15-20,25] Postprandial triglyceridemia was also reported to be linked with the degree of carotid atherosclerosis.[26]

We hypothesized that changes in the postprandial lipid profile (especially higher TG increases) may exert a more pronounced effect in the calculation of postprandial AIP (PAIP), thereby reflecting more accurate prediction ability when compared with the fasting AIP (FAIP). Notably, the existing literature predominantly focuses on fasting AIP values, leaving a significant gap in understanding the potential utility of postprandial lipid profiles. Therefore, our aim in this study was to address this gap by investigating the diagnostic significance of PAIP in the development of CIN in a comparable manner with FAIP in ACS patients undergoing PCI.

Methods

Enrollment of the Patients

Prospective data of the patients admitted to our clinic with an ACS between November 2023 and September 2024 were analyzed retrospectively. Inclusion criteria were: patients >18 years of age and serum creatinine level performed daily within at least a 3-day period. Exclusion criteria were: missing study data, acute kidney failure, end-stage kidney disease, being on lipid lowering therapy, familial hyperlipidemia, cardiogenic shock, contrast-agent exposure within 1 week from the index intervention, patients rejecting coronary angiography, patients with a decision for emergency coronary bypass surgery, malignancy, chronic inflammatory diseases, being on steroid therapy or oral contraceptive use, pregnancy or lactation, and fasting blood samples to measure fasting lipid panel not obtained within 24 hours of hospitalization. A total of 1102 patients were evaluated, and after exclusion of 220 patients on the basis of our exclusion criteria, 370 were enrolled into the postprandial group while 512 were included into the fasting group. In our center, lipid profiles are routinely obtained in patients admitted with ACS as part of standard clinical evaluation. For the present retrospective study, patients were classified according to whether their lipid samples had been drawn in the fasting or postprandial state, based on the timing of meal ingestion before sampling. Therefore, no additional blood sampling, intervention, or patient contact occurred beyond routine clinical practice. The study protocol was approved by the local ethics committee, and the requirement for individual informed consent for publication was waived due to the retrospective design and anonymized data.

Data Collection and Postprandial Lipid Panels

Baseline demographic and clinical characteristics of the patients encompassing age, sex, body mass index, heart rate, blood pressure, history of previous diseases and medications used, data regarding angiographic procedures were collected retrospectively from the digital hospital archive and patient files. Postprandial lipemia peaks at approximately the fourth hour after ingestion of a meal.[26-28] Since significant increase and decrease in the TG levels start at 2nd and 5th post-ingestion hours, respectively,[28] and TG levels slowly return to initial serum levels 6 to 8 hours after a meal,[29] we asked all the patients whether or not they consumed a meal within 2 to 5 hours before the index intervention. Blood samples for the measurement of postprandial lipid panel of the patients having a meal within this specific time interval were collected on admission and these patients were enrolled into the postprandial group. On the other hand, 512 patients whose venous blood samples were collected to measure fasting lipid panel between 6:00 and 8:00 am after 8 to 12 hours of fasting within the 24-hours of hospitalization were included into the fasting group. Baseline blood biochemistry including serum creatinine level was obtained upon hospital admission before the index PCI procedure, and post-PCI blood biochemistry was measured every day for at least 3 days to detect the presence of CIN.

Total cholesterol, HDL-C and TG levels were measured enzymatically (Roche Hitachi Cobas c8000 autoanalyzer, Roche Diagnostic Corp., Mannheim, Germany) and the Friedewald formula was utilized to calculate low-density lipoprotein cholesterol (LDL-C) levels from these lipid parameters. However, because the Friedewald formula may be less accurate in non-fasting states, particularly when TG levels are elevated, direct LDL-C measurements were used in patients with TG levels exceeding 400 mg/dl, consistent with routine clinical practice. A Beckman Coulter (Miami, FL, USA) was used for complete blood count measurement.

Definitions and Diagnosis of Contrast-induced Nephropathy (CIN)

CIN was diagnosed if at least 0.5 mg/dl or 25% increase in the baseline serum creatinine levels within 3 days following contrast medium administration during the index PCI.[2]

All the patients underwent transthoracic echocardiographic assessment (Vivid S70; GE Medical System, Horten, Norway) and Simpson's method was utilized in the calculation of the left ventricular ejection fraction (LVEF).[30] Hypertension was defined as using anti-hypertensive medications or repeated office blood pressure measurement of >140/90 mmHg prior to operation or a previous definitive diagnosis. Diabetes mellitus (DM) was diagnosed based on a fasting blood glucose level of ≥126 mg/dl and/or hemoglobin A1c value >6.5% or use of antidiabetic medication.

