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Original Article

Fibrinogen and Antithrombin-III as Combined Predictors for Contrast-Induced Nephropathy in Coronary Artery Disease Patients during Percutaneous Coronary Intervention

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Abstract:

Background: Coronary artery disease (CAD) patients undergoing percutaneous coronary intervention (PCI) face the risk of contrast-induced nephropathy (CIN).

Aim: To explore the combined predictive value of fibrinogen (FIB) and antithrombin-III (AT-III) in identifying CIN risk in CAD patients undergoing PCI.

Methods: A total of 400 patients with ischemic heart diseases (IHD) undergoing PCI were enrolled in the study. Baseline assessments included demographic information, clinical parameters, and laboratory investigations, covering triglycerides, total cholesterol, HDL-C, LDL-C, albumin, glucose, FIB, neutrophil-to-lymphocyte ratio, AT-III, cystatin-C, serum uric acid, platelet-to-lymphocyte ratio, eGFR, and pre-procedural serum creatinine levels to establish baseline kidney function.

Results: Out of the 400 patients who underwent PCI, 32 (8%) developed acute kidney injury (AKI). Multivariate analysis identified several factors as independent predictors for AKI in PCI patients, including higher BMI, lower triglyceride levels, lower albumin levels, elevated glucose levels, and increased fibrinogen levels. Fibrinogen exhibited a high predictive value for AKI (AUC: 0.829) with an optimal cut-off value of 2.91 g/l, sensitivity of 93.7%, and specificity of 56.2%. Antithrombin III had an AUC of 0.661, with an optimal cut-off value of 85.6%, sensitivity of 62.5%, and specificity of 74.7%. Combining FIB and AT-III improved predictive accuracy, yielding an optimal cut-off value of 0.0923, with a diagnostic sensitivity of 75% and specificity of 79.3%.

Conclusion: The combined assessment of FIB and AT-III significantly enhances the predictive accuracy of CIN. Elevated FIB levels and decreased AT-III levels may indicate an increased risk of developing CIN.

Keywords: Coronary artery disease, Percutaneous coronary intervention, contrast-induced nephropathy, fibrinogen, antithrombin-III & predictive biomarkers.

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Introduction

Coronary artery disease (CAD) remains a leading cause of morbidity and mortality globally, prompting the widespread use of percutaneous coronary intervention (PCI) as a therapeutic modality. While PCI has revolutionized the management of CAD, it introduces the potential risk of contrast-induced nephropathy (CIN). PCI frequently involves the use of iodinated contrast media for imaging purposes, and while it is generally considered safe, the potential for renal complications, particularly CIN, is a recognized risk. [1] CIN is characterized by a sudden deterioration in renal function following exposure to contrast agents, typically within 48 to 72 hours after the procedure. Several factors contribute to the development of CIN during PCI. [2]

The prediction of CIN involves a multifaceted assessment of various risk factors and biomarkers to identify individuals at heightened risk following exposure to contrast media during PCI. Established clinical and demographic factors such as age, pre-existing renal conditions, and comorbidities play a crucial role, while considerations like the volume and type of contrast, inflammatory markers, hemodynamic parameters, medication usage, genetic factors, procedural characteristics, and hydration status are also integral. Additionally, novel risks scores and biomarkers, including kidney injury molecule-1 and neutrophil gelatinse-associated lipocalin, contribute to a more comprehensive risk assessment [3]

Fibrinogen, a pivotal factor in blood coagulation, assumes a dual role as both an essential clotting component and a marker of systemic inflamemation. The administration of contrast media, as seen in PCI, triggers an inflammatory response, leading to elevated fibrinogen levels. This systemic inflammation, reflected in heightened fibrinogen, may contribute to the pathogenesis of CIN, emphasizing the intricate connection between coagulation and renal injury. [4] Moreover, contrast exposure can compromise endothelial function, causing vasoconstriction and reduce renal perfusion. Elevated fibrinogen levels, indicative of impaired endothelial function, may exacerbate these vascular changes. Additionally, fibrinogen 's pro-thrombotic properties raise the possibility of microvascular thrombosis within the renal vasculature, a factor considered in the etiology of CIN.[5] Despite these mechanistic associations, the clinical evidence on the predictive value of fibrinogen for CIN risk remains nuanced.

