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CONTRAST INDUCED NEPHROPATHY IN PATIENTS UNDERGOING PRIMARY ANGIOPLASTY FOR ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION

Cardiology

Dr. Prashanth DC* Assistant Professor, Dept Of Cardiology, Raichur Institute Of Medical Sciences, Raichur, Karnataka, India. *Corresponding Author

Dr. Shankar Gadwal Assistant Professor, Dept Of Nephrology, Raichur Institute Of Medical Sciences, Raichur, Karnataka, India.

Dr. Tom Devasia Professor, Dept Of Cardiology, Kasturba Medical College, Manipal, Karnataka, India.

Dr. Ganesh P Associate Professor Dept Of Cardiology, Kasturba Medical College Manipal, Karnataka, India.

Dr. Prasad Shetty PhD Research Scholar Kasturba Medical College Manipal, Karnataka, India.

ABSTRACT

Background: Contrast induced nephropathy (CIN) known to be associated with higher morbidity and mortality after percutaneous coronary intervention (PCI). Patients undergoing Primary PCI for acute ST elevation myocardial infarction (STEMI) may be at higher risk of CIN because of emergency nature of the procedure and unfeasibility of sufficient CIN prophylaxis and also hemodynamic instability in acute myocardial infarction (AMI) patients, making it essential to study CIN in patients undergoing Primary PCI. **Results:** Contrast induced nephropathy (CIN) occurred in 34(19.3%) patients. Patients with CIN as compaired to No-CIN patients more frequently presented with anterior wall MI [26(68.4) v/s 73(45.9) p 0.037] in addition they had higher incidence of pulmonary oedema [21(55.3) v/s 43(27) p 0.002], Shock 8(21.1) v/s 3(1.9) p 0.0005]. Left ventricular dysfunction & LVEF <40% were higher in CIN patients [15(39.5) v/s 25(15.7) p 0.003]. Baseline Nt-ProBNP [4257± 5649 v/s 1258±5649 p 0.001] and Trop T [2.22±2.59 v/s 0.81±2.59 p 0.002] were higher at presentation. In-hospital adverse events were higher patients with CIN patients. In-hospital mortality was high [4(10.5%) v/s 2(1.3%) p 0.013], length of hospital stay was higher 6.6 days vs 4.8 days. Dialysis was required in 11(32%) patients with CIN. There was a more complicated in-hospital course in CIN patients with higher incidence of heart failure[21(55.3%) v/s 43(27%) p 0.002] requiring ventilator/NIV support, atrial fibrillation [7(18.4%) v/s 8(5.1) p 0.005], serious ventricular arrhythmias[7(18.4%) v/s 11(6.9%) p 0.027]. **Conclusion:** Contrast-induced nephropathy is a frequent and life threatening complication in patients with STEMI undergoing primary PCI. Patients with CIN had more complicated in-hospital course, longer hospitalization & higher In-hospital mortality.

KEYWORDS

Myocardial Infarction, Primary PCI, Contrast induced nephropathy.

BACKGROUND:

Contrast induced nephropathy (CIN) is an iatrogenic kidney injury that follows intravascular administration of radiopaque contrast medium (CM). Contrast induced nephropathy is a well known complication following exposure to contrast medium (CM) during diagnostic or therapeutic procedure. CIN is defined as an relative 25% increase in serum creatinine (SCr), or an absolute increase of 0.5 mg/dl from baseline SCr, within 48-72 hours of contrast exposure [1-4]. CIN is one of the leading cause of iatrogenic kidney injury and is associated with significant morbidity and mortality[5-7]. Several risk factors for CIN have been identified old age, diabetes, hypotension/shock, heart failure, pre-existing renal dysfunction are some of the well known risk factors [8]

Current evidence suggest that primary PCI achieving rapid restoration of coronary artery patency which helps in preserving ventricular function and thus survival in patients presenting with STEMI [9-10]. However patients treated via primary PCI may be at increased risk of CIN because of emergency nature of the procedure, hemodynamic instability such as hypotension/shock or pulmonary oedema, unfeasibility of sufficient CIN prophylaxix and use of large volume of CM during primary PCI [9-10], Which makes it essential to study CIN in STEMI patients undergoing primary PCI.

