ISSN: 0975-3583,0976-2833 VOL13, ISSUE 03, 2022

ORIGINAL RESEARCH

Metabolic Syndrome as a Risk Factor for Development of Contrast Induced Nephropathy after Percutaneous Coronary Intervention among Non-Diabetic Indian Patients: A Prospective, Case- Control Observational Study

[1]Dr Chandra Mohan, [2]Dr. Anurag Rawat, [3]Dr Mansi Kala, [4]Dr Kunal Gururani

[1,4]Assistant Professor, [2]Associate Professor, Department of Cardiology, Himalyan Institute of Medical Sciences, Dehradun, Uttarakhand, India
[3]Associate Professor, Department of Pathology, Himalyan Institute of Medical Sciences, Dehradun, Uttarakhand, India

Correspondence:
Dr Kunal Gururani
Assistant Professor, Department of Cardiology, Himalyan Institute of Medical Sciences, Dehradun, Uttarakhand, India
Email: drgururanik@gmail.com

Abstract

Background: Contrast-induced nephropathy (CIN) after percutaneous coronary intervention (PCI) is associated with prolonged hospitalization along with long-term mortality and morbidity rates and increased financial expenditure. Diabetes is already a well known risk factor for development of CIN after percuataneous coronary intervention. Non-diabetic patients with Metabolic Syndrome (MetS) may have increased risk of developing CIN after PCI. At present, little data exist about the impact of metabolic syndrome as a risk factor in non-diabetic patients after PCI.

Aim: The purpose of the study is to evaluate MetS as a risk factor for the development of CIN after elective PCI among hemodynamically stable non-diabetic patients.

Materials and methods: This was a single center, prospective, case-control, observational study conducted between March, 2017 and December, 2017. A total of 500 patients were enrolled, out of which 267 patients were included in MetS group and 233 patients were included in Non-Mets group. Data regarding baseline demographic, procedural and angiographic characteristics and hospital outcomes were collected. All the patients were evaluated for the development of CIN after elective PCI.

Results: The incidence of CIN was observed in 44 patients (16.8%) in the MetS group and 14 patients (6%) in the Non-MetS group (p=<0.001). MetS(OR: 1.4, 95% CI:2.1-9.3; p=0.03), eGFR<60 ml/min/1.73m^2(OR: 13.4, 95% CI:3.9-45.4; p<0.001)and contrast volume>200 ml(OR: 16.7, 95% CI:4.6-57.6; p<0.001)were independent predictors of CIN. The MetS patients who developed CIN had prolonged hospital stay (4.7 ± 1.4 vs.3.7 ± 0.15; p=0.048)and more complicated clinical course than Non-MetS patients. No mortality was observed in both the groups.

Conclusion: The MetS was found as a risk factor for the development of CIN after elective PCI among non-diabetic patients. Hence, prior to PCI, the identification of MetS patients are of utmost importance to prevent the incidence of CIN.

Keywords: Metabolic syndrome, nephropathy, percutaneous coronary intervention, risk factor.

ISSN: 0975-3583,0976-2833 VOL13, ISSUE 03, 2022

Introduction

Contrast-induced nephropathy (CIN) has been emerged as a critical complication of percutaneous coronary intervention (PCI) (1), resulting from administration of iodinated contrast media(2).CIN is defined as an elevation of serum creatinine (SCr) of ≥ 50% (1.5-fold from baseline), ≥ 0.3 mg/dl (≥ 26.4 μmol/l) from baseline or a reduction in urine output (documented oliguria of less than 0.5 ml/kg per hour for more than six hours) within 48 hours of intravascular contrast media administration (3). CIN is the third common cause of hospital acquired acute kidney injury (AKI), accounting for approximately 12% of the cases (4). The reported incidence of CIN after PCI has been ranged from less than 2% to 50%, based on risk factors and diagnostic criteria used (5,6). Screening of patients undergoing PCI for likelihood to develop CIN is of paramount importance, as CIN is related to prolong hospitalization along with long-term mortality and morbidity rates and increased financial expenditure (7).

