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Post Cardiac Intervention Contrast Induced Nephropathy

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ABSTRACT

Background: Contrast-induced nephropathy is one of the leading causes of acute kidney injury acquired in the hospital. It may also become more prevalent with percutaneous coronary procedures. **Objective:** To determine the incidence of contrast induced nephropathy in patients undergoing cardiac intervention. **Study Design:** Descriptive longitudinal study. **Settings:** Department of Cardiology at National Institute of Cardiovascular Diseases (NICVD) Karachi, Pakistan. **Duration:** From 1st July 2022 to 31st December 2022. **Methods:** A total of 163 individuals with acute coronary syndrome and blood creatinine levels of 1.2mg/dl received cardiac intervention. Serum creatinine levels were evaluated at baseline and 48 hours post intervention. Contrast-induced nephropathy was labelled if post intervention level was higher than 1.2 mg/dl. The descriptive statistics were computed. After stratification, the Chi-square test was used, with a p-value of <0.05 considered significant. **Results:** Mean baseline serum creatinine level was 0.86 ± 0.15 mg/dl whereas mean post-PCI serum creatinine level was 1.07 ± 0.133 mg/dl. Nephropathy developed in 25 (15.3 %) of the patients. There was a significant relationship between nephropathy and diagnosis category generally and when the age was greater than 60 years. **Conclusion:** The findings show that contrast-induced nephropathy is common in individuals undergoing PCI.

Keywords: *Contrast induced nephropathy, Cardiac intervention, Acute coronary syndrome.*

INTRODUCTION

Contrast-induced nephropathy (CIN) is an iatrogenic acute kidney injury observed following intravascular infusion of contrast medium for diagnostic or therapeutic intravascular procedures.[1,2,3]

CIN has been documented in 1–25% of cases of hospital-acquired AKI and is the third most common cause of acute tubular necrosis in hospitalized patients, leading to prolonged hospitalization.[4]

As most regular diagnostic tests require contrast media, there is always the danger of contrast-induced nephropathy. This consequence may become more common during percutaneous coronary interventions as the frequency of cardiac procedures increases, patients with various co-morbidities undergo percutaneous coronary interventions, and large quantities of contrast media are used for complex coronary lesions.[5,6] Contrast induced nephropathy is regarded an intrinsic acute kidney injury, usually with preserved diuresis, but in severe cases, acute tubular necrosis and even end-stage renal disease can occur, resulting in debilitating morbidity and fatality from acute renal failure. As a result, early diagnosis of contrast-induced nephropathy is increasingly critical.[5,6]

Renal toxicity can develop soon after intravascular contrast media injection and, in most cases, has no major clinical effects. However, renal function can deteriorate and serum creatinine levels can rise. Serum creatinine normally rises within the first 48 hours after contrast

exposure, peaks at 7 days, and falls to near baseline within 1-3 weeks. 1Controlled hydration is used in high-risk individuals.[7,8,9,10]

Reported incidence of contrast-induced nephropathy after percutaneous coronary intervention varies greatly and is highest among emergency percutaneous coronary intervention.[7,8,9,10] Furthermore, this is not a benign complication and can result in permanent kidney damage, need for short or long-term dialysis, which will place a psychological and financial burden on the patient. After extensive literature search we found that there is a lack of studies conducted on the frequency of contrast induced nephropathy after percutaneous coronary intervention in our settings, so our study will be an effort to determine the incidence of contrast-induced nephropathy in patients receiving PCI.

METHODS

This descriptive longitudinal study was conducted between from 1st July 2022 to 31st December 2022at the Department of Cardiology, National Institute of Cardiovascular Diseases (NICVD) Karachi, Pakistan. The determined sample size was 163, using the percentage of contrast-induced nephropathy following percutaneous coronary intervention (12.0%), d=0.05, and a confidence interval of 95%. To enroll people, non-probability consecutive sampling was utilized.

Subjects aged 30-70 years, of any gender, undergoing cardiac intervention for Acute Coronary Syndrome (unstable angina, non-ST elevation myocardial infarction, and ST elevation myocardial infarction) within 24 hours, and with a normal serum creatinine level (1.2mg/dl) were included in the study, whereas patients with a history of chronic kidney disease or end-stage renal disease, coronary artery bypass grafting, or dialysis were excluded.

