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Risk Factors Associated with Contrast-Induced Nephropathy after Primary Percutaneous Coronary Intervention

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ABSTRACT

Background: Contrast induced nephropathy (CIN) could be a serious outcome complicating primary percutaneous coronary intervention for STEMI patients affecting both morbidity and mortality. **Objective:** To determine the frequency of CIN and evaluate risk factors that predispose and increase incidence of CIN to determine risky individuals before primary PCI to put potential preemptive strategies to minimize the CIN. **Patients and Methods:** This study was conducted at the department of cardiovascular medicine, Tanta University hospital at the period between June 2021 to December 2021. Prospectively carried out on 100 patients diagnosed definitively with STEMI and treated with primary percutaneous coronary intervention to assess risk factors of contrast induced nephropathy after the procedure. **Results:** In the present study we found that old age, diabetes mellitus, ischemic cardiomyopathy, ischemic stroke, increased random blood sugar at admission and increase amount of contrast during the procedure have statistically significant relation with developing CIN on the other hand smoking, gender, dyslipidemia, infarcted artery, TIMI flow post procedure have no statistically significant relation with the risk of developing CIN. **Conclusion:** CIN after primary PCI is a common complication, and patients with older age, diabetes mellitus, previous ischemic cardiomyopathy (heart failure), previous ischemic stroke, hyperglycemia at admission are at increased risk of development of CIN. Also an increased amount of contrast during PCI procedure significantly increase the risk of post-procedure CIN.

Keywords: Contrast induced nephropathy, primary cutaneous coronary intervention, ST-segment elevation myocardial infarction

1. Introduction

Acute ST-segment myocardial infarction is one of the most important cardiovascular diseases that increase risk of morbidity and mortality (Steg *et al.,* 2018).

The primary goal in management of acute STEMI is reperfusion therapy with intravenous fibrinolysis or primary percutaneous intervention (O'Gara *et al.,* 2013).

Primary cutaneous coronary intervention (PCI) is the recommended and preferred reperfusion strategy for the patients with ST-segment elevation myocardial infarction (STEMI). When performed within the 12-hour window of symptom onset, it is an effective treatment strategy associated with a significant reduction in mortality and morbidity as compared to thrombolysis (Ibanez *et al.,* 2017).

A decrease in glomerular filtration rate after procedure due to injection of contrast media, known as contrast-induced nephropathy (CIN), remains a real risk and is associated with increased risk of mortality and morbidity, consequently leading to prolongation of hospital stay, increased utilization of resources, and increased healthcare cost (Batra *et al.,* 2018 & Azzalini *et al.,* 2016).

In recent literature, the frequency of CIN after primary PCI is reported to range from 10.4% to 23.2% (Batra *et al.,* 2018 & Ozturk *et al.,* 2016). CIN is reported to be associated with increased in-hospital mortality rate after primary PCI, with around four to eightfold increased risk of in-hospital mortality (Batra *et al.,* 2018 & Lucreziotti *et al.,* 2014).

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Despite widespread use of contrast agents in intervention and radiographic studies, the pathophysiology of CIN is complex and not fully understood and involves various mechanisms, such as oxidative stress, vasoconstriction, medullary ischemia, and allergic reactions to contrast media (Azzalini *et al.,* 2016 & Persson and Tepel, 2006). Various patient- and procedure-related factors have been observed to be reasons that can the exaggerate acute kidney injury (AKI), such as preexisting chronic kidney diseases (CKDs), diabetes, hemodynamic alterations, and volume depletion due to cardiogenic shock or heart failure, complex interventional procedures, and use of increased amount of contrast during the procedure (Batra *et al.,* 2018 & Wang *et al.,* 2019).

Potential preemptive strategies to minimize the CIN are risk stratification of high-risk individuals, optimization of volume status, monitoring of adequate hydrations and infusion of normal saline before and after the procedure, and use of minimum contrast amount during the procedure. Guidelines on various preventive nonpharmacological and pharmacologic agents are not clear, and there is poor concordance in the literature, and despite the varying degree of agreement, iso-osmolar contrast or nonionic low-osmolar contrast is the preferred media among the interventional cardiology (Kumar *et al.,* 2020 & Liu *et al.,* 2005).

Aim of the work

The aim of this work was to determine the frequency of CIN after primary PCI and its correlation with risk factors in patients with STEMI.