Metabolic syndrome (MetS) is a set of risk factors including central obesity, hypertriglyceridemia, dyslipidemia, impaired glucose tolerance, and hypertension(8), which are strongly related with increased morbidity and mortality events of diabetes, cardiovascular and renal diseases(9,10,11). To the best knowledge, very few publications are available in the literature that discuss the impact of MetS as a risk factor for the development of CIN (12,13); no large-scale study in Indian population has been reported. The present study is thus to examine the effect of MetS as a risk factor for the development of CIN after PCI in Indian non-diabetic patients.

Methods

A single center, prospective, case-control, observational study was performed in patients who developed CIN after elective PCI at our center. The study enrolled 568 patients from March, 2017 until December, 2017 at a tertiary healthcare center. Patients were assigned into two groups: 267 in the MetS group and 233 in the non-MetS group. The end point of study was the risk assessment of MetS for the development of CIN.

All the patients, who required intravenous injection of iodinated contrast media during elective PCI were included. Exclusion criteria were patients with diabetes, cardiogenic shock, hypotension, acute left ventricular failure (LVF) or clinical heart failure, administration of iodinated contrast in 7 days, estimated glomerular filtration rate (eGFR)<30ml/min/1.73m^2 or known case of end-stage renal disease or patient on hemodialysis, left ventricular ejection fraction (LVEF)<30%, contrast allergy, history of acute kidney injury (AKI) in last 7 days, exposure to nephrotoxic agents in last 7 days, acute renal failure of any cause in last 7 days, pregnant females, and patient not willing to give consent for study. The study complied with the Declaration of Helsinki and was approved by the Institutional Ethics Committee.

Blood samples were collected to measure white blood cells count, high sensitivity c-reactive protein (hs-CRP), troponin -I (in cases of acute coronary syndrome), lipid profile, renal and liver profile, baseline blood sugar, and erythrocyte sedimentation rate. Anthropological measurements were also carried out for waist measurement and hip measurement. Serum creatinine levels were measured before and after (48 hours) the procedure. eGFR was calculated using modification of diet in renal disease (MDRD) formula: 186.3X [S. Cr $^{-1.154}$] X [Age $^{-0.203}$] [X 0.742 for females] [X 1.210 for African Americans](14)using a mobile application.

The CIN was defined as an absolute increase in serum creatinine of ≥0.3 mg/dl (≥26.4 μmol/l), a percentage increase in serum creatinine of ≥50% (1.5-fold from baseline), or a reduction in urine output (documented oliguria of less than 0.5 ml/kg per hour for more than six hours) within 48 hours of intravascular contrast media administration (3). Identification of MetS confirmed to the definition used by the International Diabetes Federation (IDF) 2005 guidelines. MetS was defined as the presence of central obesity (defined as waist

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circumference [if BMI is >30kg/m², central obesity can be assumed, and waist circumference does not need to be measured] with ethnicity specific values) plus any two of the following four factors: hypertriglyceridemia (≥150 mg/dL), low high density-lipoprotein (HDL) cholesterol (<40 mg/dL in males and <50 mg/dL in females), high blood pressure (≥ 130/85 mmHg or drug treatment for hypertension), high fasting blood glucose (FBG) (> 100 mg/dl or previously diagnosed type 2 diabetes) (15). Diabetes mellitus was defined as a fasting blood glucose level >126 gm/dl, or a clinical diagnosis of diabetes with dietary, oral, or insulin treatment. The impaired fasting glucose level or pre-diabetes was defined as > 110mg/dl and serum glucose level as <126 mg/dl (16). According to National Kidney Foundation (NKF) guidelines, renal sufficiency was divided into three groups: mild (cutoff value of≥60 to ≤89 ml/min/1.73m^2), moderate (cutoff value of ≥30 to ≤59 ml/min/1.73m^2), and severe (cutoff value of <30 ml/min/1.73m^2) (17).

Coronary angiography was performed using a low osmolar, iodinated contrast agent (iohexol, Excelscan300/ iohexol, Omnipaque). Iodinated non-ionic contrast agent (iodixanol, Visipaque) was used in patients with the history of pre-procedural renal impairment.