The liver produces antithrombin-III, a serine protease inhibitor in the coagulation cascade that is encoded by the gene Serpin C1. Heparin-like compounds have the ability to significantly speed up the anticoagulant process, which AT-III influences in conjunction with thrombin factors VIIa, IXa, Xa, and Xia.[6] Furthermore, independent of its anticoagulant function, AT-III possesses potent anti-inflammatory properties.[7,8] Exogenous AT-III administration has been shown to provide protective benefits against harm to the myocardial injury, kidneys, lungs, and liver. [9,10] In contrast, renal ischemia / reperfusion injury in rats is worsened by endogenous AT-III atrophy.[11] After coronary angiography (CAG), patients with low AT-III activity had an increased risk of developing CIN. [12]

Despite the individual associations of fibrinogen and AT-III with CIN, the potential synergistic or antagonistic effects of combining these predictors have received limited attention in the literature. This study aims to delve into the unexplored territory of their combined predictive value in identifying patients at heightened risk of CIN during PCI, shedding light on potential biomarkers that could refine risk assessment and guide personalized preventive strategies.

Materials and Methods:

This prospective study enrolled 400 patients who underwent PCI for ischemic heart disease (IHD) at cardiology Departments, from May 2023 to October 2023.

Ethical Approval: The Ethics Board of approved the study and informed written consent was taken from each participant in the study.

Exclusion criteria:

We excluded patients who received radiocontrast agents within 48 hours before the procedure or 72 hours after the procedure, used non-steroidal nephrotoxic drugs 48 hours before or 72 hours after PCI, malignant tumors, who had chronic kidney disease, infectious or inflammatory diseases, with pulmonary thromboembolism, autoimmune diseases, recent surgery, or a history of trauma wi-

thin the past month, venous thromboembolism, and a history of peripheral vascular disease.

Methods:

Baseline assessments encompassed demographic information, clinical parameters such as blood pressure and heart rate, and a thorough medication history, with a focus on anticoagulants, antiplatelet agents, and nephrotoxic drugs. Laboratory investigations included measuring baseline levels of triglycerides, total cholesterol, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), albumin, glucose, fibrinogen, AT-III, neutrophil-to-lymphocyte ratio (NLR), platelet/lymphocyte ratio, cystatin-C, serum uric acid, eGFR as well as pre-procedural serum creatinine (SCr) levels to establish baseline kidney function.

We measured SCr level using an Olympus AU2700 automatic biochemical analyzer (Olympus Corporation). fibrinogen and AT-III concentrations were determined utilizing the original matching reagent and a Sysmex CA-7000 automatic coagulation analyzer (JEOL, Ltd.).

A modification of diet in renal disease equation was employed to compute the estimated glomerular filtration rate for patients, which is as follows: GFR (ml/min/1.73m2) =186 x Scr (mg/dl) -1.154x age (years)-0.203x (0.742 if female). [13]

Percutaneous Coronary Intervention (PCI) was executed using introducers ranging from 5F to 6F, therapeutic catheters, conventional and pharmacological stents, and radial, or femoral access. During stent implantation, introducers were either pre-or post-intracoronary balloon dilated. Ventriculography was performed after stent implantation. PCI was performed with unfractionated heparin (100 IU/kg), protamine sulphate reversal at the conclusion of the procedure, and 300 mg/ml of low-osmolar iobitridol (henetix®) for non-ionic contrast. The angiographic success of the procedure was assessed using the Thrombolysis in Myocardial Infarction (TIMI) flow classification. [14] which took into account four distinct flow levels: the minimum, Grade 0, if the anterograde flow reaches the occlusion point; and the maximum, Grade3, if anterograde flow completes coronary perfusion in the distal bed as rapidly as in the proximal bed. TIMI score 3 patients were the only ones included in this cohort.