There are very few studies assessing kidney function in setting of primary PCI and examining impact of CIN on clinical outcome. The purpose of this study was to determine incidence of CIN, clinical predictors and clinical outcome of CIN after primary PCI.

METHODS

Study Population And Design

This is a single-centre, prospective, observational study. Of a total 204 consecutive patients admitted with STEMI between may 2018 to august 2018, 7 were excluded (6 patients expired within 48 hours & 1 patient underwent emergency CABG). Hence a total of 197 STEMI patients undergoing primary PCI were included in the study, at a tertiary care hospital in South India. Approval for the study protocol was obtained from the institutional ethics committee & informed consent was obtained from all patients.

According to our institute protocol, patients were taken for primary PCI if they presented within 12 h (18 h for AMI complicated by cardiogenic shock) from the onset of symptoms, characteristic chest pain lasting for at least 30 min, not responsive to nitrates, with electrocardiographic ST-segment elevation of at least 0.2 mV in two or more contiguous leads, or left bundlebranch block). Patients were excluded from the study if coronary anatomy was not suitable for PCI or if emergency bypass grafting was required or patient expired within 48hrs post CM exposure. Patients on chronic peritoneal or hemodialytic treatment were also excluded.

Study Protocol:

Clinical details and risk factors for coronary artery disease were noted at presentation. All patients were given loading doses of aspirin, ticagrelor, atorvastatin at presentation and unfractionated heparin during primary PCI. The procedure was done by on-duty cardiology team, isosmolar nonionic contrast medium was used in all cases (Iodixanol), physiologic (0.9%) saline, and N acetyl cysteine was administered after contrast exposure. The use of radial/femoral approach for PCI, use of drugs like betaadrenergic blocking agents, angiotensin-converting enzyme inhibitors, platelet glycoprotein IIb/IIIa receptor inhibitors, vasodilators, diuretics, or the indication to intraaortic balloon pump or inotropic drugs support, were left to the discretion of the treating cardiologists. Patients were then observed for in-hospital events and outcome.

Serum creatinine, Haemoglobin, Hematocrit, measured at the time of admission (just before primary PCI) and 48-72 hr after primary PCI. Creatinine clearance was calculated by applying the Cockcroft-Gault formula. An echocardiographic evaluation was done at the time of admission.

During hospitalization the following adverse clinical events were recorded acute pulmonary edema and requirement of NIV/intubation, tachyarrhythmia, bradyarrhythmias, renal failure requiring emergency renal replacement therapy (hemofiltration or hemodialysis), major bleeding requiring blood transfusion, stroke, duration of hospital stay

and death.

Definitions

Contrast-induced nephropathy defined as an relative 25% increase in serum creatinine (SCr), or an absolute increase of 0.5 mg/dl from baseline SCr, within 48-72 hours of contrast exposure without alternative explanation, is the most commonly used definition of CIN [4]. Cardiogenic shock was defined as marked and persistent (> 30 min) hypotension with systolic pressure less than 80 mmHg with signs of hypoperfusion caused by severe left ventricular dysfunction, right ventricular infarction, or mechanical compliocations of infraction and not due to hypovolemia, bradyarrhythmias or tachyarrhythmias. Hypertension (HT) was defined as a history of HT and/or use of antihypertensive drugs, and DM was defined as a history of DM and/or use of antidiabetic drugs before the hospital admission. Positive family history for coronary artery disease (CAD) was defined as documented evidence of CAD in a parent or sibling before 60 years of age. Blood transfusion was initiated in case of hemoglobin reduction below 8.0 g/l.

Statistical Analysis:

The collected data were analysed with IBM. SPSS statistics software 23.0 Version.To describe about the data descriptive statistics frequency analysis, percentage analysis were used for categorical variables and the mean & S.D were used for continuous variables. To find the significant difference between the bivariate samples in Independent groups the Unpaired sample t-test was used.To fit the model to identify the influencing factors for the CIN the Binary Logistics Regression with Enter method was used.To find the significance in categorical data Chi-Square test was used similarly if the expected cell frequency is less than 5 in 2×2 tables then the Fisher's Exact was used. In all the above statistical tools the probability value .05 is considered as significant level.

