Original Article

Journal of Cardiovascular Medicine and Surgery
Volume 6 Number 2, April – June 2020
DOI: http://dx.doi.org/10.21088/jcms.2454.7123.6220.6

Incidence and Predictors of Contrast Induced Nephropathy after Percutaneous Coronary Intervention at a Tertiary Care Hospital in Western India

Cinosh Mathew[1], Rahul Jain[2], Nishant Saxena[3], Jayesh Rawal[4]

Authors Affiliation: [1] Associate professor, [2–3] Senior Resident, [4] Professor, Department of Cardiology, SBKS Medical Institute and Research Centre, Sumandeep Vidyapeeth University, Vadodara, Gujarat 391760, India.

Abstract

Aims: This observational study was undertaken to identify the incidence of contrast induced nephropathy and the predictors for development of Contrast induced Nephropathy after Coronary Angioplasty in a tertiary care institute. *Materials and Methods:* The study consisted of 520 patients who underwent Percutaneous Coronary Angioplasty at our institute during one year duration and who satisfied the inclusion criteria were enrolled in the study. Renal parameters such as serum Creatinine and Blood Urea level was measured at baseline and at 24 hours and 48 hours post procedure. Contrast induced nephropathy was defined as an increase of >25% or >0.5 mg/dl in pre-catheterization serum creatinine at or after 48h after percutaneous coronary intervention. Standard definitions were used to define the variables. *Results:* 67 patients (12.88%) developed Contrast induced Nephropathy post PCI in the study. Elderly population, presence of diabetes mellitus, hypertension, anemia were associated with the risk of developing CIN and found to be statistically significant. Increased Contrast volume and raised baseline creatinine also were also found to be associated with CIN. *Conclusions:* Development of CIN is a known complication of PCI and can be mostly prevented if patients with risk factors are identified and preventive measures taken based on guideline based recommendations.

Keywords: Contrast induced nephropathy; Percutaneous coronary intervention; Contrast media.

How to cite this article:
Cinosh Mathew, Rahul Jain, Nishant Saxena, et al. Incidence and Predictors of Contrast Induced Nephropathy after Percutaneous Coronary Intervention at a Tertiary Care Hospital in Western India. J Cardiovasc Med Surg 2020;6(2):127-131.

Introduction

Contrast media is been increasingly used for diagnostic and therapeutic interventions for structural delineations. The use of intravascular contrast media has revolutionized the field of interventional cardiology with increasing number of procedures being now been done by percutaneous route. One of the serious adverse effect of intravenous contrast media is Contrast induced nephropathy(CIN).[1,2] CIN is defined as impairment of renal function (measured as either a 25% increase in serum creatinine from baseline or a 0.5 mg/dL increase in absolute serum creatinine value) within 48–72h of administration of contrast material.[1,2] Various studies have reported the incidence of CIN to be between 2% and 30%, depending on the definition of CIN.[2–4] However, it has been associated with worsening outcome causing long term morbidity and increased mortality, prolonged hospitalization, requirement for dialysis adding to increasing financial burden on health resources.[6]

The incidence of CIN in high-risk patients undergoing PCI has been reported to vary from 10% to 20%.[7] In India more than 3.5 lac PCI are being done every year;[8] the number of percutaneous

Corresponding Author: Rahul Jain, Senior Resident Department of Cardiology, SBKS Medical Institute and Research Centre, Sumandeep Vidyapeeth University, Vadodara, Gujarat 391760, India.

Email: cardiology.sbks@gmail.com

Coronary Intervention has been increasing every year and more complex cases are now being treated with PCI. This study was conducted to detect the incidence of CIN post PCI and to identify the risk factors for developing CIN in patients attending a tertiary care institute in western India.

Materials and Methods

Study population

A total of 636 patients who underwent PCI from January 2017 to December 2018 were evaluated. One hundred and sixteen patients were excluded from the study due either due acute renal failure, preexisting severe CKD on hemodialysis or other exclusion criteria of hypotension or nonavailability of follow up data of serum creatinine. Therefore, a total of 520 patients who underwent elective or emergency coronary interventional procedure were evaluated in our study. All patients received evidence based standard pre and post procedural care as per standard guidelines. Institutional ethics committee approval was obtained prior to the study and informed written consent for PCI and for the CIN study was taken from all patients.