The primary outcome measure for this study would likely be the incidence or development of CIN after PCI. The CIN was identified in accordance with the KDIGO protocol. CIN was defined as any of the follow items: an increase in SCr of at least 0.3 mg/dl (26,5 μmol/l) within a 48-hour period; a rise in SCr to ≥ 1.5 times baseline that has occurred or is presumed to have occurred within the previous 7 days; or a urine volume of < 0.5 ml/kg/h for a duration of 6 hours. [15].

We monitored SCr level at specified intervals post-procedure (e.g. 24, 48, and 72 hours) to detect any changes indicative of CIN. Clinical assessments, including urine output and signs of renal dysfunction, also contributed to the outcome measurement. Additionally, a 3-month follow-up had been conducted to assess the recurrence of CIN and evaluate any potential long-term impacts on renal function.

Statistical analysis:

The data were analyzed employing the IBM Statistical Package for Social Sciences software (SPSS), 25th edition, from IBM, United States. The Kolmogorov-Smirnov Test was employed to evaluate the normal distribution of continuous data. The presentation of results involved expressing qualitative data as numbers and percentages, while quantitative data with parametric distribution were represented by mean, standard deviations, and ranges. Non-parametrically distributed quantitative data were presented as median within interquartile range (IQR). Analytic statistics included the Chi-square test for exploring associations between two qualitative variables, Student t-test for normally distributed quantitative variables when comparing two groups, and Mann-Whitney test for abnormally distributed quantitative variables when comparing two groups. Logistic Regression was employed to measure the relationship between the categorical target variable and one or more independent variables. Additionally, the ROC Curve (receiver operating characteristic) was utilized to assess sensitivity and specificity for fibrinogen, AT-III and combination of fibrinogen with AT-III. P-value below 0.05 is considered significant.

Results of the study:

Baseline characteristics:

The recruited cohort consisted of 400 patients undergone PCI with a mean age of 62.24±13.9 years, where 247 patients (61.8%) were males. A

total of 32 (8%) patients developed acute kidney injury (AKI).

We found that BMI and diastolic blood pressure were significantly higher in AKI group. Distribution of age, gender, hypertension, DM, systolic blood pressure and smoking status did not differ significantly between the two groups. Use of Diuretics and LMWH were significantly more common in AKI group, while no statistically significant differences were identified in the utilization of ACEI/ARBs, β-blockers, statins, CCBs, or nitrates between the AKI and non-AKI groups (p>0.05) (Table 1).

Laboratory Data:

AKI was associated with elevated levels of NLR, glucose, decreased levels of triglycerides, and platelet-to-lymphocyte ratio (PLR). Higher levels of fibrinogen (P=0.001), (Fig.1). and decreased AT-III activity (P < 0.001) were frequently observed in patients who developed AKI(Fig.2). Furthermore, albumin levels were likely to be significantly lower in these patients with AKI (P=0.001). In addition, AKI patients demonstrate a greater significant peak S. creatinine compared to non-AKI patients after PCI (P=0.001). The laboratory analysis results are displayed in (Table 2).

Multiple logistical regression analysis included the following factors: BMI, diastolic blood pressure (mm/Hg), diuretic and low molecular weight heparin, triglyceride, albumin, glucose, fibrinogen, AT-III, NLR and PLR. Multivariate analyses indicated that BMI [odds ratio (OR),1.288;95% confidence intervals (CI),1.078-1.538; P=0.005), triglyceride [OR,0.002;95% CI,0.00-0.035; P<0.001), albumin [OR,0.817;95% CI,0.720-0.927; P=0.002), glucose (OR,1.343;95% CI,1.061-1.699; P=0.014) and fibrinogen (OR,6.159;95% CI,4.959-12.95; P<0.001), were independent predictors for AKI (Table 3).