Sample size was estimated using reported event rates in the previously prospective studies:14% in the MetS group and 3.6% in the non-MetS group. The minimum sample size of 308 was required to detect a statistically significant difference with power of90% (α=0.05). All analyses were performed using the Statistical Package for Social Sciences, version15 (SPSS, Chicago, IL, USA). Discrete variables are expressed as frequencies, while continuous variables are expressed as mean ± SD. Univariate logistic regression was performed on the following variables: age (years), male, currents smoker, hypertension (mm Hg), history of contrast procedures, angiotensin-converting enzyme inhibitor/angiotensin receptor blocker (ACEI/ARB), statins, central obesity (waist cm), total cholesterol(mg/dl), low density lipoprotein (LDL) (mg/dl), high density lipoprotein (HDL) (mg/dl), triglyceride(mg/dl), and FBG(mg/dl), LVEF %, multivessel intervention, eGFR <60 ml/min/1.73m^2, contrast volume >200 ml, and MetS. Univariate correlates (LVEF %, multivessel intervention, eGFR <60 ml/min/1.73m^2, contrast volume >200 ml, and MetS)were included in the multiple logistic regression analyses. A two-sided p value of <0.05 was considered statistically significant. Receiver operating characteristics (ROC) curve analyses of contrast volume and FBG for the development of CIN, and determination of the cutoff point for the contrast volume and FBG were performed.

Results

A total of 568 non diabetic patients were screened for elective PCI, out of which 11 patients were excluded due to eGFR <30 ml/min/1.73m^2, 8 patients were excluded due to LVEF<40 %, and 49 patients were excluded due to loss of follow-up. Therefore, this study involved 500 patients who were allocated to MetS and non-MetS groups **(Figure 1)**.

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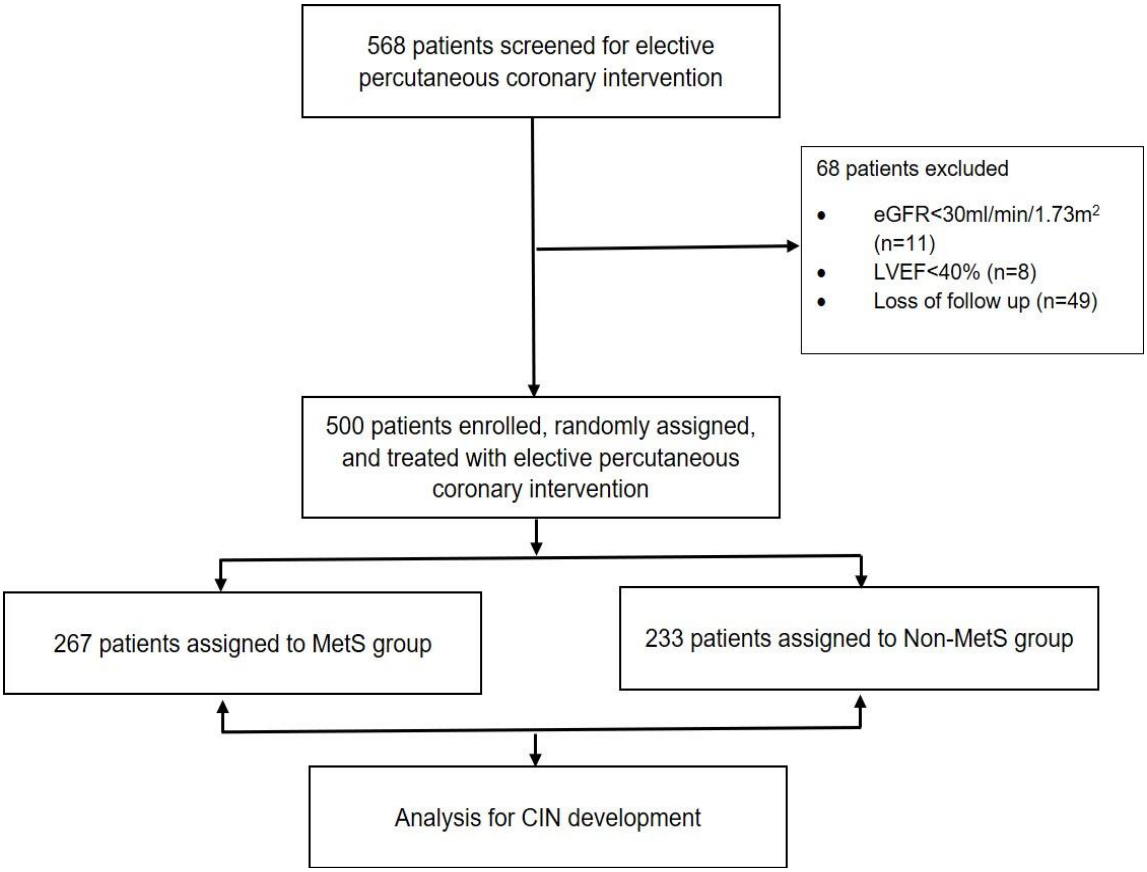