ACS was stratified into acute non-ST-segment elevation myocardial infarction (NSTEMI), acute ST-segment elevation myocardial infarction (STEMI), and unstable angina pectoris (USAP). NSTEMI was identified based on: an increase and/or decrease in cardiac troponin levels along with at least one of the following: symptoms related to cardiac ischemia;

ST-segment depression, T-wave inversion, or transient ST-segment elevation lasting less than 20 minutes; imaging evidence indicating new myocardial loss or abnormal movement of the ventricular wall; or the angiographic depiction of intracoronary thrombus. STEMI was identified as change in the cardiac troponins together with one or more of the pertinent criteria like ischemia-related symptoms; new or presumably new ST elevation in at least 2 contiguous derivations or new left bundle branch block; surrogates of viable myocardium loss or new regional wall motion anomaly during imaging; and depiction of coronary thrombus during angiography. USAP was defined symptoms related to worsening cardiac ischemia; ST-segment depression, T-wave inversion, or transient ST-segment elevation lasting $<$20 minutes; imaging evidence indicating new myocardial loss or abnormal movement of the ventricular wall; or the angiographic depiction of intracoronary thrombus in the absence of an increase in the cardiac troponin levels.[31]

Coronary Angiography

The patients enrolled in the study were treated according to the recommendations by the STEMI management guideline.[32] As soon as the written informed consent as regards coronary angiography was obtained, an emergency coronary angiography was performed using standard techniques in each patient. After wiring of the culprit artery, administration of glycoprotein IIb/IIIa inhibitor (tirofiban) or implementation of thrombus aspiration in the cases in the catheterization laboratory were dictated by the choice of the operator. Direct stent implantation to the culprit lesion was intended where appropriate, whereas pre-dilatation using a coronary balloon was preferred in the other patients. Standard clinical practice was used during primary PCI of the culprit artery and all the patients were treated with drug-eluting stents.

Iohexol (350 mg of iodine/ml; Omnipaque, Amersham Health, Princeton, New Jersey, USA), a nonionic and low-osmolality contrast agent, was used in all patients. In order to prevent CIN, intravenous hydration with saline solution (0.9%) was initiated during the catheterization procedure and was continued for at least 12 hours with an infusion rate of 1 ml/kg/h and 0.5 ml/kg/h if LV dysfunction was present.

Statistical Analysis

Statistical analysis was conducted using SPSS (Version 29.0 for Windows, IBM Corp., Armonk, NY, USA). Assessment of the distribution of quantitative data was made using the Kolmogorov-Smirnov test. Continuous data were expressed either as mean $\pm$ standard deviation or as median (25th and 75th interquartile range). Categorical variables were expressed as counts (percentages). Pairwise analysis among groups was performed using the Mann-Whitney-*U* test or independent sample *t*-test, as appropriate. Categorical variables were analyzed using the chi-square test. Spearman's rank correlation coefficient was used to evaluate bivariate relationships between continuous parameters.

Univariate and multivariate binary logistic regression analyses were performed to identify factors associated with CIN. All variables were initially included in the univariate analysis, and only those with a $P < .05$ or considered clinically relevant based on prior literature were presented in the table. Subsequently, only statistically significant variables from the univariate analysis were included in the multivariate model.

Receiver operating characteristic (ROC) curve analysis was performed to evaluate the diagnostic performance of FAIP and PAIP in predicting CIN. The optimal cutoff values were determined by the Youden index, and differences between Area Under the Curves (AUC) were assessed using DeLong's test for correlated ROC curves. A 2-sided *P* value of $<.05$ was considered statistically significant.

Results

The FAIP group included 512 patients (mean age: 62 years, 73% male) and PAIP group included 370 patients (mean age: 61 years, 74.1% male). Table 1 represents the demographics and clinical characteristics together with the laboratory findings of the FAIP and PAIP groups. Both groups were similar regarding age, gender distribution, Body mass index (BMI), diabetes mellitus (DM), hypertension (HT), smoking, family history of CAD, admission heart rate, admission systolic and diastolic blood pressure, ratio of STEMI, HDL-C, white blood cell (WBC) count, high-sensitive C-reactive protein (hs-CRP), creatinine, aspartate aminotransferase (AST), admission troponin I and LVEF ($P > .05$ for all). On the other hand, total cholesterol, TG, hemoglobin, platelet counts were greater in the PAIP group, while LDL-C and blood urea nitrogen (BUN) were greater in the FAIP group.

In both groups, the rate of CIN occurrence was similar (80 patients [15.6%] in FAIP group and 60 patients [16.2%] in PAIP group, $P = .813$). AIP was greater in the PAIP groups compared with the FAIP groups (0.643 ± 0.263 vs 0.492 ± 0.262, respectively, $P < .001$).

A further subgroup analysis was carried out based on CIN occurrence in both FAIP and PAIP groups as CIN− and CIN+ subgroups (Table 2). CIN was encountered in 80 (15.6%) patients in the FAIP group. CIN+ subgroup of FAIP group had greater STEMI ratio, TG, and creatinine level compared with the CIN− subgroup of FAIP group, while HDL-C was greater in the CIN− subgroup compared with the CIN+ subgroup. AIP was greater in the CIN+ subgroup compared with the CIN− subgroup of FAIP group (0.615 ± 0.260 vs 0.469 ± 0.256, respectively, $P < .001$).

CIN was encountered in 60 (16.2%) of the patients in PAIP group. CIN− subgroup had greater LDL-C and HDL-C levels and LVEF compared with the CIN+ subgroup of PAIP group, while TG and creatinine levels were greater in the CIN+ subgroup compared with the CIN− subgroup (Table 2). AIP was

Table 1. Baseline Clinical and Biochemical Characteristics of Patients According to Fasting Atherogenic Index of Plasma (FAIP) and Postprandial Atherogenic Index of Plasma (PAIP).