Value of fibrinogen and AT-III in predicting AKI:

AUC was calculated to be 0.829 in relation to fibrinogen and its ability to predict AKI. The optimal cut-off value was 2.91g/l with a sensitivity of 93.7% and specificity of 56.2% (95% CI,0.612-0.707; P < 0.0001). For AT-III, the optimal cut-off value was 85.6%, and the AUC was 0.661, with a sensitivity of 62.5% and specificity of 74.7% (95% CI,0.612-0.707; P=0.002). fibrinogen and AT-III were incorporated into the AKI risk factor regression analysis. Logit (P)=-13.35+4.536 fibrinogen 0.034 AT-III Was the probability that the combination of fibrinogen and AT-III would predict the developpment of AKI. The AUC achieved by combining fibrinogen and AT-III was 0.845. The optimal cut-off value was 0.0923, with a diagnostic sensitivity of 75% and specificity of 79.3% (95% CI,0.806-0.879; P < 0.001). The results of ROC analysis are presented in (Table 4) and (Fig.3).

Table (1): Baseline characteristics of the AKI and non-AKI groups.

		Non-AKI group (n=368)	AKI group (n=32)	p-value
Age (years)	**Mean ±SD**	62.45±13.9	59.78±13.86	0.302*
	Range	32-96	40-86	
Gender, n (%)	**Male**	228 (62%)	19 (59.4%)	0.782‡
	Female	140 (38%)	13 (40.6%)	
Smoking, n (%)		125 (34%)	13 (40.6%)	0.447‡
HTN, n (%)		211 (57.3%)	18 (56.3%)	0.905‡
DM, n (%)		91 (24.7%)	8 (25.0%)	0.973‡
BMI (Kg/m^2)	**Mean ±SD**	27.03±3.7	29.17±3.72	**0.002***
	Range	16-96	21.3-36.8	
SBP (mm/Hg)	**Mean ±SD**	136.37±26.8	140.28±21.47	0.339*
	Range	65-191	90-180	
DBP (mm/Hg)	**Mean ±SD**	86.6±17.61	93.72±15.29	**0.027***
	Range	43-130	60-122	
AMI, n (%)		118 (32.1%)	15 (46.9%)	0.088‡
β-blocker, n (%)		309 (84%)	27 (84.4%)	0.852‡
ACEI/ARB, n (%)		206 (56%)	17 (53.1%)	0.755‡
CCB, n (%)		103 (28%)	9 (28.1%)	0.987‡
Diuretics, n (%)		126 (34.2%)	17 (53.1%)	**0.033‡**
Statins, n (%)		344 (93.5%)	31 (96.9%)	0.446‡
LMWH, n (%)		168 (45.7%)	21 (65.6%)	**0.030‡**
Nitrates, n (%)		235 (63.9%)	21 (65.6%)	0.857‡

M Mann-Whitney U test, ‡ Chi-square test, *Student T test

AMI, acute myocardial infarction; ACEI/ARB, angiotensin converting enzyme inhibitors/angiotensin receptor blocker; CCB, calcium channel blockers; LMWH, low molecular weight heparin.