Figure 1: Study Flowchart.

‡eGFR, estimated glomerular filtration rate; LVEF, left ventricular ejection fraction; MetS, metabolic syndrome; CIN, contrast induced nephropathy.

Baseline demographic, procedural and angiographic characteristics were significantly different between the two study groups **(Table 1)**.

Table 1: Baseline demographics, procedural and angiographic characteristics of study groups

Characteristics	Patients with MetS (N=267)	Patients without MetS (N=233)	p value
Age, years	60.3 ± 10.2	59.3 ± 9.4	0.207
Male	187(70%)	185 (79.4%)	0.017
Current smoker	64 (23.9%)	48 (20.6%)	0.37
Hypertension, mm Hg	114 (42.7%)	26 (11.1%)	<0.01
FBG, mg/dl	97.3 ± 18.2	83.4 ± 16.1	<0.01
BMI, kg/m^2	26.73 ± 3.67	22.99 ± 3.53	<0.01
Central obesity (waist cm)	94.9 ± 7.4	83.4 ± 16.1	0.034
Total cholesterol, mg/dl	166.50 ± 49.65	162.40 ± 71.03	0.25
HDL, mg/dl	39.54 ± 8.75	43.06 ± 9.25	<0.01
LDL, mg/dl	95.12 ± 41.8	97.70 ± 68.00	0.27
Triglyceride, mg/dl	158.04 ± 65.4	113.62 ± 38.91	<0.01
Baseline Medications			
ACEI/ARB	173 (64.79%)	154 (66.1%)	0.76
Statins	233 (87.26%)	215 (92.2%)	0.07
Beta blocker	198 (74.16%)	164 (70.38%)	0.34
CCB	63 (23.6%)	23 (9.01%)	<0.01
Previous MI	179	171	0.12

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LVEF, %	59.29 ± 10.9	58.20 ± 10.3	0.14
Clinical Indications for PCI			
Previous AWMI	56 (20.98%)	36 (14.45%)	0.11
Previous IWMI	35 (13.10%)	46 (19.74%)	0.044
SIHD	38 (14.23%)	20 (8.58%)	0.049
UA	50 (18.73%)	42 (18.02%)	0.84
NSTEMI	88 (32.95%)	89 (38.19%)	0.22
History of contrast procedures	71 (26.6%)	53 (22.75%)	0.32
Procedural Characteristics			
Single vessel intervention	109 (40.82%)	100 (42.92%)	
Multiple vessel intervention	158 (59.18%)	133 (57.09%)	0.64
Volume of contrast agent, ml	170.39 ± 40.90	176.48 ± 61.22	0.12

‡ MetS, metabolic syndrome; FBG, fasting blood glucose; BMI, body mass index; LDL, low-density lipoprotein; ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; CCB, calcium channel blocker; MI, myocardial infarction; LVEF, left ventricular ejection fraction; AWMI, anterior wall myocardial infarction; IWMI, interior wall myocardial infarction; SIHD, stable ischemic heart disease; UA, unstable angina; NSTEMI, non-ST segment elevation myocardial infarction; SD, standard deviation.
Data are presented as mean ± SD or n (%).