Study Protocol

Radial route was the most commonly used vascular access technique. Patients received a bolus 100 IU/kg of heparin before the PCI procedure started and further anticoagulation was given as warranted based on the Activated Clotting Time (ACT). Standard care like appropriate hydration pre and post procedure, withholding nephrotoxic drugs were taken as per guidelines. CAG was performed in the Cardiac Catheterization Laboratory by Experienced Interventional Cardiologist using the Philips FD10 equipment. Tri-iodinated nonionic water-soluble radiocontrast (Omnipaque™, IOHEXOL, GE Healthcare) was used with iodine content is 350 mg/L, osmolality is 780 mOsm/kg H_2O) The absolute volume of contrast media was recorded for each PCI. Laboratory investigations including pre- and postprocedural serum creatinine at 24, 48 and 72 hours post procedure, blood glucose, and baseline hemoglobin were collected. Serum creatinine was measured by Jaffe's reaction method in automatic biochemistry analyzer. If a patient developed CIN, patient was treated by medical management or by dialysis based on consultation with nephrologist. Contrast-induced nephropathy was defined as an increase of >25% or >0.5 mg/dl in pre-PCI serum creatinine at or after 48 h after PCI according to the Acute Kidney Injury Network (AKIN) definition.[1,2] Myocardial infarction, hypertension, hypotension, diabetes, Chronic kidney disease, and cardiogenic shock, were defined as per standard definitions.

Results and Analysis

Out of 520 patients who were evaluated in the study 67(12.88%) developed Contrast induced Nephropathy. Baseline demographics of the study population who developed CIN vs. those who did not develop CIN are presented in Table 1 and clinical characteristics of both are presented in Table 2.

Table 1: Baseline demographics and clinical characteristics.

Characteristics	CIN group(n=67)	No CIN group(n=453)	P value (significant if <0.05)
Age(in years)	61±17	63±19	0.415
Elderly (>70 yrs age)	14 (21%)	36(8%)	<0.001
Male sex	45(67.16%)	280(61.81%)	0.399
Smoker	28(41.79%)	240(52.98%)	0.087

Table 2: Evaluation of Risk factors and predictors for development of CIN.

Characteristics	CIN group(n=67)	No CIN group(n=453)	P value (significant if <0.05)
Diabetes Mellitus	52(77.61%)	223(49.22%)	<0.001
Hypertension	44(65.67%)	253(55.84%)	0.095
Dyslipidemia	48(71.64%)	265(58.49%)	0.04
Anemia	48(71.64%)	236(52.09%)	0.002
LV Dysfunction	31(46.26%)	185(40.83%)	0.4
STEMI	18(26.86%)	108(23.84)	0.38
NSTEMI/Unstable Angina	49(73.13%)	345(76.15%)	0.59
Multivessel PCI	47(70.14%)	35(52.23)	0.006
S. Creatnine	1.34(±0.16)	0.86(±0.11)	<0.001
Contrast volume	178(±42)	142(±38)	<0.001

Total of 50 patients >70 yrs were enrolled in the study. 14 patients among them developed CIN 28% vs 11.27% in patients less than 70 yrs of age, which was significant at p <0.001. (Table 1)

No statistical significance was found to be related to gender (p=0.399) and smoking status (p=0.087).

We evaluated the presence of CIN in presence and absence of established risk factors for the same (Table 2). Among 275 patients with Diabetes mellitus 52 developed CIN, 18.90% vs 6.12% in non diabetic group, p<0.001 which was statistically significant.

Total number of hypertensive patients were 297 out of which 45 developed CIN 15.15% vs 9.86 among non hypertensives, p 0.07 which was not statistically significant. The nature of ACS (STEMI, NSTEMI or UA) was not associated with development of CIN. Among 284 patients with anemia 48 developed CIN 16.9% *vs* 8.05 in non anemic group p = 0.002 which was highly significant. No statistically significant difference was found in patients with dyslipidemia and LV dysfunction with respect to developing CIN. The baseline serum creatinine in patients who developed CIN was raised 1.34±0.16 vs 0.86±0.11, p <0.001 which was highly significant. Also volume of contrast used and a Multivessel PCI procedure was a risk factor for development of CIN which was statistically significant between both the groups. Volume of contrast used in patients who developed CIN was mean of 170 ml compared to 140 ml in those who didn't develop CIN(p<0.001)

Discussion

Contrast induced nephropathy is the third leading cause of renal dysfunction and is associated with increased morbidity, mortality and increased duration of hospitalization.[9] Also it is a major complication faced after coronary intervention procedures and its incidence is on the rise due to increasing number of complex coronary interventions being performed each year due to associated comorbidites and more volume of contrast being used with these complex procedures.

The incidence of contrast induced nephropathy in our study was 12.88% which was consistent with earlier studies showing an incidence of 10-20%.[6,10] Compared to some previous studies where the incidence of CIN was upto 20%[11] in our study it was not as high as patients with conditions like cardiogenic shock and CKD were excluded who already are at higher risk of developing CIN. Female gender was shown to be more at risk for developing CIN in study by Iakovou et al.[12] but in our study there was no statistically significant difference related to gender with respect to development of CIN.