Baseline Characteristics

Baseline characteristics	No-CIN	CIN	p-value
Age(yrs)	62±12	64±13	0.245 #
Gender			
Male	120(75.5)	29(76.3)	1.000 #
Female	39(24.5)	9(23.7)	
Type of MI			
AWMI	74(46.5)	25(65.8)	0.086 #
IWMI	67(42.1)	8(21.1)	
IPWMI	16(10.1)	5(13.2)	
IWMI+RVMI	2(1.3)	0	
BMI	22.9±3.4	22.8±4.0	0.847 #
Underweight	16(10.1)	5(13.2)	0.834 #
Normal Weight	92(57.9)	22(57.9)	
Overweight	49(30.8)	10(26.3)	
Obese	2(1.3)	1(2.6)	
'0'Hr Creatinine	1.04±0.43	1.02±0.29	0.872 #
Creatinine >1.5	11(6.9)	1(2.6)	0.324#
Pulse	83.6±60.9	84.4±21.3	0.938 #
Systolic BP(mmHg)	123±26.9	114.2±22.3	0.062 #
Diastolic BP(mmHg)	75.7±12.1	73.3±13.2	0.284 #
Mean LVEF(%)	49.1±8.4	43.8±7.6	0.0005**
Nt-Pro BNP	1258.3±959.5	4527±5649	0.001**
Trop T	0.81±1.6	2.22±2.59	0.002**
HbA1C	6.61±1.66	6.64±1.54	0.913 #
eGFR	71.1±26.9	68.5±28.9	0.596 #
Hb% at admission	13.7±2.2	13.2±2.1	0.232 #
Hb% post procedure	12.6±2.3	11.9±2.5	0.146 #
Dyslipidemia	19(11.9)	4(10.5)	1.000 #
Diabetes	55(34.6)	14(36.8)	0.7#
Hypertension	73(49.5)	18(47.4)	0.8#
Smoking/Tobacco use	51(35.1)	11(28.9)	0.7#
Past H/O IHD	5(3.1)	1(2.6)	0.8#
Pulmonary Edema at admission	43(27)	21(55.3)	0.002 **
Killip class II	28(17.6)	8(21.1)	0.0005 **
Killip class III	14(8.8)	13(34.2)	
Killip class IV	1(0.6)	4(10.5)	
**** Highly Sig at p < 0.01 and # No Sig. At p > 0.05**			

Procedural Characteristics

Procedural characteristics	NO CIN	CIN	p value
Culprit vessel			
LAD	73(45.9)	26(68.4)	0.037 *
RCA	66(41.5)	8(21.1)	
LCX	20(12.6)	4(10.5)	
Dominant vessel			
RCA	148(93.1)	34(89.5)	0.217 #
LCX	7(4.4)	4(10.5)	
Co-Dominant	4(2.5)	0	
Number of Diseased vessel			
SVD	76(47.8)	17(44.7)	0.287 #
DVD	58(36.5)	11(28.9)	
TVD	25(15.7)	10(26.3)	
Number of stents			0.916 #
1 stent	130(81.8)	31(81.6)	
2stents	27(17)	7(18.4)	
3stents	1(0.6)	0	
POBA	1(0.6)	0	
Number of vessel revascularised			
1	143(89.9)	33(86.8)	0.564 #
2	16(10.1)	5(13.2)	
LVEF			
<40%	25(15.7)	15(39.5)	0.003 **
>40%	134(84.3)	23(60.5)	
Rv dysfunction			
Absent	134(84.3)	31(81.6)	0.685 #
Present	25(15.7)	7(18.4)	
Contrast Volume(ml)			
Mean Contrast volume	122.4±32.3	132.5±34.3	0.088#
<100ml	43(27)	7(18.4)	0.307 #
>100ml	116(73)	31(81.6)	
**** Highly Sig at p < 0.01,* Sig at p < 0.05 and # No Sig. At p > 0.05**			

RESULTS:

Incidence of CIN & baseline characteristics:

A total of 197 consecutive STEMI patients fulfilling criteria for primary PCI were included in the study. Mean (±SD) age of the patients was 62.26(±12.1) years and 149(75.6%) were males 48(24.4%) were females. Incidence of CIN was 19.3% (n=34) and dialysis was required in 32% (n=11) patients with CIN.