A higher prevalence of male patients was found in both the groups (70% vs. 79.4%, p=0.017), with a mean age of 60.3 ± 10.2 and 59.3 ± 9.4 years, respectively. All the components of the MetS were significantly higher in MetS group compared to non-MetS group: central obesity (94.9 ±7.4 cm vs. 83.4 ±16.1 cm; p=0.034); triglycerides (158.04 ± 65.4 vs. 113.62± 38.91; p<0.01); hypertension (42.7% vs. 11.1%; p<0.01); FBG (97.3 ± 18.2 vs. 83.4 ± 16.1; p<0.01). The lower level of HDL in MetS group (39.54 ± 8.75 vs. 43.06 ± 9.25; p<0.01) was found compared to non-MetS group. Both groups were injected with mean volume of contrast agent (170.39 ± 40.90 ml vs. 176.48 ± 61.22 ml; p=0.12). **Table 2** shows baseline renal parameters and post PCI changes of SCr and GFR in the study groups.

Table 2: Baseline renal parameters and post PCI changes in the study groups

Parameter	**Patients with MetS (N=267)**	**Patients without MetS (N=233)**	**p value**
SCr, mg/dl			
Baseline	1.01 ± 0.27	0.97 ± 0.24	0.12
Post-procedure*	1.17 ± 0.39	1.05 ± 0.27	<0.01
Absolute change	0.264 ± 0.267	0.078 ± 0.184	<0.001
eGFR (ml/min/1.73m^2)			
Baseline	79.8 ± 26.4	85.5 ± 25.11	0.03
Post-procedure*	69.71 ± 24.61	77.15 ± 22.27	<0.01
Absolute change	-10.65 ± 17.97	-7.728 ± 16.96	0.03
CIN	**44(16.8%)**	**14(6%)**	**<0.001**
Baseline SCr of patients with CIN, mg/dl	1.26 ± 0.20	1.17 ± 0.37	0.1
Post procedural SCr of patients with CIN, mg/dl	1.89 ± 0.21	1.46 ± 0.52	<0.001
Absolute change	0.63 ± 0.22	0.29 ± 0.36	<0.001
Baseline eGFR of patients with CIN, mg/dl	59.22 ± 12.54	66.26 ± 18.72	0.06lk
Post procedural eGFR of patients with CIN, mg/dl	36.69 ± 6.34	57.01 ± 32.63	<0.001
Absolute change	-22.53 ± 11.87	-9.25 ± 21.31	0.002
* Procedure after 48 hours.			

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‡ MetS, metabolic syndrome; SCr, serum creatinine; eGFR, estimated glomerular filtration rate; CIN, contrast-induced nephropathy; SD: standard deviation.
Data are presented as mean ±SD or n (%).

Baseline and post procedure SCr were higher in the MetS group, while baseline and post procedure GFR were found to be lower. Compared to non-MetS group, a significantly higher rate of CIN was found in the MetS group(16.48% vs. 6.0%; p<0.001). The amounts of the contrast volume were similar in both the groups(170.39 ± 40.90ml vs. 176.48 ± 61.22ml; p=0.12).

The predictive values of various factors were evaluated to identify the impact of MetS on CIN.

Univariate regression model found the following significant risk factors for the development of CIN:hypertension≥130/85 mm Hg, central obesity(waist cm), triglyceride ≥150 mg/dl, FBG>100 mg/dl, LVEF<40%, multivessel intervention, eGFR<60ml/min/1.73m^2, contrast volume>200ml, and presence of MetS.

As shown in **Table 3**, multivariate regression model identified the following five attributes as risk factors for the development of CIN:LVEF<40%, multivessel intervention, eGFR<60 mL/min/1.73m^2, contrast volume>200ml, presence of MetS. (LVEF<40%, p=0.002; multivessel intervention, p=0.03; eGFR<60 ml/min/1.73m^2, p<0.001; contrast volume >200 ml, p<0.001; MetS, p=0.03).