2.Patients and Methods

Patient population: This study was conducted at the department of cardiovascular medicine, Tanta University hospital at the period between June 2021 to December 2021. Prospectively carried out on 100 patients diagnosed definitively with STEMI and treated with primary percutaneous coronary intervention to assess risk factors of contrast induced nephropathy after the procedure.

STEMI is defined as: Persistent ST-segment elevation ≥ 2 mm in men or ≥1.5 mm in women in leads V2–V3, or ≥1 mm in two other contiguous chest or limb leads, with a shape consistent with ischemic ST elevation or: Isolated or most prominent ST-segment depression in leads V1–V3which is reciprocal to posterior ST elevation in leads V7-V9 (true posterior STEMI). In leads V7–V9, the ST-segment elevation cutoff is only 0.5 mm according to ESC guidelines (ESC, 2018).

CI AKI is defined as: Either an absolute increase in serum creatinine (Cr) concentration of (0.5 mg/dL) or a 25% relative increase in Cr from baseline (Lip *et al.,* 2012 & Lane *et al.,* 2012).

Inclusion criteria: Patients with ST segment elevation myocardial infarction undergoing PCI either gender, between 30 and 70 years of age, and normal serum creatinine level (<1.2 mg/dL) at baseline.

Exclusion criteria: Hemodynamically unstable patients, such as patients in shock or Killip class IV or those with pre-existing CKD or end-stage renal disease were excluded from the study.

The patients were subjected to: An informed consent taken from all patients. Full history taking with emphasis on: Age, sex, history of risk factors for coronary artery disease (CAD) as: Diabetes Mellitus, hypertension, smoking and past history and family history of CAD. Systemic hypertension: defined as systolic blood pressure of 140 mm Hg or more and/or diastolic blood pressure of 90 mm Hg or more measured on 3 separate occasions with or without treatment before admission (Williams *et al.,* 2018).

Diabetes: defined as having diabetes according to one of the following criteria (American Diabetes Association(ADA), 2014; Self-reported diabetes which was previously diagnosed by physicians or use of glucose-lowering drugs before hospitalization. Diabetes listed in the medical records as the secondary discharge diagnosis. Glycated hemoglobin A1c (HbA1c) concentration ≥ 6.5%.

Dyslipidemia is defined as serum total cholesterol level over 200mg /dl or triglycerides more than 150 mg /dl or current treatment with lipid lowering medication (P.B., 2012).

Current smoking an adult who has smoked 100 cigarettes in his or her lifetime and who currently smokes cigarettes (Botvin *et al.,* 1993).

Family history of premature coronary artery

Comprehensive Cardiac History: with a special focus on Previous history of ischemic heart disease (Previous MI/ ACS, Previous Revascularization e.g. PCI or surgical… etc.), history of cardiomyopathies**.**

Renal impairment: Chronic kidney disease is defined based on the presence of either kidney damage or decreased kidney function for three or more months, irrespective of the cause

Kidney damage is defined according to KDIGO 2012 CKD Guidelines as follows: (Nephrology and Dialysis, 2017).

Markers of kidney damage (one or more)	Albuminuria (AER ⩾30 mg/24 h; ACR ⩾30 mg/g (⩾3 mg/mmol))
	Urine sediment abnormalities
	Electrolyte and other abnormalities due to tubular disorders
	Abnormalities detected by histology
	Structural abnormalities detected by imaging
	History of kidney transplantation
Decreased GFR	GFR <60 ml/min per 1.73 m^2 (GFR categories G3a–G5)

Fig. 1: Criteria for CKD diagnosis according to KDIGO 2012 CKD.

History of past cerebrovascular events
Drug history: focusing on nephrotoxic drugs, history of previous statins therapy, OACs, Antiplatelet therapy.

Full clinical examination
Vital signs: heart rate, blood pressure and respiratory rate**.**

General examination: with attention to Height, weight, patient look, decubitus, cyanosis, jaundice, with special attention to signs of heart failure**.**

Local cardiac examination: abnormal pulsation, Heart sounds & murmurs**.**
Then clinical examination assessment of severity of heart failure using Killip score

Table 1: Hemodynamic Classification of Patients with Acute Myocardial Infarction. Modified from Killip T, Kimball (Killip and Kimball, 1676)

Class	Clinical presentation
I	Rales and S3 absent
II	Crackles, S3 gallop, elevated jugular venous pressure
III	Frank pulmonary edema
IV	Shock

Resting 12 leads ECG (ESC, 2018).