Table (2): laboratory Data of the AKI and non - AKI groups

	Groups	Mean ±SD/ Median (IQR)		Range			P-value
Triglyceride (mmol/l)	**Non-AKI group**	1.35	(1.10-1.73)	1.01	-	1.94	**<0.001#**
	AKI group	1.13	(1.04-1.35)	0.95	-	1.52	
Total cholesterol level (mmol/l)	**Non-AKI group**	3.81	±1.63	0.89	-	7.92	0.985♦
	AKI group	3.80	±1.72	1.40	-	7.25	
HDL (mmol/l)	**Non-AKI group**	1.13	(1.02-1.25)	1.91	-	9.96	0.426#
	AKI group	1.13	(1.06-1.26)	1.98	-	1.36	
LDL (mmol/l)	**Non-AKI group**	2.69	±1.27	0.0	-	5.66	0.998♦
	AKI group	2.69	±1.05	0.35	-	4.85	
Albumin level (g/dl)	**Non-AKI group**	40.92 (35.2-48.75)		29.55	-	56.3	**0.026#**
	AKI group	38.53 (33.69-43.62)		28.65	-	50.8	
Blood glucose (mmol/l)	**Non-AKI group**	7.11	±2.84	0.13	-	14.87	**0.001♦**
	AKI group	8.88	±3.09	3.80	-	15.3	
Fibrinogen level (g/l)	**Non-AKI group**	2.88	±0.30	2.0	-	3.9	**<0.001♦**
	AKI group	3.29	±0.27	2.85	-	3.79	
Antithrombin-III (%)	**Non-AKI group**	95.85	±15.88	59	-	182	**0.002♦**
	AKI group	86.75	±15.49	61.38	-	114.73	
Neutrophil to lymphocyte ratio	**Non-AKI group**	3.56	±0.89	1.50	-	5.08	**<0.001♦**
	AKI group	4.30	±1.05	2.65	-	5.85	
Platelet to lymphocyte ratio	**Non-AKI group**	139.99	±19.99	105	-	178	**0.002♦**
	AKI group	151.68	±19.21	116.47	-	186.71	
Baseline S. creatinine (µmol/l)	**Non-AKI group**	66.19	(62.0-72.99)	57	-	89	0.808#
	AKI group	66.30	(62.5-71.0)	56.2	-	75.4	
Cystatin C (mg/l)	**Non-AKI group**	0.81	(0.76-0.84)	0.7	-	75	0.094#
	AKI group	0.79	(0.75-0.82)	0.71	-	0.88	
Serum uric acid (µmol/l)	**Non-AKI group**	303.96	±36.96	236	-	364	0.262♦
	AKI group	310.03	±28.18	265	-	355	
eGFR (ml/min)	**Non-AKI group**	119.0	(95.0-145.5)	26	-	174	0.871#
	AKI group	118.0	(82.0-154.0)	35	-	191	
Peak S. creatinine after PCI (µmol/l)	**Non-AKI group**	68.25 (59.20-76.00)		57	-	85.20	**<0.001♦**
	AKI group	89.50 (75.31-105.2)		59.35	-	107	

p>0.05isnon-significant, p < 0.05is significant, #Mann-WhitneyUTest♦Student T T Test

Table (3): Multiple logistic regression analysis of risk factors for AKI

Parameters	B	S.E.	Wald	P-value	Odds ratio (OR)	95%CI	
						Lower limit	Upper limit
BMI	0.253	0.091	7.797	**0.005**	1.288	1.078	1.538
DBP (mm/Hg)	0.044	0.026	2.922	0.087	1.045	0.994	1.100
Diuretics	-0.760	0.818	.864	0.353	0.467	0.094	2.324
LMWH	0.779	0.691	1.272	0.259	2.179	0.563	8.433
Triglyceride	-6.426	1.565	16.860	**<0.001**	0.002	0.000	0.035
Albumin	-0.202	0.064	9.823	**0.002**	0.817	0.720	0.927
Blood glucose	0.295	0.120	6.033	**0.014**	1.343	1.061	1.699
Fibrinogen level	5.051	1.195	17.852	**<0.001**	6.159	4.998	12.950
Anti-thrombin III	-0.002	0.020	0.010	0.921	.998	0.959	1.039
Neutrophil to lymphocyte ratio	.0686	0.401	2.920	0.088	1.986	0.904	4.362
Platelet to lymphocyte ratio	0.011	0.017	0.452	0.501	1.011	0.979	1.045

B: Regression coefficient; S.E.: Standard error, CI: Confidence interval, DBP: Diastolic blood pressure

Table (4): Validity (AUC, sensitivity, specificity) for Cystatin D level, in prediction and detection of RA.