Variable	Fasting atherogenic index of plasma (FAIP; n = 512)	Postprandial atherogenic index of plasma (PAIP; n = 370)	*P*-value
Age, y	62 (55-70)	61 (53-69)	.093
Gender (male), n (%)	374 (73%)	274 (74.1%)	.738
Body mass index (kg/m^2)	27.4 (25.4-29.4)	27.5 (25.4-29.7)	.336
Diabetes mellitus, n (%)	139 (27.1%)	93 (25.1%)	.503
Hypertension, n (%)	215 (42%)	142 (38.4%)	.281
Smoking, n (%)	260 (50.8%)	212 (57.3%)	.056
Positive family history, n (%)	149 (29.1%)	113 (30.5%)	.644
Heart rate, bpm	75 (70-88)	75 (70-89)	.614
Systolic blood pressure, mmHg	110 (105-130)	120 (110-130)	.077
Diastolic blood pressure, mmHg	70 (65-80)	70 (65-80)	.877
ST-elevation myocardial infarction (STEMI), n (%)	236 (46.1%)	173 (46.8%)	.846
Total cholesterol, mg/dl	171 (145-201)	189 (160-220)	**<.001**
LDL-C, mg/dl	179 ± 50	162 ± 54	**<.001**
HDL-C, mg/dl	41 (35-49)	40 (33-47)	.117
Triglycerides, mg/dl	126 (93-175)	181 (136-227)	**<.001**
Hgb, g/dl	13.5 (12.5-14.6)	14 (12.3-15.1)	**.019**
White blood cells, 10^3/µl	8.6 (7.4-10.5)	8.9 (7.3-11)	.120
Platelets, 10^3/µl	240 ± 63	257 ± 62	**.002**
Creatinine, mg/dl	0.9 (0.73-1)	0.84 (0.71-1)	.118
BUN, mg/dl	32 (26-40)	30 (24-37)	**.002**
AST	20 (16-27.7)	19 (15-24)	.071
Hs-CRP	5.8 (2.5-17.2)	6.1 (2.9-14.5)	.743
Hs-troponin	73 (20-356)	56 (21-353)	.837
AIP	0.4927 ± 0.2620	0.6433 ± 0.2637	**<.001**
CIN, n (%)	80 (15.6%)	60 (16.2%)	.813
LVEF, %	50 (40-58)	49.5 (40-46)	.237
Contrast volume, ml	149 ± 22	144 ± 18	.373

Boldfaced values indicate statistically significant differences between the groups ($P < .05$).
Abbreviations: AIP, Atherogenic Index of Plasma; Hgb, Hemoglobin; LDL-C, Low-density Lipoprotein Cholesterol; HDL-C, High-density Lipoprotein Cholesterol; CIN, Contrast-Induced Nephropathy; LVEF, Left Ventricular Ejection Fraction; BUN, Blood urea nitrogen.

greater in the CIN+ subgroup compared with the CIN− subgroup of PAIP group (0.753 ± 0.287 vs 0.642 ± 0.244, respectively, $P < .001$).

Table 3 demonstrates the bivariate correlation analysis according to AIP in all patients. AIP was correlated positively with CIN ($r = .223$, $P < .001$), age ($r = .186$, $P < .001$), hemoglobin ($r = .142$, $P < .001$), WBC count ($r = .121$, $P < .001$), total cholesterol ($r = .228$, $P < .001$), TG ($r = .905$, $P < .001$), and creatinine ($r = .071$, $P = .035$), while correlated negatively with LDL-C ($r = -.324$, $P < .001$) and HDL-C ($r = -.621$, $P < .001$).

Binary logistic regression analysis in the prediction of CIN was presented in Table 4. AIP and LVEF were independently associated with CIN occurrence in all patients (odds ratio [OR] for AIP: 9.166 [3.666-22.919], $P < .001$ and OR for LVEF: 0.976 [0.959-0.995], $P = .011$). In the FAIP group, AIP was independently associated with CIN occurrence (OR: 6.330 [1.851-21.648], $P = .003$). In the PAIP group, AIP, LVEF and LDL-C independently associated with CIN occurrence (OR for AIP: 46.567 [8.502-255.048], $P < .001$; OR for LVEF: 0.968 [0.940-0.997], $P = .028$; and OR for LDL-C: 0.993 [0.985-1.000], $P = .049$). The OR of AIP in PAIP group was greater than that of AIP in the FAIP group.

In ROC curve analysis, the cutoff of FAIP to predict CIN was 0.560 with 63.8% sensitivity and 66.7% specificity (AUC: 0.670, $P < .001$). On the other hand, cutoff of PAIP was 0.687 with 71.7% sensitivity and 62.3% specificity (AUC: 0.712, $P < .001$). AUC of PAIP was greater than the AUC of FAIP ($P = .03$). (Figure 1)

Discussion

The primary results of the present study were: 1- AIP was increased in ACS patients undergoing PCI who developed CIN. 2- Both FAIP and PAIP were independently associated with CIN development. 3- PAIP was superior to FAIP in the prediction of CIN owing to its greater OR and ROC AUC. To our knowledge, our study is the first to propose usage of PAIP in the prediction of a disease condition.

Table 2. Comparison of Clinical and Biochemical Parameters Between CIN-negative and CIN-positive Groups in Patients With Fasting Atherogenic Index of Plasma (FAIP) and Postprandial Atherogenic Index of Plasma (PAIP).