When comparing patients with and without CIN, patients with CIN more frequently presented with anterior wall MI [26(68.4) v/s 73(45.9) p 0.037] in addition they had higher incidence of pulmonary oedema, Killip Class II & above [21(55.3) v/s 43(27) p 0.002], Shock 8(21.1) v/s 3(1.9) p 0.0005]. Left ventricular dysfunction & LVEF <40% were higher in CIN patients [15(39.5) v/s 25(15.7) p 0.003]. Baseline Nt-ProBNP [4257± 5649 v/s 1258±5649 p 0.001] and Trop T [2.22±2.59 v/s 0.81±2.59 p 0.002] were higher at presentation.

There was no statistically significant difference in baseline creatinine 1.04±0.43 versus 1.02±0.29 (p 0.84) and contrast volume used during the procedure 132ml versus 122 ml (p 0.088) [Table 1].

There was no significant association was found between number of diseased vessels, and number of vessels revascularised in the same sitting presented in. The other comparisons of demographic, clinical and procedural characteristics between patients with and without CIN did not differ [Table 2].

In-Hospital Events

In hospital events	No CIN	CIN	p value
Pulmonary Edema	43(27)	21(51.3)	0.001**
Ventilator/NIV Support	36(22.6)	19(50)	0.001**
Shock	3(1.9)	8(21.1)	0.001**
AKI Requiring Dialysis	3(1.9)	11(28.9)	0.001**
IABP	11(6.9)	10(26.3)	0.001**
Stroke	2(1.3)	1(2.6)	0.5#
Blood Loss Requiring Transfusion	7(4.4)	5(13.2)	0.058 #
CHB	6(3.8)	2(5.3)	0.676#
AF	8(5.1)	7(18.4)	0.027#
VT	11(6.9)	7(18.4)	0.027#
AIVR	20(12.6)	5(13.2)	0.923#

In Hospital Mortality	2(1.3)	4(10.5)	0.013 *
**** Highly Sig at p < 0.01,* Sig at p < 0.05 and # No Sig. At p > 0.05**			

In-hospital adverse events were higher patients who developed CIN. In-hospital mortality was high [4(10.5%) v/s 2(1.3%) p 0.013], length of hospital stay was higher 6.6 days vs 4.8 days... Dialysis was required in 11(32%) patients with CIN. There was a more complicated in-hospital course in CIN patients with higher incidence of heart failure[21(55.3%) v/s 43(27%) p 0.002] requiring ventilator/NIV support, atrial fibrillation [7(18.4%) v/s 8(5.1) p 0.005], serious ventricular arrhythmias[7(18.4%) v/s 11(6.9%) p 0.027]. CIN incidence was higher in patients who needed IABP support [11(28%) v/s 3(1.9%) p 0.001] [Table 3].

DISCUSSION:

This study examining the incidence and impact of CIN in patients undergoing primary PCI for STEMI demonstrated that CIN is observed frequently after primary PCI, even in patients with normal renal function, and it is associated with higher In-hospital adverse events, higher In-hospital mortality & prolonged hospitalization.

The reported incidence of CIN varies widely across the literature, depending on the patient population and the baseline risk factors. It's reported incidence ranges from 8 to 25% in the general population [11-12]. In our study the incidence of CIN was 19.3% and dialysis was required in 11 patients with CIN.

Advances in medical sciences & emerging evidence suggest that primary PCI is the most effective treatment strategy of STEMI. Many studies have demonstrated that ventricular function is preserved and survival was better when STEMI patients undergo primary PCI with aim of achieving patency of infract related artery as early as possible [9,10]. In our study population, the in-hospital mortality rate was 10.5% in CIN patients & 1.2% in No CIN patients, a value comparable with those reported in other clinical trials [5,10]. We found that, most of the patients developing CIN presented with AWMI, larger infarct size, and were more likely to have a reduced left ventricular systolic function and in pulmonary oedema, hypotension/shock. CIN incidence was higher in patients who needed IABP support, as the patients requiring IABP were more critical with reduced left ventricular function and most of them were in cardiogenic shock. Interestingly risk of CIN was higher in patients who had VT or AF during hospitalization. One of the key modifiable risk factor for CIN is the volume of contrast medium used during primary PCI. Cigarroaet al [13]have suggested a formula for maximum allowable contrast dose (5 × body weight/SCr). Our study demonstrated that low volume of contrast infused during primary PCI can be a handy modifiable CIN risk factor which can limit the development of CIN, average contrast volume use in our study was 132.5±34.3 in CIN patients V/S 122.4±32.3 ml (p 0.088) in No-CIN patients. There was no significant association was found with CIN between number of diseased vessels, and number of vessels revascularised in the same sitting.