	Cutoff value	AUC	95%CI	Sensitivity	Specificity	PPV	NPV	P-value
Fibrinogen	2.91g/l	0.829	0.612-0.707	93.7%	56.2%	68.1%	98.9%	**<0.001**
Anti-thrombin III	85.6	0.661	0.612-0.707	62.5%	74.7%	68.8%	65.7%	**0.002**
Fibrinogen+ Anti-thrombin III	0.0923	0.845	0.806-0.879	75%	79.3%	78.4%	76%	**<0.001**

AUC: Area Under a Curve, p-value: Probability value NPV: Negative predictive value PPV: Positive predictive value*: Statistically significant at p≤0.05

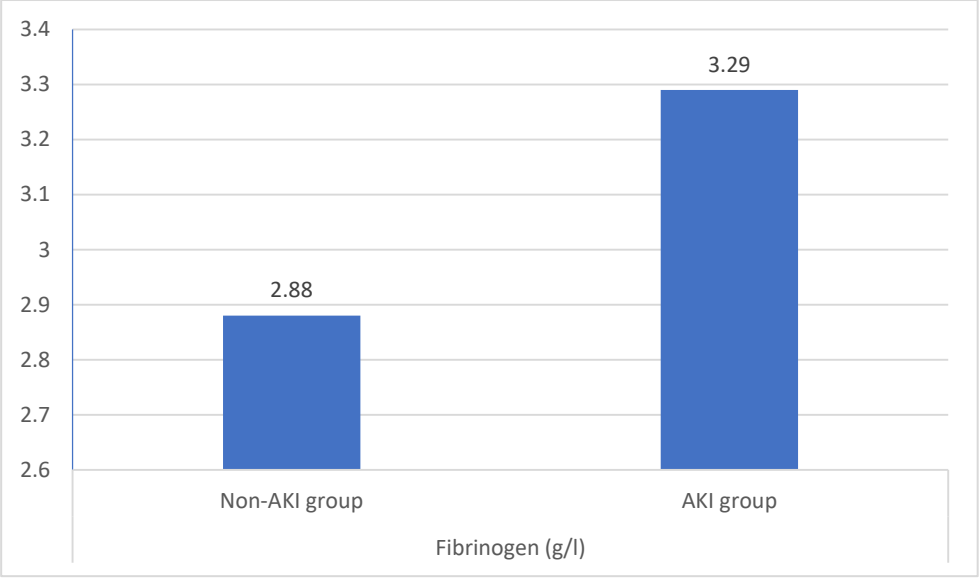

Figure (1): Mean fibrinogen level in AKI and non - AKI groups

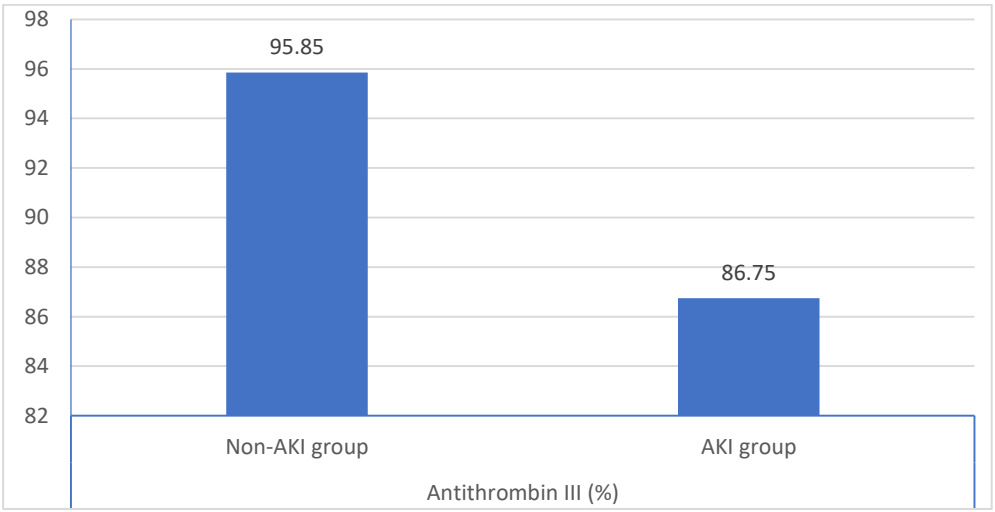

Figure (2): Mean Antithrombin – III activity level in AKI and non -AKI groups

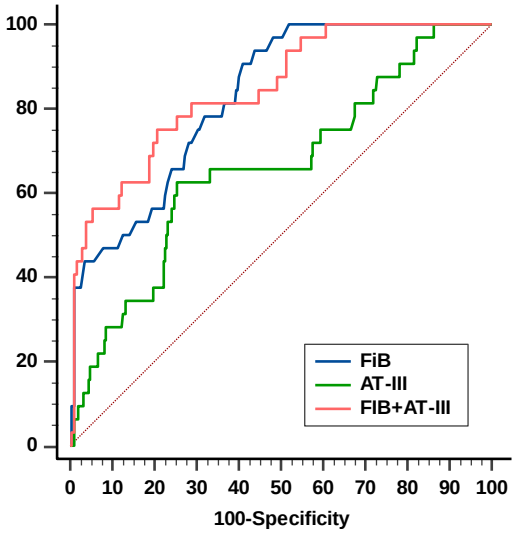

Figure (3): ROC curve of Fibrinogen, Anti-thrombin-III and combined Fibrinogen + Anti-thrombin-III in differentiation between AKI group from non-AKI group

Discussion

CIN refers to renal dysfunction or AKI that occurs as a result of the use of contrast agents during medical imaging procedures, such as PCI. [16]

The risk of CIN is particularly heightened in patients with pre-existing renal impairment, diabetes, and advanced age. The type and volume of contrast media utilized during PCI contribute significantly to the nephrotoxic potential, with higher volumes and certain agents associated with an increased risk. Hemodynamic changes induced by PCI, such as alterations in blood pressure and systemic perfusion, can further compromise renal blood flow, exacerbating the likelihood of CIN. Effective monitoring of renal function post-PCI is imperative for early detection and intervention in cases of CIN. Regular assessments of SCr levels allow clinicians to promptly identify signs of renal impairment and implement appropriate management strategies. Risk stratification is essential, and an individualized approach to preventive measures is crucial in optimizing patient outcomes . [17]

Fibrinogen is a protein involved in blood clotting, and increased levels may indicate a pro-thrombotic state [18]. AT-III, on the other hand, is a