	Fasting atherogenic index of plasma (FAIP; n = 512)			Postprandial atherogenic index of plasma (PAIP; n = 370)		
Variables	CIN negative n = 432 (84.4%)	CIN positive n = 80 (15.6%)	*P**	CIN negative n = 310 (83.7%)	CIN positive n = 60 (16.2%)	*P**
Age, y	62 (55-70)	63 (56-69)	.739	61 (53-69)	62 (51-68.7)	.803
Gender (male), n (%)	314 (72.7%)	60 (75%)	.668	224 (72.3%)	50 (83.3%)	.073
Body mass index (kg/m^2)	27.4 (25.4-29.3)	27.4 (25.4-29.9)	.430	27.5 (25.4-29.7)	27.4 (25.2-29.6)	.693
Diabetes mellitus, n (%)	117 (27.1%)	22 (27.5%)	.939	80 (25.8%)	13 (21.7%)	.499
Hypertension, n (%)	183 (42.4)	32 (40%)	.694	119 (38.4%)	23 (38.3%)	.994
ST-elevation myocardial infarction (STEMI), n (%)	190 (44%)	46 (57.5)	**.026**	148 (47.7%)	25 (41.7%)	.388
Total cholesterol, mg/dl	173 ± 42	174 ± 42	.869	193 ± 46	188 ± 50	.381
LDL-C, mg/dl	180 (149-211)	170.6 (139-202)	.107	164 (123-205)	135 (105-166)	**<.001**
HDL-C, mg/dl	42 (35-50)	39 (32-43)	**.001**	41 (34-49)	34 (30-42)	**<.001**
Triglycerides, mg/dl	124 (91-166)	155.5 (120-221)	**<.001**	177 (131-218)	209 (167-310)	**<.001**
Hgb, g/dl	13.5 (12.5-14.6)	13.5 (12.1-15)	.935	14 (12.3-15)	14.4 (12.2-15.4)	.388
White blood cells, 10^3/μl	8.6 (7.4-10.5)	8.3 (7.2-10.8)	.766	8.9 (7.3-10.9)	9.6 (7.5-11.3)	.467
Platelets, 10^3/μl	240 ± 62	244 ± 68	.600	259 ± 61	249 ± 65	.429
Creatinine, mg/dl	0.86 (0.72-0.99)	1 (0.89-1.12)	**<.001**	0.81 (0.71-0.94)	1.02 (0.83-1.18)	**<.001**
BUN, mg/dl	32 (26-39.6)	32.7 (26.1-40)	.599	29.3 (24-36)	33.2 (24-40.8)	.037
AST U/l	20 (16-27.5)	20 (16-29)	.989	19 (15-24)	19 (15-25)	.634
Hs-CRP mg/l	5.5 (2.6-16.6)	6 (2-23.7)	.789	6.3 (3-14.5)	5 (2.9-14.8)	.562
Hs-troponin ng/l	74 (19-359)	66 (30-321)	.463	45.5 (21-490)	171.5 (38.7-269.2)	.285
AIP	0.4699 ± 0.2561	0.6159 ± 0.2608	**<.001**	0.6425 ± 0.2440	0.7535 ± 0.2870	**<.001**
LVEF, %	50 (40-58)	48 (40-55)	.122	50 (40-58)	45 (37.2-54.2)	**.032**
Contrast volume, ml	153 ± 22	150 ± 18	.673	143 ± 19	148 ± 24	.245

Values are n (%), mean ± standard deviation, or median (25th and 75th percentiles).
Boldfaced values indicate statistically significant differences between the groups ($P < .05$).
Abbreviations: AIP, Atherogenic Index of Plasma; Hgb, Hemoglobin; LDL-C, Low-density Lipoprotein Cholesterol; HDL-C, High-density Lipoprotein Cholesterol; CIN, Contrast-Induced Nephropathy; LVEF, Left Ventricular Ejection Fraction; BUN, Blood urea nitrogen.
*P-values for comparisons between the 'CIN negative' and 'CIN positive' groups.

Table 3. Bivariate Correlation Analysis According to AIP in All Patients.

AIP	Coefficient (*r*)	*P*-value
CIN status	.223	<.001
Age, y	.186	<.001
Hgb, g/dl	.142	<.001
White blood cells, 10^3/μl	.121	<.001
Total cholesterol, mg/dl	.228	<.001
LDL-C, mg/dl	−.324	<.001
HDL-C, mg/dl	−.621	<.001
Triglycerides, mg/dl	.905	<.001
Creatinine, mg/dl	.071	.035
LVEF, %	−.033	.336

Abbreviations: AIP, Atherogenic Index of Plasma; Hgb, Hemoglobin; LDL-C, Low-density Lipoprotein Cholesterol; HDL-C, High-density Lipoprotein Cholesterol; CIN, Contrast-Induced Nephropathy; LVEF, Left Ventricular Ejection Fraction.

Zilversmit first described nearly 30 years ago the significance of postprandial TG levels and proposed that the occurrence of atherosclerosis may be a postprandial phenomenon.[33] Moreover, Stampfer et al[34] reported that plasma TG level around 4 hours after a meal was better in predicting future development of ACS compared with the fasting plasma TG level.