Standard 12-lead ECG was obtained within 10 minutes of first medical contact (FMC) according to ESC guidelines 2017 including: (limb leads I, II, III, aVR, aVL, aVF, and Chest leads from V1to V6) for all patients on admission to the hospital.

Right pericardial leads (V3R, V4R, V5R, V6R) and posterior chest leads (V7 to V9) were done for some patients to detect posterior wall and right ventricular infarction.

Typical criteria for ST-segment elevation in acute myocardial infarction (ESC, 2018): Measured at the J point, it should be found in two contiguous leads and be: ≥0.25 mV in men below the age of 40 years in leads V2, V3. ≥0.2 mV in men over the age of 40 years in leads V2, V3, or ≥0.15 mV in leads V2, V3 in women and or, ≥0.1 mV in other leads (in the absence of left ventricular (LV) hypertrophy or left bundle branch block (LBBB).

Table 2: Localization of site of STEMI using surface ECG (Thejanandan Reddy *et al.,* 2013):

LV wall affected	ST segment elevated in the following leads
Septal	V1, V2
Anterior	V1 to V6
Antro-septal	V1 to V4
Antro-lateral	V3 to V6 & Avl
Extensive anterior	V1 to V6, I & Avl
Inferior	II, III, Avf
Right	V3R, V4R, V5R
Posterior	V7 to V9

Venous sampling for laboratory data including:

Venous blood sample will be obtained on admission from each patient in the coronary care unit before PCI. Analysis of Cardiac Enzymes (Troponin I; CK-MB). Creatinine level before and after primary PCI. Estimated glomerular filtration rate (eGFR) before and after primary PCI. Complete Blood Count (CBC). RBG was tested once patients admitted to CCU.

Primary percutaneous intervention for Infarct related artery (IRA):

Preparation of the patient: Coronary angiography is performed under local anesthesia. The procedure is sterile, and all potential access sites is disinfected, shaved, and sterilized. At the beginning of the procedure, the patient lies down in supine position on the cardio-angiograph table, and is prepared for the procedure in sterile conditions.

Arterial puncture:

At the site of arterial puncture, an appropriate femoral or radial pulse has to be palpated in order to locate the artery. A local anesthesia is applied, usually with 10 ml of 1% or 2% lidocaine for local infiltration of the skin and subcutaneous tissues. The puncture of the femoral artery is performed some 2 cm below the inguinal ligament. All patients were subjected to primary percutaneous coronary intervention for the Infarct related artery (IRA). All patients receive aspirin (Loading dose of 150–300 mg orally) and Clopidogrel (Loading dose of 600 mg orally) or Ticagrelor (Loading dose of 180 mg orally) and un fractionated heparin 70–100 U/kg i.v bolus when no GPIIb/IIIa inhibitor was planned and 50–60 U/kg i.v bolus with GP IIb/IIIa inhibitors before the procedure. The decision to administer glycoprotein IIb/IIIa inhibitors was made by the interventional cardiologist. Angiographic films were reviewed and interpreted by an experienced interventional cardiologist as regard to TIMI flow before and after revascularization, no-slow reflow, contrast volume, fluoroscopy time, stenting, using DES, thrombus aspiration and use of GPIIB/IIIA receptors inhibitors upon the decision of the operator.

In hospital follow up:

Follow up of all patients included in the study as regard in hospital mortality and short term incidence of MACE (Major adverse cardiovascular events) defined as Kip *et al.,* (2008). All-cause mortality. Other major cardiovascular events, including e.g.: recurrent myocardial infarction, stent thrombosis, cardiogenic shock, ventricular arrhythmia and stroke.

Statistical analysis of the data

Data were fed to the computer and analyzed using IBM SPSS software package version 20.0. (Armonk, NY: IBM Corp) Qualitative data were described using number and percent. The Kolmogorov-Smirnov test and Shapiro-Wilk test was used to verify the normality of distribution Quantitative data

were described using range (minimum and maximum), mean, standard deviation, median and interquartile range (IQR). Significance of the obtained results was judged at the 5% level.