Table 3: Analysis of risk factors for CIN

Variable	Univariate			Multivariate		
	OR	**95% CI**	**p value**	**OR**	**95% CI**	**p value**
Old age (>65 years)	1.79	1.03-3.12	0.38			
Male	0.8	0.44-1.15	0.5			
Current smoker	1.1	0.59-2.12	0.73			
Hypertension≥ 130/85 mm Hg	2.75	1.57-4.80	0.004			
History of contrast procedures	1.08	0.61-1.93	0.77			
ACEI/ARB	1.76	0.93-3.3	0.07			
Statins	0.5	0.23-1.06	0.07			
Central obesity*(waist cm)	4.27	1.79-10.1	0.001			
Total cholesterol>200 mg/dl	0.66	0.30-1.44	0.3			
LDL>100 mg/dl	0.78	0.44-1.38	0.39			
HDL, males (<40 mg/dl)or females(<50 mg/dl)	1.36	0.77-2.37	0.28			
Triglyceride ≥ 150 mg/dl	2.1	1.24-3.7	0.006			
FBG>100 mg/dl	4.04	2.08-7.8	<0.001			
LVEF< 40%	2.61	1.17-5.85	0.02	9.862	2.3-41.2	0.002
Multivessel intervention	1.85	1.03-3.33	0.03	1.85	1.03-3.33	0.03
eGFR<60 ml/min/1.73m^2	9.36	5.17-16.9	<0.001	13.4	3.9-45.4	<0.001
Contrast volume >200 ml	5.63	3.11-10.2	<0.001	16.7	4.6 - 57.6	<0.001
MetS	3.08	1.64-5.79	0.005	1.4	2.1-9.3	0.03

*If BMI is >30kg/m², central obesity can be assumed, and waist circumference does not need to be measured.
‡ ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; LDL, low-density lipoprotein; HDL, high-density lipoprotein; FBG, fasting blood glucose; LVEF, left ventricular ejection fraction; eGFR, estimated glomerular filtration rate; MetS, metabolic syndrome; OR, odds ratio; CI, confidence interval.

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Multivariate regression model applied separately on individual components of MetS revealed that hypertension≥130/85 mm Hg and FBG>100 mg/dl) increased the probability for the development of CIN (p value = 0.019 and 0.046), as demonstrated in **Table 4.**

Table 4: Multi variate regression analysis for component of MetS as risk factors for CIN

Variable	Multivariate		
	OR	**95% CI**	**p value**
Triglyceride ≥ 150 mg/dl	3.06	0.81-10.7	0.07
Hypertension ≥ 130/85 mm Hg	3.95	1.24-12.48	0.019
FBG >100mg/dl	3.03	1.03-9.885	0.046
Central obesity*(waist cm)	1.3	0.2-8.9	0.73
HDL, males (<40 mg/dl) or females(<50 mg/dl)	3.3	0.8-12.5	0.76

* If BMI is >30kg/m², central obesity can be assumed, and waist circumference does not need to be measured.
‡ FBG, fasting blood glucose; HDL, high-density lipoprotein; OR, odds ratio; CI, confidence interval.

The ROC curve analysis of contrast volume identified the best cut-off value for contrast volume as 202 ml for patients with MetS, with a sensitivity value of 67% and a specificity value of 78% (area under curve (AUC):0.694, p=0.004). A higher cut-off value for contrast volume was found among patients without MetS (cut-off value: 232 ml, sensitivity 71%, specificity 77%, AUC: 0.882, p=0.01; **Figure 2**).