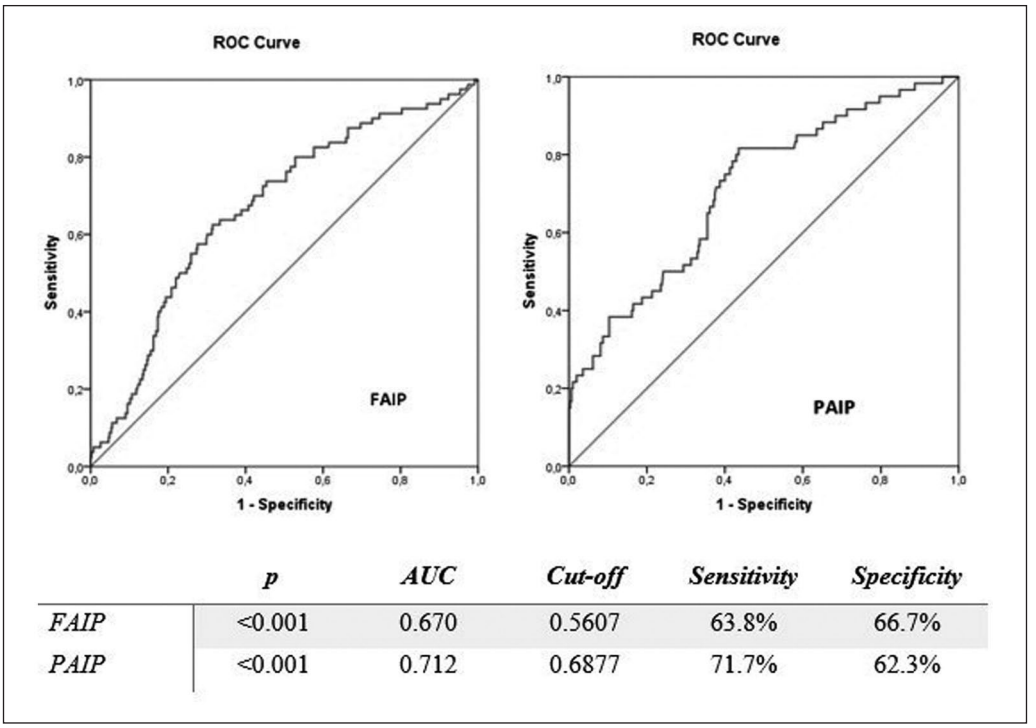
In normal individuals, LDL-C, HDL-C and total cholesterol are reduced up to 3 to 5 hours after the last meal, while TG level increases until 5 to 6 hours after normal food intake.[35] On the other hand, non-HDL-C level does not change in response to normal food consumption.[35,36] However, disturbances in this postprandial lipid panel have also been observed in type 2 diabetic patients, in individuals with obesity or metabolic syndrome, and chronic renal failure.[28]

The maximum changes following normal food digestion are −7.72 mg/dl for total cholesterol, −3.86 mg/dl for HDL-C, −7.72 mg/dl for LDL-C, and +26.57 mg/dl for TG.[37] As one can appreciate, the postprandial changes in the HDL, LDL-C, and total cholesterol levels are trivial, while changes in the TG levels are more pronounced. It is for this reason that current guidelines recommend blood sampling for lipid parameters in the non-fasting state.[38] Moreover, this increase in the

Table 4. Binary Logistic Regression Analysis for Predicting Contrast-induced Nephropathy (CIN) in All Patients, FAIP and PAIP Groups.

	All patients					
	Univariate analysisα			Multivariate analysisβ		
Variable	β	OR (95% CI)	*P*-value	β	OR (95% CI)	*P*-value
AIP	2.557	12.893 (6.195-26.836)	<**.001**	2.216	9.166 (3.666-22.919)	<**.001**
LVEF, %	−.021	.979 (.962-.997)	**.019**	−.024	.976 (.959-.995)	**.011**
Female	.330	1.390 (.900-2.147)	.137			
Age, y	.001	1.001 (.984-1.017)	.932			
Hgb, g/dl	.011	1.011 (.917-1.114)	.831			
Total cholesterol, mg/dl	−.001	.999 (.995-1.003)	.677			
LDL-C, mg/dl	−.006	.994 (.990-.997)	<**.001**	−.002	.998 (.994-1.002)	.375
HDL-C, mg/dl	−.054	.948 (.929-.967)	<**.001**	−.012	.988 (.961-1.016)	.399
	FAIP$^{\mu}$			PAIP$^{¥}$		
AIP	1.845	6.330 (1.851-21.648)	**.003**	3.841	46.567 (8.502-255.048)	<**.001**
LVEF, %	−.019	.981 (.957-1.005)	.117	−.033	.968 (.940-.997)	**.028**
LDL-C, mg/dl	.001	1.001 (.994-1.006)	.943	−.007	.993 (.985-1.000)	**.049**
HDL-C, mg/dl	−.015	.985 (.949-1.023)	.442	.011	1.011 (.966-1.057)	.642

Boldfaced values indicate statistically significant differences between the groups ($P < .05$).
$^{\alpha}$Univariate Analysis for All Patients.
$^{\beta}$Multivariate Analysis for All Patients.
$^{\mu}$Multivariate Analysis for Fasting Atherogenic Index of Plasma (FAIP) Patients.
$^{¥}$Multivariate Analysis for Atherogenic Index of Plasma (PAIP) Patients, the table shows the β coefficient, odds ratio (OR) with 95% confidence intervals (CI), and *P*-values for each variable.
AIP, Atherogenic Index of Plasma; Hgb, Hemoglobin; LDL-C, Low-density Lipoprotein Cholesterol; HDL-C, High-density Lipoprotein Cholesterol; CIN, Contrast-Induced Nephropathy; LVEF, Left Ventricular Ejection Fraction; BUN, Blood urea nitrogen.



	p	*AUC*	*Cut-off*	*Sensitivity*	*Specificity*
FAIP	<0.001	0.670	0.5607	63.8%	66.7%
PAIP	<0.001	0.712	0.6877	71.7%	62.3%

Figure 1. Receiver operating characteristic (ROC) analysis depicting sensitivity and specificity of fasting atherogenic index of plasma (FAIP) and postprandial atherogenic index of plasma (PAIP) in the prediction of contrast-induced nephropathy (CIN).