The used tests were

Chi-square test: For categorical variables, to compare between different groups. Fisher's Exact or Monte Carlo correction: Correction for chi-square when more than 20% of the cells have expected count less than 5. Student t-test: For normally distributed quantitative variables, to compare between two studied groups. Paired t-test: For normally distributed quantitative variables, to compare between two periods. Mann Whitney test: For not normally distributed quantitative variables, to compare between two studied groups. Logistic Regression analysis: To detect the most independent/ affecting factor for affecting CIN

3.Results

Table 3: Distribution of the studied cases according to frequency of CIN (n = 100)

Frequency of CIN	No.	%
No CIN	87	87.0
CIN	13	13.0

Table 4: Comparison between the two studied groups according to demographic data

	Total (n = 100)		No CIN (n = 87)		CIN (n = 13)		Test of Sig.	p
	No.	%	No.	%	No.	%		
Sex								
Female	28	28.0	22	25.3	6	46.2	χ^2= 2.443	^{FE}p= 0.182
Male	72	72.0	65	74.7	7	53.8		
Age (/years)								
Min. – Max.	32.0 – 70.0		32.0 – 70.0		52.0 – 70.0			
Mean ± SD.	56.10 ± 11.12		54.89 ± 11.20		64.23 ± 6.21		U= 288.50*	0.004*
Median (IQR)	58.50 (47.50 – 66.50)		58.0 (45.0 – 63.0)		66.0 (64.0 – 69.0)			

IQR: **Inter quartile range** SD: **Standard deviation** **U: Mann Whitney test**
χ^2: **Chi square test** FE**: Fisher Exact**
p: p value for comparing between the two studied groups
*: Statistically significant at $p \leq 0.05$

Table 5: Comparison between the two studied groups according to co-morbidity

	Total (n = 100)		No CIN (n = 87)		CIN (n = 13)		χ^2	p
	No.	%	No.	%	No.	%		
DM	39	39.0	28	32.2	11	84.6	13.069*	<0.001*
HTN	59	59.0	51	58.6	8	61.5	0.040	0.842
Smoking	51	51.0	48	55.2	3	23.1	4.662*	0.861
Dyslipidemia	63	63.0	52	59.8	11	84.6	2.995	FEp=0.124
Previous ischemic cardiomyopathy	6	6.0	4	4.6	2	15.4	2.333	FEp=0.041*
I. Stroke	3	3.0	1	1.1	2	15.4	7.876*	FEp=0.044*

χ^2: **Chi square test** FE**: Fisher Exact**
p: p value for comparing between the two studied groups
*: Statistically significant at $p \leq 0.05$

Table 6: Comparison between the two studied groups according to laboratory investigation

	Total (n = 100)	No CIN (n = 87)	CIN (n = 13)	T	P
RBS at admission					
Min. – Max.	90.0 – 425.0	90.0 – 270.0	260.0 – 425.0		
Mean ± SD.	196.68 ± 74.75	172.01 ± 37.70	361.77 ± 44.49	16.535*	<0.001*
Median (IQR)	182.0 (144.5 – 216.5)	172.0 (140.0 – 203.5)	376.0 (347.0 – 387.0)		
Systolic blood pressure (mmHg)					
Min. – Max.	90.0 – 200.0	90.0 – 180.0	90.0 – 200.0		
Mean ± SD.	129.30 ± 24.63	128.62 ± 22.58	133.85 ± 36.41	0.503	0.623
Median (IQR)	130.0 (110.0 – 140.0)	130.0 (110.0 – 140.0)	120.0 (110.0 – 150.0)		
Diastolic blood pressure (mmHg)					
Min. – Max.	60.0 – 120.0	60.0 – 120.0	60.0 – 100.0		
Mean ± SD.	80.30 ± 12.75	80.23 ± 12.67	80.77 ± 13.82	0.142	0.888
Median (IQR)	80.0 (70.0 – 90.0)	80.0 (70.0 – 90.0)	80.0 (70.0 – 90.0)		

IQR: **Inter quartile range** SD: **Standard deviation** **t: Student t-test**
p: p value for comparing between the two studied groups
*: Statistically significant at $p \leq 0.05$

Table 7: Comparison between the two studied groups according to Cr:

Cr	Total (n = 100)	No CIN (n = 87)	CIN (n = 13)	T	p
Before					
Min. – Max.	0.60 – 1.20	0.60 – 1.20	0.80 – 1.20		
Mean ± SD.	0.96 ± 0.17	0.95 ± 0.17	1.04 ± 0.15	1.716	0.089
Median (IQR)	0.94 (0.80 – 1.10)	0.90 (0.80 – 1.10)	1.10 (0.90 – 1.20)		
48h after					
Min. – Max.	0.70 – 2.90	0.70 – 1.40	1.70 – 2.90		
Mean ± SD.	1.21 ± 0.45	1.06 ± 0.17	2.23 ± 0.34	12.066*	<0.001*
Median (IQR)	1.10 (0.90 – 1.20)	1.10 (0.90 – 1.20)	2.20 (2.0 – 2.50)		
t_0	6.128*	9.376*	13.218*		
p_0	<0.001*	<0.001*	<0.001*		

IQR: **Inter quartile range** SD: **Standard deviation** **t: Student t-test**
t_0: Paired t-test
p: p value for comparing between the two studied groups
p_0: p value for comparing between **Before** and **After** in each group
*: Statistically significant at $p \leq 0.05$

Table 8: Comparison between the two studied groups according to Urea

Urea	Total (n = 100)	No CIN (n = 87)	CIN (n = 13)	T	P
Before					
Min. – Max.	23.0 – 70.0	23.0 – 70.0	30.0 – 55.0		
Mean ± SD.	38.50 ± 8.57	38.09 ± 8.78	41.23 ± 6.65	1.234	0.220
Median (IQR)	39.0 (33.0 – 42.0)	39.0 (30.50 – 42.0)	40.0 (37.0 – 47.0)		
48h after					
Min. – Max.	18.0 – 114.0	18.0 – 80.0	50.0 – 114.0		
Mean ± SD.	46.30 ± 18.36	41.06 ± 10.12	81.38 ± 22.64	6.327*	<0.001*
Median (IQR)	40.0 (35.0 – 50.0)	40.0 (35.0 – 45.0)	78.0 (69.0 – 96.0)		
t_0	4.958*	4.329*	6.854*		
p_0	<0.001*	<0.001*	<0.001*		

IQR: **Inter quartile range** SD: **Standard deviation** **t: Student t-test**
t_0: Paired t-test
p: p value for comparing between the two studied groups
p_0: p value for comparing between **Before and After** in each group
*: Statistically significant at $p \leq 0.05$

Table 9: Comparison between the two studied groups according to eGFR:

eGFR	Total (n = 100)	No CIN (n = 87)	CIN (n = 13)	T	P
Before					
Min. – Max.	60.0 – 114.0	60.0 – 114.0	60.0 – 87.0		
Mean ± SD.	77.72 ± 13.77	78.83 ± 13.93	63.31 ± 10.25	2.653*	0.066
Median (IQR)	73.50 (67.50 – 85.0)	74.0 (68.50 – 90.0)	65.0 (61.0 – 68.0)		
After					
Min. – Max.	29.0 – 100.0	58.0 – 100.0	29.0 – 50.0		
Mean ± SD.	68.79 ± 16.99	73.53 ± 12.36	37.08 ± 5.99	17.150*	<0.001*
Median (IQR)	67.0 (60.0 – 80.0)	70.0 (63.50 – 82.0)	37.0 (33.0 – 40.0)		
t_0	8.552	12.586*	16.351*		
p_0	<0.001*	<0.001*	<0.001*		

IQR: **Inter quartile range** SD: **Standard deviation** **t: Student t-test**
t_0: Paired t-test
p: p value for comparing between the two studied groups
p_0: p value for comparing between **Before** and **After** in each group
*: Statistically significant at $p \leq 0.05$

Table 10: Comparison between the two studied groups according to contrast dose

	Total (n = 100)	No CIN (n = 87)	CIN (n = 13)	U	P
Contrast dose (/ml)					
Min. – Max.	120.0 – 250.0	120.0 – 220.0	240.0 – 350.0		
Mean ± SD.	189.5 ± 24.64	181.7 ± 14.26	320.9 ± 11.46	2.500*	<0.001*
Median (IQR)	180.0 (180.0 – 200.0)	180.0 (180.0 – 190.0)	325.0 (320.0 – 335.0)		

IQR: **Inter quartile range** SD: **Standard deviation**
U: Mann Whitney test
p: p value for comparing between the two studied groups
*: Statistically significant at $p \leq 0.05$