protein that inhibits clotting factors, and decreased levels may suggest an impaired ability to prevent excessive clotting . [18] Both are implicated in inflammatory and thrombotic processes, which are relevant to CIN and potential involvement of these factors in the vascular response to contrast media.

While various scoring system have been established to predict CIN by considering known risk factors, the exploration of novel biomarkers could enhance the identification of patients at risk. Individually, fibrinogen or AT-III show limited utility in predicting CIN risk . (20) The lack of available studies examining these biomarkers in combination for CIN prediction suggests that the collective impact of fibrinogen and AT-III on renal outcomes in the context of PCI remains largely unexplored. Our study aimed to investigate the potential synergistic effects of these biomarkers and their combined predictive utility for CIN. Addressing this void in knowledge could contribute valuable insights into refining risk assessment strategies and developing targeted interventions to mitigate the risk of CIN in CAD patients undergoing PCI.

The current study involved 400 patients undergone PCI presents valuable insights in to the prediction CIN. Notably, 8% of the cohort developed CIN, and we found an association between AKI and higher BMI, diastolic blood pressure, diuretic and low molecular weight heparin (LMWH) usage, as well as elevated levels of glucose, NLR, PLR, and fibrinogen. Multivariate analysis identified BMI, triglycerides, albumin, glucose, and fibrinogen as independent predictors for AKI post-PCI.

The robust AUC of 0.829 for fibrinogen, along with an optimal cut off value of 2.91g/l, while for AT-III, the AUC was 0.661, with an optimal cutoff value of 85.6% underline their potential as a predictive biomarker. Combining fibrinogen and AT-III further improved predictive accuracy, as evidenced by the elevated AUC of 0.845. These findings suggest a promising role for fibrinogen and AT-III in risk stratification for AKI in PCI patients.