In-hospital adverse events were higher patients who developed CIN. In-hospital mortality was high, length of hospital stay was higher. There was a more complicated in-hospital course in CIN patients with higher incidence of heart failure requiring ventilator/NIV support, atrial fibrillation, serious ventricular arrhythmias. In our study it was not possible to identify the variables that traditionally increase the risk of CIN onset; they were older age, higher baseline creatinine & diabetes. In traditional risk scores for CIN, such as Mehran [14] and Bartholomew [15] these variables generate points in their respective forecast ranges of risk categorization.

All patients with acute myocardial infarction undergoing primary PCI may be at a higher risk for developing CIN. The most effective preventive measure, which is hydration prior to use of contrast is difficult to perform due to the emergency nature of the procedure. Innovative preventive strategies aimed at protecting the kidneys from contrast toxicity and ischemic burden during the acute phase of infarction need to be developed and tested, even in patients with normal renal function.

N-acetylcysteine, despite being studied for a long time, data is still conflicting as to its power to prevent CIN after coronary angiography studies[16]. Pentoxifylline was not protective for the occurrence of CIN in low-risk samples (elective coronary angiography) [17-18] we are still missing data on patients undergoing emergency coronary angiography. In a recent meta-analysis, intravenous fenoldopam was not superior to saline or N-acetylcysteine[19]. Rosuvastatin and atorvastatin are promising drugs that have proven effective in preventing CIN in a meta-analysis based on randomized clinical trials[20]. Newer pharmacologic [21] or nonpharmacologic [22-23] strategies that showed a benefit in terms of CIN prevention among patients undergoing elective PCI should be evaluated also in high-risk AMI patients treated with primary PCI.

Study Limitations.

Some limitations of our study were, first, our study included a small population, admitted to a single centre. Second, there are creatinine limitations in the context of acute renal injury. Currently, studies using biomarkers point to the role of NGAL in both the risk of detecting (by the dosage prior to the use of contrast) as in the early diagnosis (by measuring it one day after the use of contrast) CIN. Third, although all our patients effectively underwent contrast media exposure and 19.3% developed renal dysfunction within 48 h to 72 h after it, we cannot exclude the possibility that other factors, such as hemodynamic instability, might have contributed, at least in part, to renal impairment, and influenced the clinical outcome of our patients.

CONCLUSION.

In the era of primary PCI for AMI, CIN is a frequent complication, even in patients with normal renal function, and is associated with a more complicated in-hospital course and high mortality rate. Thus, newer preventive strategies of renal protection during primary PCI are warranted. It is meaningful to make all attempts at the time of intervention to prevent CIN in high-risk patients, including adequate hydration, minimization of contrast use, use of non ionic low osmolar or iso osmolar contrast agent.

List Of Abbreviations:

AMI: Acute myocardial infarction
CABG: Coronary-artery bypass grafting
CAD: Coronary artery disease
CIN: Contrast induced nephropathy
CM: Contrast medium
DM: Diabetes mellitus
HTN: Hypertension
NIV: Non invasive ventilation
PCI: Percutaneous coronary intervention
SCr: Serum Creatinine
STEMI: ST Elevation myocardial infarction

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Availability Of Data And Materials

The dataset supporting the results and conclusions of this article will be available from the corresponding author on request.

Ethics Approval And Consent To Participate

The study protocol was approved by the Kasturba Medial College and Kasturba Hospital Institutional Ethics Committee (IEC number: 291/2018).

Consent to participate: Taken.

Consent For Publication

Not applicable.

Competing Interests

The authors declare that they have no competing interests.

Authors' Contributions

Research idea, study design and statistical analysis were done by DCP and TD.

Data collection was done by DCP. Data analysis and interpretation was carried out by all authors. The article was written predominantly by DCP. All authors read and approved the final manuscript.

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