Table 11: Comparison between the two studied groups according to nephrotoxic drugs, needs fluids and need dialysis

	Total (n = 100)		No CIN (n = 87)		CIN (n = 13)		χ^2	^{FE}p
	No.	%	No.	%	No.	%		
Nephrotoxic drugs (NSAIDS & diuretics)								
Absent	89	89.0	79	90.8	10	76.9	2.226	0.153
present	11	11.0	8	9.2	3	23.1	7.726	0.088
Needs fluids								
Absent	87	87.0	87	100.0	0	0.0	100.0*	<0.001*
Present	13	13.0	0	0.0	13	100.0		
Need dialysis								
Absent	98	98.0	87	100.0	11	84.6	13.658*	0.016*
Present	2	2.0	0	0.0	2	15.4		

χ^2: **Chi square test** MC: **Monte Carlo** FE**: Fisher Exact**
p: p value for comparing between the two studied groups
*: Statistically significant at $p \leq 0.05$

Table 12: Comparison between the two studied groups according to in-hospital stay

	Total (n = 100)	No CIN (n = 87)	CIN (n = 13)	U	P
In-hospital stay (/day)					
Min. – Max.	2.0 – 12.0	2.0 – 3.0	6.0 – 12.0		
Mean ± SD.	3.57 ± 1.81	2.92 ± 0.27	7.92 ± 1.71	0.000*	<0.001*
Median (IQR)	3.0 (3.0 – 3.0)	3.0 (3.0 – 3.0)	7.0 (7.0 – 8.0)		

IQR: **Inter quartile range** SD: **Standard deviation** **U: Mann Whitney test**
p: p value for comparing between the two studied groups
*: Statistically significant at $p \leq 0.05$

4. Discussion

In concordant to our study, In Kurtul *et al.,* (2017) in their study that aimed for development and validation of a pre-PCI risk model for CIN prediction and included 159 patients who developed CIN showed that those patients tend to be older with mean age (70.8 12.0) (p. value <0.001).

Similarly, In Inohara *et al.* (2015) that aimed for development and validation of a pre-PCI risk model for CIN prediction and included 358 patients who developed CIN in a COHORT study showed that those patients tend to be older with mean age 72.1±12.1.

Similarly, Chou *et al.,* (2016) reported that most of patients who had been diagnosed with CIN were more older in age compared with whom not Diagnosed with CIN with mean (67.6 ± 13.0 years), (p. value<0.001)

Also Gurm *et al.,* (2013) reported that patients diagnosed with CIN post PPCI were more older than who not diagnosed with CIN with mean age (70.3 ± 12.3 years) (P. Value<0.001)

In Andò *et al.,* (2015), the study reported that patients diagnosed with CIN post PPCI were more older than who not diagnosed with CIN with mean age (73 ± 10) (P. value < 0.001)

The present study, there is no significant correlation between sex with incidence of AKI as in group I who developed CIN after primary PCI, 7 patients were male (53.8%) while 6 patients were female (46, 2%). (P. Value=0.182)

In concordant to our study, in Andò *et al.,* (2015) the study also reported that (72 %) patients who were diagnosed with CIN post PPCI were male and 73%of patient who were not diagnosed with CIN were male with no statistical significance between two groups according to sex (P.value=0.63)

On the opposite side, in Kurtul *et al.,* (2017) in their study showed that 72 from 159 (45.3%) patients who developed CIN patients were female (P. value <0.001).

Also on the opposite side, Inohara *et al.* (2015) demonstrated CIN is commoner among female gender in their study.

The present study showed that there is statistically significant difference between the overall incidence of diabetes mellitus in the studied as in group I, 11 patients (84.6%) were found to be diabetics, (P value <0.001).

In concordant to our study, in Kurtul *et al.,* (2017) their study also demonstrated diabetes mellitus as strong independent risk predictor for CIN as 75 of 159 patients (47.2%) who developed CIN after PCI were diabetic (P. value <0.001)

In concordant to our study, in a study carried on Italian patients by Evola *et al.,* (2012) to assess risk factors of contrast induced nephropathy 42% of 105 patients who developed CIN were found diabetics with P.value 0.03 in comparison with those who did not develop AKI.

Similarly, Merihan *et al.,* (2004), their study also demonstrated diabetes mellitus as strong independent risk predictor for CIN as 19 % of 729 patients who developed CIN after PCI were diabetic, in a multivariate logistic regression model (OR;1.73- 95%confidence interval (CI) 1.48-2.02, (P. value <0.0001).