TG level may be steeper under certain disease conditions like diabetes mellitus, coronary artery disease, obesity, metabolic syndrome, and chronic renal failure.[24]

It was reported that elevated postprandial TG level better be viewed as a risk factor of metabolic syndrome like cholesterol values.[39] Postprandial TG exert direct influences on endothelial functions by the effect of lipoprotein remnants and free fatty acids through stimulation of synthesis and secretion of humoral endothelial markers driving atherogenesis and hypertension.[39] High postprandial TG levels induce the formation of VLDL and chylomicron remnants, as well as free fatty acids. It was shown in the previous studies that lipoprotein remnants and free fatty acids are associated with impaired endothelial nitric oxide (NO) secretion thereby indirectly increasing the counteraction of endothelin-1, a potent vasoconstrictor agent and disrupting the endothelium-dependent vasomotor functioning.[40] Moreover, Young et al[41] demonstrated that increased postprandial lipemia induces endothelial oxidative stress and increases the production of vascular inflammation molecules like interleukin-8 and intercellular adhesion molecule-1 (ICAM-1). In another study, oxidized chylomicrons increased monocyte adhesion to endothelium mediated by increased levels of inflammatory markers such as E-selectin and ICAM-1 and VCAM-1.[42] Free fatty acids that were produced by the enzyme lipoprotein lipase were reported to exert more cytotoxic effect on endothelium after ingestion of polyunsaturated oil compared with the saturated oil.[43] Therefore, it is prudent to link the association of specifically the postprandial PAI with CIN could partially be explained by deterioration in the endothelial vasomotor response, increased endothelial cytotoxicity and vascular inflammation exerted by disturbed postprandial lipidemia in patients with coronary artery disease. Although trivial, HDL-C decreases after meal consumption which may operate as an escalating factor of AIP after consuming a meal. HDL-C has a number of favorable bodily effects such as atheroprotection through reverse cholesterol carriage, antioxidant, and anti-inflammatory actions. Murphy et al[44] showed that apo-A1 and HDL-C particles exerted anti-inflammatory effect on the monocytes by deactivating CD11b. Tolani et al[45] reported that HDL-C was negatively correlated with monocyte number in their animal model, suggesting a more balanced cholesterol influx-efflux mechanism might be more stable monocyte production and hence inflammation. In this regard, HDL-C decrease in our postprandial patients is, to some extent, likely to contribute to their higher PAIP levels in CIN+ subjects.

CIN is linked with adverse clinical prognosis, among which are prolonged hospitalization, renal replacement therapy and greater morbidity and mortality. Therefore, the detection of patients with higher risk of CIN is of paramount importance to let the clinicians take timely and appropriate precautions. Because renal vasoconstriction injury, inflammation and oxidative stress are basic pathogenesis of CIN, understanding these complex pathways could help prevent it. In a recent study by Toprak,[22] FAIP was greater in acute NSTEMI patients with CIN compared to the those without CIN, and FAIP was significantly associated with the occurrence of CIN. The author gave a FAIP cutoff of 0.62 with a sensitivity and specificity of 70% and 58%, respectively. In our study, however, FAIP cutoff was 0.56 and PAIP was 0.68. Our study funding is quite like the study by Toprak. Moreover, we further expanded his study by adding a PAIP group in our study cohort and demonstrated a preliminary finding that PAIP would be more valuable and superior when compared with the FAIP in the prediction of CIN in patients with ACS undergoing primary PCI.

Our results suggest that PAIP is a superior predictor of CIN compared to FAIP, highlighting the importance of evaluating postprandial lipid profiles in clinical practice. This finding may influence routine risk stratification by encouraging clinicians to consider postprandial lipid testing for patients undergoing PCI. Moreover, this study opens avenues for exploring the broader utility of postprandial lipid profiles in cardiovascular risk management, potentially guiding tailored preventive and therapeutic strategies in high-risk populations. Moreover, our study may direct the researchers to investigate in the future the possible genetic basis of the postprandial and fasting lipid profiles within a given subject in a comparable manner in terms of their respective cardiovascular effect.

At first glance, our study cohort is relatively small, and our findings need to be validated by further larger-cohort studies. Moreover, we did not measure both fasting and postprandial lipid panels from the same subject within a 24-hours period. This study aimed to provide a preliminary insight into whether postprandial lipid panels might be superior to fasting lipid panels in the calculation of AIP. Future studies with paired measurements of FAIP and PAIP within the same individuals would enhance statistical robustness and allow direct comparison.

Another limitation is that we did not standardize calorie intake or provide predefined meal compositions within a specific time interval for all patients. Instead, we relied on patient-reported information regarding their meal consumption, which may introduce variability.

To build on these findings, future research should focus on prospective, multicenter studies with larger sample sizes to validate the predictive value of PAIP. Additionally, incorporating standardized postprandial testing protocols and exploring the integration of PAIP into risk assessment tools for CIN would provide more definitive evidence and enhance clinical applicability.