Only 1 patient (0.19%) developed CIN severe enough to undergo renal replacement therapy which was much lower as compared to previous studies by Mokadam et al.[13] and King SB et al.[14] which showed 0.3–4% patients needing hemodialysis, this could be related to the exclusion criteria of removing patients with CKD, hypotension, and appropriate measures like pre procedural hydration, avoiding nephrotoxic drugs etc. Elderly population have been considered to be more at risk for developing CIN in prev studies[7,15,16] and similar outcomes were noted in our study, with 28% of those above 70 years of age developing CIN(p<0.001). Increased incidence of CIN in elderly may be due to reduction in GFR as well as Multivessel disease, increased contrast volume needed and other existing comorbidities.[17]

Diabetes Mellitus has been found to be a risk factor for developing CIN in studies by Lautin et al.[18] and Mc Collough et al.[19] and in our study similar result was found, incidence of CIN in diabetics was 18.90% vs 6.12% in non diabetic group, p<0.001. Nikolsky et al.[11] reported that lower baseline hematocrit was an independent predictor of contrast-induced nephropathy, each 3% decrease in baseline hematocrit resulted in significant increase in the odds of contrast-induced nephropathy in patients with and without chronic kidney disease. Among 284 patients with anemia 48 developed CIN 16.9% vs 8.05 in non anemic group p = 0.002 which was highly significant and correlated with earlier study results. Baseline serum creatinine was found to be raised in patients who developed CIN which co related with previous studies by Davidson et al. and McCullough et al.[19,20] In a series of 7,586 patients undergoing cardiac catheterization, Rihal et al.[21] found a low risk (2.4%) of CIN in patients with normal renal function, but a high risk (30.6%) in those with serum creatinine levels ≥ 3.0 mg/dl. Volume of contrast used and Multivesssel PCI was found to be associated with development of CIN which correlated with previous studies by McCullough et al.[19] found that 100 ml contrast medium was the cutoff dose below which there was no CIN requiring dialysis undergoing coronary angiography. Hence limiting the amount of contrast used will help in preventing incidence of CIN. Previous studies have shown Hypertension to be a risk factor for developing CIN[22–24] but in our study incidence of CIN was not found to be statistically higher in

hypertensives (p=0.09). Previous studies have shown dyslipidemia to be associated with CIN and study by had shown LV dysfunction to be associated with CIN but in our study they were not found to be statistically significant.

Because CIN is mostly a preventable complication it is important to identify the risk factors and predictors for development of CIN. Several risk prediction models have been previously proposed. The Mehran scoring system requires clinical, laboratory and procedural data. We tried to correlate our data to established risk factors to evaluate the risk predictors at our centre. Identification of the risk factors for the CIN in our study will help to identify risk group for development of CIN. Prevention of CIN would help to reduce the mortality, duration of hospitalization and need for renal replacement therapy.

Study Limitations

As per definition of CIN the rise in creatinine would occur by 48–72 hours which was also measured in this study. However, late rise in creatinine due to CIN could not be ruled out and some patients of renal dysfunction may be missed in the study. A long term follow up of patients would be needed to see the late consequences of CIN which was not done in this study so, the over all effect of CIN on morbitdity and mortality was not studied. Patients with preexisting CKD were not included in the study so the effect of contrast media on worsening of renal function couldnot be studied. Other reasons could also contribute to renal dysfunction like ischemia, hypotension atheroembolism which could not be differentiated and all the patients who developed CIN may not be due to contrast media alone. The definition of CIN while being based on creatinine value, creatinine levels are not an accurate indicator for renal dysfunction. Cystatin C is a more accurate marker for GFR[25] and for evaluating renal injury following contrast administration which was not measured here.

Conclusions

Development of CIN is a known complication of PCI and can be mostly prevented if patients with risk factors are identified and treated based on guideline based recommendations. Elderly age, patients with preexisting renal dysfunction, diabetes mellitus, anemia are at increased risk for developing CIN and should be treated with measures like pre hydration and limiting volume of contrast to as low as possible.