Also, in Chou *et al.,* (2016) their study also demonstrated that the DM is a strong independent risk predictor for CIN. In a logistic regression model (OR; 0.64- 95%confidence interval (CI) 3.06 (1.72-5.47), P. value=<0.001)

In Andò *et al.,* (2015) their study also, the study reported that 13 (52%) patients diagnosed with CIN post PPCI were diabetic while 130 (29%) patient not diagnosed with CIN post PPCI were diabetic (P.value=0.02)

The present study showed that there is no statistically significant difference between the two groups as regard hypertension as in group I, 8 patients (61.5%) were hypertensive and 5 patients (38.5%) were not hypertensive. (P. value 0.842)

In concordant to our study, IN Jain *et al.,* (2018) their study reported that HTN is not an independent risk factor for CIN after primary PCI.

On the opposite side, Merihan *et al.,* (2004) their study also demonstrated hyper tension as strong independent risk predictor for CIN as 15.9 % of 729 patients who developed CIN after PCI were hypertensive, in a multivariate logistic regression model (OR;1.45- 95%confidence interval (CI) 1.24-1.71, P. value<0.0001)

The present study showed that there is no statistically significant difference between the two groups as regard smoking as a risk factor of CIN after primary PCI as in group I, 3 patients (23.1%) were smokers and 10 patients (76.9%) were nonsmokers. (P. value 0.861)

In concordant to our study, in Andò *et al.,* (2015) the study also demonstrated that smoking is not an independent risk predictor for CIN (P. value=0.983)

The present study cannot find any significant correlation between dyslipidemia and incidence of CIN. (P value =0.124)

In concordant to our study, their study show that hypercholesterolemia was found to have no significant correlation with contrast induced nephropathy in their (33%) affected population (P. value = 0.62).

Similarly, In Kurtul *et al.,* (2017) their study also demonstrated that hyper cholestrolaemia was found to have no significant correlation with contrast induced nephropathy in their (26.4%) affected population (P. value = 0.755).

Also in Andò *et al.,* (2015) their study show that hyper cholestrolaemia was found to have no significant correlation with contrast induced nephropathy as 14 (56%) patients diagnosed with CIN post PPCI were dyslipidaemic while 262 (57%) patient not diagnosed with CIN post PPCI were dyslipidaemic (P. value =1.0)

The present study showed that there is statistically significant correlation between previous ischemic heart failure and CIN (P. value 0.041).

In concordant to our study, in Chou *et al.,* (2016) their study also demonstrated that the congestive heart failure is a strong independent risk predictor for CIN. In a multivariate logistic regression model (OR; 3.10- 95%confidence interval (CI) 1.58-6.10, P. value=0.001).

The present study showed that there is statistically significant difference between the two groups as regard ischaemic stroke as in group I, 2 patients (15.4%) has ischaemic stoke. (P. value =0.044)

In concordant to our study, Kurtul *et al.,* (2017)) their study demonstrated that ischaemic stroke as a risk predictor for CIN was greater (p. value=0.035)

Similarly, Merihan *et al.*, (2004) their study also demonstrated ischaemic stroke as independent risk predictor for CIN, as 11% of patients have ischaemic stroke and 18% of patient who developed CIN after primary PCI were having ischaemic stroke.in a multivariate logistic regression model (OR;1.37- 95% confidence interval (CI) 1.10-1.71, P. value=0.0007)

Disconcordant to our study, in Chou *et al.,* (2016) their study demonstrated that the ischaemic stroke is not a strong independent risk predictor for CIN. In a multivariate logistic regression model (OR; 0.64- 95%confidence interval (CI) 0.15-2.76, P. value=0.548).

The present study showed that there is strong statistically significant difference between the two groups as regard Random blood sugar at admission and demonstrated that preprocedural hyperglycemia is a strong risk factor for CIN as in group I: R.B.S at admission ranged from 260-425 with mean 361.77± 44.49. (P. value<0.001).

In concordant to our study, in Lin *et al.,* (2018) demonstrated that hyperglycemia is a strong risk factor for CIN as in their study 103 (18.5%) patients of 558 patients had preprocedural hyperglycemia and 89 (15.9%) patients developed CI-AKI. Multivariate analysis indicated that preprocedural hyperglycemia was an independent predictor of CI-AKI (odds ratio = 1.971, 95% confidence interval (CI): 1.129-3.441; P <0.05).