Conclusion

Both FAIP and PAIP are greater in ACS patients with CIN compared with the patients without CIN. FAIP and PAIP are both independently associated with the occurrence of CIN in patients with ACS. However, PAIP may be more valuable and superior when compared with FAIP due to its greater OR and ROC AUC. Future studies are needed to validate our study

results. Future research should include prospective multicenter studies with standardized postprandial lipid protocols and interventional trials to validate PAIP's predictive value and explore its role in improving outcomes for ACS patients at risk of CIN.

Author Note

The study was conducted at Kırşehir Ahi Evran Education and Research Hospital.

ORCID iDs

Muhammet Salih Ateş https://orcid.org/0000-0003-4099-0064
Erdoğan Sökmen https://orcid.org/0000-0002-8170-5912
Mustafa Çelik https://orcid.org/0000-0003-4102-1564
Muhammed Fatih Kaleli https://orcid.org/0000-0001-7649-5503

Ethical Considerations

The study received approval from the local ethics committee (Approval No: 2023/4646, Date: 17.11.2023).

Author Contributions

All authors contributed to: (1) substantial contributions to conception and design, or acquisition of data, or analysis and interpretation of data, (2) drafting the article or revising it critically for important intellectual content, and (3) final approval of the version to be published.

Funding

The authors received no financial support for the research, authorship, and/or publication of this article.

Declaration of Conflicting Interests

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

References

1. Kusirisin P, Chattipakorn SC, Chattipakorn N. Contrast-induced nephropathy and oxidative stress: mechanistic insights for better interventional approaches. J Transl Med. 2020;18:400.
2. Kitajima K, Maeda T, Watanabe S, Sugimura K. Recent issues in contrast-induced nephropathy. Int J Urol. 2011;18:686-690.
3. Rear R, Bell RM, Hausenloy DJ. Contrast-induced nephropathy following angiography and cardiac interventions. Heart. 2016;102:638-648.
4. Jones DA, Beirne AM, Kelham M, et al. Inorganic nitrate benefits contrast-induced nephropathy after coronary angiography for acute coronary syndromes: the NITRATE-CIN trial. Eur Heart J. 2024;45:1647-1658.
5. Heyman SN, Rosen S, Khamaisi M, Idée JM, Rosenberger C. Reactive oxygen species and the pathogenesis of radiocontrast-induced nephropathy. Invest Radiol. 2010;45:188-195.
6. Kwasa EA, Vinayak S, Armstrong R. The role of inflammation in contrast-induced nephropathy. Br J Radiol. 2014;87:20130738.
7. Maioli M, Toso A, Leoncini M, Gallopin M, Musilli N, Bellandi F. Persistent renal damage after contrast-induced acute kidney injury: incidence, evolution, risk factors, and prognosis. Circulation. 2012;125:3099-3107.
8. James MT, Samuel SM, Manning MA, et al. Contrast-induced acute kidney injury and risk of adverse clinical outcomes after coronary angiography: a systematic review and meta-analysis. Circ Cardiovasc Interv. 2013;6:37-43.
9. Sielski J, Kaziród-Wolski K, Piotrowska AM, et al. Contrast-induced nephropathy (CIN) after invasive treatment of acute coronary syndromes—predictors, short and long-term outcome. J Clin Med. 2025;14:3725.
10. Kaliyaperumal Y, Sivadasan S, Aiyalu R. Contrast-induced nephropathy: an overview. Dr. Sulaiman Al Habib Med J. 2023;5:118-127.
11. Qin Z, Zhou K, Li Y, et al. The atherogenic index of plasma plays an important role in predicting the prognosis of type 2 diabetic subjects undergoing percutaneous coronary intervention: results from an observational cohort study in China. Cardiovasc Diabetol. 2020;19:23.
12. Zhang Y, Chen S, Tian X, et al. Association between cumulative atherogenic index of plasma exposure and risk of myocardial infarction in the general population. Cardiovasc Diabetol. 2023;22:210.
13. Rabiee Rad M, Ghasempour Dabaghi G, Darouei B, Amani-Beni R. The association of atherogenic index of plasma with cardiovascular outcomes in patients with coronary artery disease: a systematic review and meta-analysis. Cardiovasc Diabetol. 2024;23:119.
14. Zheng H, Wu K, Wu W, et al. Relationship between the cumulative exposure to atherogenic index of plasma and ischemic stroke: a retrospective cohort study. Cardiovasc Diabetol. 2023;22:313.
15. Wu J, Zhou Q, Wei Z, Wei J, Cui M. Atherogenic index of plasma and coronary artery disease in the adult population: a meta-analysis. Front Cardiovasc Med. 2021;8:817441.
16. Li Y, Feng Y, Li S, et al. The atherogenic index of plasma (AIP) is a predictor for the severity of coronary artery disease. Front Cardiovasc Med. 2023;10:1140215.
17. Ulloque-Badaracco JR, Hernandez-Bustamante EA, Alarcon-Braga EA, et al. Atherogenic index of plasma and coronary artery disease: a systematic review. Open Med. 2022;17:1915-1926.
18. Si Y, Fan W, Han C, Liu J, Sun L. Atherogenic index of plasma, triglyceride-glucose index and monocyte-to-lymphocyte ratio for predicting subclinical coronary artery disease. Am J Med Sci. 2021;362:285-290.
19. Shin HR, Song S, Cho JA, Ly SY. Atherogenic index of plasma and its association with risk factors of coronary artery disease and nutrient intake in Korean adult men: the 2013–2014 KNHANES. Nutrients. 2022;14:1071.
20. Guo Q, Zhou S, Feng X, et al. The sensibility of the new blood lipid indicator—atherogenic index of plasma (AIP) in menopausal women with coronary artery disease. Lipids Health Dis. 2020;19:1-8.
21. Üntan İ, Doğan AE. Monocyte-to-high-density lipoprotein-cholesterol ratio as a predictor and severity indicator of erectile dysfunction. Androl Bul. 2023;25:17-180.
22. Toprak K. Atherogenic index of plasma is an independent risk factor for contrast induced nephropathy in patients with non-st elevation myocardial infarction. Angiology. 2023;74:427-434.
23. Keirns BH, Sciarrillo CM, Koemel NA, Emerson SR. Fasting, non-fasting and postprandial triglycerides for screening cardiometabolic risk. J Nutr Sci. 2021;10:e75.