In the same line, Sun et al. [21] conducted a study, which was the only trial utilizing the combination of fibrinogen and AT-III for predicting CIN in patients undergoing PCI. The study included 394 patients who underwent PCI, of whom 12% developed CIN, distinctive biomarker and clinical profiles were observed, with elevated fibrinogen and decreased AT-III activity, as well as lower albumin levels. Myocardial infarction emerged as a significant risk factor for CIN, while within the CIN group, the utilization of diuretics and low molecular weight heparin increased in prevalence. Lower albumin levels, increased fibrinogen levels, and decreased AT-III levels were identified by multivariate analysis as independent predictors of CIN. The AUC of 0.653 for fibrinogen, along with an optimal cutoff value of 3.48g/l, while for AT-III, the AUC was 0.711, with an optimal cutoff value of 89.5% underline their potential as a predictive biomarker. Combining fibrinogen and AT-III further improved predictive accuracy, as evidenced by the elevated AUC of 0.747. Despite similar biomarker findings in our study and Sun et al. study, differences in patient characteristics, risk factors, and independent predictors highlight the multifaceted nature of CIN development. Notably, both studies utilized distinct sets of risk factors and reported variations in diagnostic accuracy, with the combination of fibrinogen and AT-III consistently enhancing predictive capabilities. These findings underscore the complexity of CIN prediction, emphasizing the importance of considering a range of factors and combined biomarkers for a comprehensive risk assessment.

On the other hand, the study of Wu et al. [12]. which focused on patients undergoing CAG, with 27.17% developing CIN. Low AT-III activity was associated with a significantly higher incidence of CIN. The study classified AT-III activity into four grades and found that lower AT-III activities were associated with higher CIN incidence. The study identified AT-III activity < 75% as an independent predictor of CIN, along with baseline SCr.

The study of Li et al. [22] focused on acute coronary syndrome (ACS) patients and found that elevated baseline fibrinogen levels, when combined with CHA2DS2-VASC scores, were positively correlated and associated with a higher incidence of CI-AKI. The combination had specificity of 72.6%, an AUC of 0.727, and a sensitivity of 63.3%. Both studies underscore the importance of considering hemostatic and inflammatory markers in assessing the risk of renal complications in cardiovascular patients, providing valuable insights for risk stratification and potential preventive measures. The differences in patient populations and methodologies highlight the versatility of these markers across various cardiovascular conditions.

The Celik et al. [23] study included a larger sample size, in which CI-AKI occurring in 10.6% of 706 patients. This study reported that serum fibrinogen levels were significantly elevated in patients who developed CI-AKI compared to those who did not. The multivariate logistic regression analysis in this study revealed that serum fibrinogen level was associated with the development of CI-AKI, with an OR of 1.006. They suggested a cutoff value of 410 mg/dl for fibrinogen for predicting CI-AKI.

Our results underscore the significance of various clinical and biochemical parameters in predicting the risk of CIN in patients undergoing PCI. The association of higher BMI, lower levels of triglycerides and albumin, elevated glucose levels, and increased fibrinogen with the development of AKI suggests the multifactorial nature of renal injury in this context. The combination of fibrinogen and AT-III as independent predictors enhances the predictive accuracy, as indicated by the improved AUC. The study's findings may have clinical implications, potentially guiding risk assessment and preventive measures in PCI patients to mitigate the occurrence of AKI.
It's worth noting that the study's limitations, such as its single-center design. Additionally, the identified biomarkers, while promising, should be further evaluated in larger cohorts and diverse populations to establish their generalizability and clinical applicability. Overall, these findings contribute valuable insights into the potential utility of specific biomarkers, particularly fibrinogen and AT-III, in predicting and managing AKI in the context of PCI, offering a foundation for future research and clinical considerations.

Conclusion:

The current study highlights BMI, triglycerides, albumin, glucose, fibrinogen and AT-III as independent predictors for CIN. The combination of fibrinogen and AT-III significantly improves predictive accuracy. If the fibrinogen level is found to be elevated and the AT-III level is decreased, it may indicate an increased risk of developing CIN. These findings emphasize the multifactorial nature of CIN risk and suggest potential biomarkers for enhanced risk stratification in PCI patients.

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