The present study showed that there is no correlation between systolic blood pressure (P. Value=0.623) and diastolic blood pressure (P. Value=0.888) as a risk factor and AKI post PPCI.

In concordant to our study, in Kurtul *et al.,* (2017) the study reported that there no was statistically significant correlation between systolic and diastolic blood pressure on admission and occurance of CIN post PPCI (P. Value=0.143) (P. Value=0.964) respectively.

The present study showed that there is strong statistically significant difference between the two groups as regard contrast dose and demonstrated that in cease the contrast dose is a strong risk factor for CIN as In group I: Contrast dose ranged from 240-350 ml with mean 320.9± 11.46 ml but In group II: Contrast dose ranged from 120-220 ml with mean 181.7 ± 14.26 ml (P. value<0.001).

In concordant to our study, in Merhan *et al.,* (2004) the study show that there is correlation between contrast volume and developing CIN after primary PCI, in a multivariate logistic regression model (OR;1.276- 95%confidence interval (CI) 1.197-1.360, P. value < 0.0001).

Disconcordant to our study, in Andò *et al.,* (2015) their study demonstrated that total contrast volume is not independent risk predictor for CIN its amount during PPCI did not differ between patients with or without AKI (165 ± 79 mL vs 163 ± 62 ml, respectively (P. value = 0.88).

The present study showed that There was no statistically significant difference between the two studied groups as regard nephrotoxic drugs (eg NSAIDS & diuretics) as a risk factor for CIN (P value =0.088) as In group I, 10 patients (76.9%) had no history of intake nephrotoxic drugs and 3 patients (23.1%) had history of intake nephrotoxic drugs.

In concordant to our study, in Diogo *et al.,* (2010) their study demonstrated that there was no association between the use of NSAIDs and the development of CIN with OR of 1.293 95% CI (0.46-4.2).

In the present study show that presence of multi vessle disease of the patient was not found to have any statistical significant relation with the risk of developing CIN (P value=0.052).

Des concordant to our study, in Kurtul *et al.,* (2017) the study also demonstrated the 68.8% of the patient who suffered from CIN post PPCI had multi vessle disease and there is statistical significant relation with the risk of developing AKI (P value<0.001).

In the present study show that using of GPIIbIIIa receptors inhibitors during PPCI was not found to have any statistical significant relation with the risk of developing CIN (P value=1.169).

In concordant to our study, in Kurtul *et al.,* (2017) their study also demonstrated that using of GPIIbIIIa receptors inhibitors or not during PPCI is not a strong independent risk predictor for CIN (P. Value=0.767).

In the present study show that the infarcted artery has no statistical significant relation with the risk of developing CIN (P value=0, 785).

In concordant to our study, in Kurtul *et al.,* (2017) their study also demonstrated that the infarcted artery is not a strong independent risk predictor for CIN (P. Value=0.767).

The present study show that TIMI flow post procedure was found to have statistical significant relation with the risk of developing AKI (P value=0.04).

In concordant to our study, in Andò *et al.,* (2015) the study also demonstrated that TIMI flow post procedure is a strong independent risk predictor for CIN as 19 (76%) of patient who are diagnosed with AKI had TIMI III Post procedure while 91.7% of patient who were not diagnosed with AKI had TIMI III post procedure (P. value <0.001).

In the present study show that hospital stay for the patient who were diagnosed with CIN post PPCI was more prolonged than of the patient who were not diagnosed with CIN with statistical significant relation between two study groups regarding in –hospital stay (P value<0.001).

In concordant to our study, in Andò *et al.,* (2015), their study also demonstrated that In-hospital stay for patients who were diagnosed with AKI post PPCI was 9 ± 5 days while who In-hospital stay for patients who were not diagnosed with AKI post PPCI was 7 ± 3 days (P. Value<0.001).

5. Conclusion

In conclusion, CIN after primary PCI is a common complication, and patients with older age, diabetes mellitus, previous ischemic cardiomyopathy (heart failure), previous ischemic stroke, hyperglycemia at admission are at increased risk of development of CIN. Also an increased amount of contrast during PCI procedure significantly increase the risk of post-procedure CIN.

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