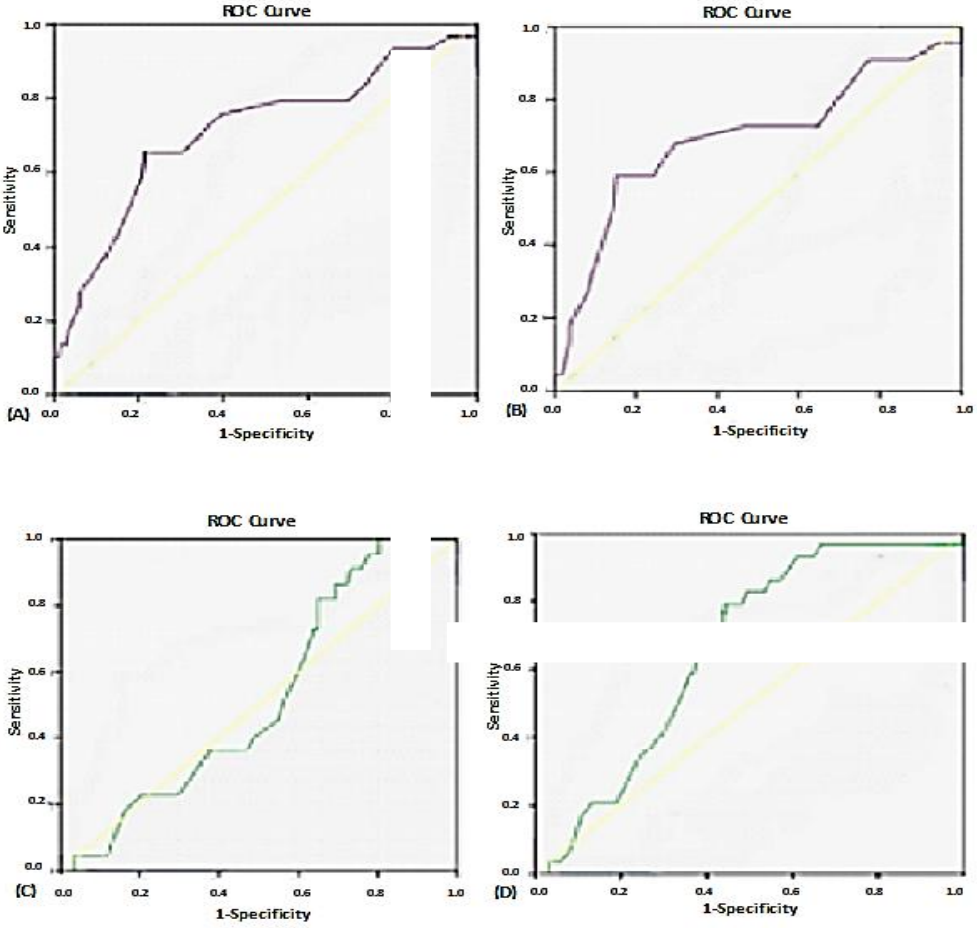

Figure 2: ROC curve for the development of CIN (A) Contrast volume in MetS patients, cutoff value: 202 ml/min, sensitivity 67%, specificity 78%, AUC:0.694, p=0.004,(B) Contrast volume in non-MetS patients, cutoff value: 232 ml/min, sensitivity 71%, specificity 77%,

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AUC:0.882, p=0.01,(C) FBG in MetS patients, cutoff value: 95 ml/min, sensitivity 71%, specificity 77%, AUC:0.659, p=0.005,(D) FBG in Non-MetS patients, cutoff value: 95 ml/min, sensitivity 67%, specificity 65%, AUC:0.734, p=0.038

‡ AUC, area under curve; CIN, contrast-induced nephropathy; ROC, receiver-operating characteristic; MetS, metabolic syndrome.

For ROC curve analysis of FBG (mg/dl), the cut-off value was same in both groups having high risk for the development of CIN(for MetS cut-off value: 95 mg/dl, sensitivity 71%, specificity 77%, AUC:0.659, p=0.005 while for non-MetS cut-off value: 95 mg/dl, sensitivity 67% ,specificity 65%, AUC:0.734, p=0.038; **Figure 2**). It concluded that in the non-MetS group, FBG >95mg/dl was found to be high-risk predictor for the development of CIN.

Table 5 presents the in-hospital outcomes in study groups. No mortality event was reported in both the groups. The requirement of dialysis in MetS group who developed CIN were significantly higher compared to non-MetS group with CIN (4.5% vs. 0%; p=0.18).

Table 5: In-hospital outcomes in study groups

Parameter	Patients with MetS (N=267)	Patients without MetS (N=233)	p value
Pulmonary Edema	9 (0.02%)	7 (3.0%)	0.83
Hypotension	22 (8.2%)	18 (7.7%)	0.81
Patients with CIN requiring dialysis	2 (4.5%)	0 (0%)	0.18
Length of hospital stay (days)	4.7 ± 1.4	3.7 ± 0.15	0.048
Mortality	0 (0%)	0 (0%)	-
‡ MetS, metabolic syndrome; CIN, contrast-induced nephropathy; SD: standard deviation. Data are presented as mean ±SD or n (%).			