24. Yanai H, Adachi H, Hakoshima M, Katsuyama H. Postprandial hyperlipidemia: its pathophysiology, diagnosis, atherogenesis, and treatments. Int J Mol Sci. 2023;24:13942.
25. Bansal S, Buring JE, Rifai N, Mora S, Sacks FM, Ridker PM. Fasting compared with nonfasting triglycerides and risk of cardiovascular events in women. JAMA. 2007;298:309-316.
26. Ryu JE, Howard G, Craven TE, Bond MG, Hagaman AP, Crouse JR 3rd. Postprandial triglyceridemia and carotid atherosclerosis in middle-aged subjects. Stroke. 1992;23:823-828.
27. Simons LA, Dwyer T, Simons J, et al. Chylomicrons and chylomicron remnants in coronary artery disease: a case-control study. Atherosclerosis. 1987;65:181-189.
28. Nogaroto V, Rodrigues MR, Vicari MR, et al. High postprandial triglycerides serum levels: is obesity a good predictor? An Acad Bras Cienc. 2015;87:437-445.
29. Nakamura K, Miyoshi T, Yunoki K, Ito H. Postprandial hyperlipidemia as a potential residual risk factor. J Cardiol. 2016;67:335-339.
30. Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. Eur Heart J Cardiovasc Imaging. 2015;16:233-271.
31. Thygesen K, Alpert JS, Jaffe AS, et al. Fourth Universal Definition of Myocardial Infarction (2018). J Am Coll Cardiol. 2018;72:2231-2264.
32. Ibanez B, James S, Agewall S, et al. 2017 ESC guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: the Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). Eur Heart J. 2018;39:119-177.
33. Zilversmit DB. Atherogenesis: a postprandial phenomenon. Circulation. 1979;60:473-485.
34. Stampfer MJ, Krauss RM, Ma J, et al. A prospective study of triglyceride level, low-density lipoprotein particle diameter, and risk of myocardial infarction. JAMA. 1996;276:882-888.
35. Tan Y, Lin Q, Xu J, et al. Non-fasting changes in blood lipids after three daily meals within a day in Chinese inpatients with cardiovascular diseases. Front Cardiovasc Med. 2022;9:799300.
36. Johansen MØ, Moreno-Vedia J, Balling M, Davey Smith G, Nordestgaard BG. Triglyceride content increases while cholesterol content decreases in HDL and LDL+ IDL fractions following normal meals: the Copenhagen General Population Study of 25,656 individuals. Atherosclerosis. 2023;383:117316.
37. Langsted A, Freiberg JJ, Nordestgaard BG. Fasting and nonfasting lipid levels: influence of normal food intake on lipids, lipoproteins, apolipoproteins, and cardiovascular risk prediction. Circulation. 2008;118:2047-2056.
38. Marx N, Federici M, Schütt K, et al. 2023 ESC guidelines for the management of cardiovascular disease in patients with diabetes: developed by the task force on the management of cardiovascular disease in patients with diabetes of the European Society of Cardiology (ESC). Eur Heart J. 2023;44:4043-4140.
39. Jagla A, Schrezenmeir J. Postprandial triglycerides and endothelial function. Exp Clin Endocrinol Diabetes. 2001;109:S533-S547.
40. Vogel RA, Corretti MC, Plotnick GD. Effect of a single high-fat meal on endothelial function in healthy subjects. Am J Cardiol. 1997;79:350-354.
41. Young VM, Toborek M, Yang F, McClain CJ, Hennig B. Effect of linoleic acid on endothelial cell inflammatory mediators. Metabolism. 1998;47:566-572.
42. Kurtel H, Liao L, Grisham MB, et al. Mechanisms of oxidized chylomicron-induced leukocyte-endothelial cell adhesion. Am J Physiol. 1995;268:H2175-H2182.
43. Chung BH, Hennig B, Cho BH, Darnell BE. Effect of the fat composition of a single meal on the composition and cytotoxic potencies of lipolytically-releasable free fatty acids in postprandial plasma. Atherosclerosis. 1998;141:321-332.
44. Murphy AJ, Woollard KJ, Hoang A, et al. High-density lipoprotein reduces the human monocyte inflammatory response. Arterioscler Thromb Vasc Biol. 2008;28:2071-2077.
45. Tolani S, Pagler TA, Murphy AJ, et al. Hypercholesterolemia and reduced HDL-C promote hematopoietic stem cell proliferation and monocytosis: studies in mice and FH children. Atherosclerosis. 2013;229:79-85.