References

1. Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group. KDIGO clinical practice guideline for acute kidney injury. Kidney Int Suppl 2012;2:1–138.
2. Murphy SW, Barrett BJ, Parfrey PS, et al. Contrast nephropathy. J Am Soc Nephrol 2000;11:177–82.
3. Nough H, Eghbal F, Soltani M, et al. Incidence and main determinants of contrast–induced nephropathy following coronary angiography or subsequent balloon angioplasty. Cardiorenal Med 2013;3:128–35.
4. McCullough PA, Wolyn R, Rocher LL, et al. Acute renal failure after coronary intervention: Incidence, risk factors, and relationship to mortality. Am J Med 1997;103:368–75.
5. Diaz–Buxo JA, Wagoner RD, Hattery RR, et al. Acute renal failure after excretory urography in diabetic patients. Ann Intern Med 1975;83:155–158.
6. Levy EM, Visoli CM, Horwitz RL, et al. The effect of acute renal failure on mortality. A cohort analysis. J A M A 1996;275:1489–1494. http://dx.doi. org/10.1001/jama.1996.03530430033035
7. Dangas G, Iakovou I, Nikolsky E, et al. Contrast–induced nephropathy after percutaneous coronary interventions in relation to chronic kidney disease and hemodynamic variables. Am J Cardiol 2005;95:13–9.
8. Arramraju SK, Koganti S, Janapati R, et al. The report on the Indian Coronary Intervention data for the year 2017– National Intervention Council. Indian Heart Journal 71(2019) 146–148.
9. Barrett BJ, Parfrey PS. Clinical practice Preventing nephropathy induced by contrast medium. N Engl J Med 2006;354(4):379–86.
10. Jorgensen AL. Contrast–induced nephropathy: pathophysiology and preventive strategies. Crit Care Nurse 2013;33(1):37–46.
11. Marenzi G, Lauri G, Assanelli E, et al. Contrast–induced nephropathy in patients undergoing primary angioplasty for acute myocardial infarction. Journal of the American College of Cardiology 2004;44(9):1780–1785.
12. Iakovou I, Dangas G, Mehran R, et al. Impact of gender on the incidence and outcome of contrast-induced nephropathy after percutaneous coronary intervention. J Invasive Cardiol 2003;15:18–22.
13. Mokadam NA, Melford RE, Maynard C et al. Prevalence and procedural outcomes of percutaneous coronary intervention and coronary artery bypass grafting in patients with diabetes and multivessel coronary artery disease. J Card Surg 2011;26:1–8. 10.1111/j.1540–8191.2010.01072.x

14. King SB III, Douglas JS. Indications for percutaneous transluminal coronary angioplasty and atherectomy. Hurst's: The Heart. Schlant RC, Alexander RW (ed): McGraw–Hill, Inc, New York 1994;8:1339–1143.
15. SL Chen, J Zhang, F Yei, et al. "Clinical outcomes of contrast–induced nephropathy in patients undergoing percutaneous coronary intervention: a prospective, multicenter, randomized study to analyze the effect of hydration and acetylcysteine," International Journal of Cardiology 2008;126(3): 407–413.
16. MJ Gussenhoven, J Ravensbergen, JH van Bockel, et al. "Renal dysfunction after angiography; A risk factor analysis in patients with peripheral vascular disease." Journal of Cardiovascular Surgery 1991;32:81.
17. Contrast–Induced Nephropathy in Patients Undergoing Percutaneous Coronary Intervention Sana Shoukat, Saqib A. Gowani, Asif Jafferani, Sajid H. Dhakam Cardiol Res Pract 2010;2010: 649164.
18. Lautin EM, Freeman NJ, Choenfeld AH, et al. Radiocontrast associated renal dysfunction: incidence and risk factors. AJR Am J Roentgend 1991;157:49–58. http://dx.doi.org/10.2214/ajr.157.1.2048539
19. PA McCullough, A Adam, CR Becker, et al., "Risk prediction of contrast induced nephropathy," American Journal of Cardiology 2006;98(6): 27–36.
20. CJ Davidson, M Hlatky, KG Morris et al., "Cardiovascular and renal toxicity of a nonionic radiographic Cardiology Research and Practice 9 contrast agent after cardiac catheterization. A prospective trial." Annals of Internal Medicine 1989;110(2):119–124.
21. Rihal CS, Textor SC, Grill DE, et al. Incidence and prognostic importance of acute renal failure after percutaneous coronary intervention. Circulation 2002;105:2259–2264. http://dx.doi.org/10.1161/01. CIR.0000016043.87291.33
22. Nikolsky E, Mehran R, Lasic Z, et al. Low hematocrit predicts contrast–induced nephropathy after percutaneous coronary interventions. Kidney Int 2005;67:706–13.
23. Guitterez NV, Diaz A, Timmis GC, et al. Determinants of serum creatinine trajectory in acute contrast nephropathy. J Interv Cardiol 2002;15:349–54.
24. Alamartine E, Phayphet M, Thibaudin D, et al. Contrast medium induced acute renal failure and cholesterol embolism after radiological procedures: Incidence, risk factors, and compliance with recommendations. Eur J Intern Med 2003;14: 426–431.
25. Al–Beladi FI. Cystatin C is an early marker of contrast–induced nephropathy in patients with sepsis in the intensive care unit. Saudi J Kidney Dis Transpl 2015;26:718–24.