Discussion

The present study investigated the effect of MetS as a risk factor for the development of CIN after PCI among non-diabetic patients. Although, multiple studies concluded that MetS has increased the risk of developing CIN after elective PCI (12,13), still there is lack of large-scale clinical study in Indian population. Components of MetS, such as impaired glucose tolerance(12), hypertension (20), hypertriglyceridemia (21), emerged as a risk factor for CIN.

The prognosis of patients who developed CIN after PCI is found to be worse than of those without renal injury (23).Supportive measures and dialysis are the preferred treatment for the established CIN, as the patients who developed CIN after PCI are found more susceptible for stroke, bleeding, electrolyte imbalance, sepsis, respiratory failure, and pulmonary embolus (24-26).Various risk factors such as nephrotoxic drugs, congestive heart failure, diabetes, anemia, baseline renal dysfunction, volume depletion, hemodynamic instability, and hypoalbuminemia are considered as an etiologic factor for the development of CIN(27). The Mehran Risk Score (28) is used to reduce the likelihood of developing CIN after PCI. It is directly related to persistent renal impairment after CIN (29). Numerous preventive methods are recommended for the patients who are at risk for CIN and other causes of renal failure:sodium bicarbonate infusion, high-dose oral N-acetylcysteine consumption, preprocedural hydration with saline, the cessation of nephrotoxic agents, the use of statins, iso-osmolar contrast agent replacement, and the limitation of contrast agent volume (13, 29-32). Hence, it is essential to screen high-risk patients before PCI and starting the suitable preventive regimens for the reduction of CIN.

In the current study, we showed that Mets increased the risk of CIN who underwent PCI among the non-diabetic patients in Indian population. The incidence of CIN was found to be 16.8 %. The findings obtained from our study revealed that patients with MetS showed a higher level of post procedural creatinine and a significant decrease of post procedural GFR

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in comparison with non-MetS patients. The present study also demonstrated the presence of multivessel intervention as other risk predictors for CIN. Similarly, Ozcan, et al. (13) reported a higher level of post procedural creatinine and lower level of post procedural GFR in the study patients. It also found multivessel intervention as a risk factor for CIN. In agreement with Kroneberger, et al. (33) our study also found that lower eGFR had the greatest risk for the development of CIN.

Another promising finding was that the lower level of baseline contrast volume developed CIN in MetS patients (cutoff values for baseline contrast volume to develop CIN is 202 ml vs. 232 mlin patients with MetS and non-MetS, respectively).Furthermore, lower sensitivity and specificity rates of contrast volume were found in both groups. The cutoff values of FBG were found similar in both groups for the development of CIN, whereas in these patients, higher sensitivity and specificity rates of FBG were found. As previously stated in the various studies (34,35), higher values of baseline contrast volume and FBG augment the incidence of CIN in high-risk patients. However, all these data about baseline contrast volume and FBG from our study may provide insight into the preventive measures taken by clinicians before starting the PCI procedure.

The present study has some limitations that need to be addressed. An apparent limitation is single-center study, so this study is not representative for the general Indian population but only for a region. Although the present study used the well-known and earlier investigated clinical biomarkers for the detection of renal dysfunction such as SCr and GFR, novel biomarkers (neutrophil gelatinase-associated lipocalin, N-acetyl glycosaminidase, and cystatin C) are yet to be further investigated in clinical studies. Further studies that investigate the impact of baseline medication on MetS patients for the development of CIN could prove beneficial for the future treatment of such patients.

Conclusion

This study has highlighted that MetS increases the risk for the development of CIN after elective PCI in nondiabetic patients and further identified that higher contrast volume (> 200 ml) was a significant predictor for CIN in MetS patients. Our recommendation is that clinicians should investigate the MetS patients before elective PCI to reduce the CIN incidence.

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