After receiving approval from the ethical board of Hospital, all eligible patients meeting the inclusion criteria were counselled and provided thorough information about the protocol, and the primary investigator obtained written informed consent from all patients. All patients were transported to the catheterization lab for cardiac intervention; the intervention was performed by an interventional cardiologist with more than five years of expertise; after the procedure, patients were sent to coronary care unit for monitoring. After 48 hours, serum creatinine levels were reassessed and compared to baseline levels; contrast-induced nephropathy was defined as a rise in serum creatinine levels greater than the usual threshold of 1.2 mg/dl (serum creatinine 1.2 mg is the normal standard level). All of the information was captured on a predesigned proforma. Control of confounding variables was achieved by closely adhering to the inclusion and exclusion criteria.

Version 22 of statistical program for the social sciences (SPSS) was utilized to assemble and analyze patient information. The frequency and percentages of qualitative characteristics such as gender, contrast-induced nephropathy, and diagnosis category (unstable angina (UA), non-ST elevation MI(NSTEMI), ST elevation MI(STEMI)) were computed. For quantitative variables such as age, baseline serum creatinine level, and post serum creatinine level, the mean and standard deviation were computed. The stratification was performed on the basis of gender, age, and Diagnosis category to determine the influence of these modifiers using the Chi-square test. $P < 0.05$ was deemed statistically significant.

RESULTS

Out of 163 participants, nephropathy was present in 25(15.3%) subjects, there were 108 male and 55 female participants. Mean age of study subjects was 61.12 ± 7.72 years. The frequency distribution of gender, diagnosis categories & nephropathy are presented in Table-1. The age was stratified in two groups (≤60, >60) and frequency and percentage of study variables among these groups were calculated. Table 1

Table 1: Frequency of patients according to gender, diagnosis & nephropathy (n=163)

Variable	Subcategory	Frequency	Percentage
Gender	**Male**	108	66.3%
	Female	55	33.7%
Diagnosis	**UA**	34	20.9%
	NSTEMI	56	34.4%
	STEMI	73	44.8%
Nephropathy	**Yes**	25	15.3%
	No	138	84.7%

Mean and standard deviation of age, age in groups, mean baseline & Post PCI (after 48 hours) serum creatinine level were calculated and Post serum creatinine levels were also stratified in two groups as presented in Table 2.

Table 2: Descriptive statistics of age & serum creatinine levels (n=163)

	Mean ± SD
Age (years) (n=163)	61.12 ± 7.72
≤ 60 Years (n=47)	52.65 ± 9.53
> 60 Years (n=116)	64.56 ± 2.53
Base Line Serum Creatinine Level (mg/dl) (n=163)	0.86 ± 0.15
Serum Creatinine Level (mg/dl) 48 Hours after procedure (n=163)	1.07 ± 0.133
Serum Creatinine Level (mg/dl) <1.2 mg/dl (n=135) **48 Hours after Procedure**	1.03 ± 0.09
Serum Creatinine Level (mg/dl) ≥ 1.2 mg/dl (n=28) **48 Hours after Procedure**	1.28 ± 0.09

The association of nephropathy with age, gender and diagnosis was also determined and presented in Table 3.

Table 3: Association of nephropathy with age, gender & diagnosis

Variable	Subcategory	Nephropathy		P-Value
	(n=163)	**Yes** (n=25) 15.3%	**No** (n=138) 84.7%	
Gender	**Male** (n=108)	17	91	0.841
	Female (n=55)	8	47	
Age Group	**≤60 Years** (n=47)	7	40	0.920
	>60Years (n=116)	18	98	
Diagnosis	**UA** (n=34)	3	31	0.045*
	NSTEMI (n=56)	14	42	
	STEMI (n=73)	8	65	
	(n=47)	**Yes** (n=7)	**No** (n=40)	
Gender with Age ≤60 Years (n=47)	**Male** (n=34)	5	29	0.953
	Female (n=13)	2	11	
Diagnosis in Age ≤60 Years (n=47)	**UA** (n=)	1	10	
	NSTEMI (n=12)	2	10	1.000
	STEMI (n=24)	4	20	
	(n=116)	**Yes** (n=18)	**No** (n=98)	
Gender with Age >60 Years (n=116)	**Male** (n=74)	12	62	0.783
	Female (n=42)	6	36	
Diagnosis in Age >60 Years (n=116)	**U A** (n=23)	2	21	0.037*
	NSTEMI (n=44)	12	32	
	STEMI (n=49)	4	45	

DISCUSSION

PCI is a common first-line treatment for people with ACS. However, problems such as CIN might occur, resulting in an increased hospital stay, morbidity, and mortality. So, knowing the exact complications burden is essential for future guidelines and prevention.[11]

Main findings of the present study are high incidence of CIN and significant association of nephropathy with diagnosis categories. It was also associated with diagnosis when age was >60 years. Higher incidence was found in NSTEMI. No significant association of nephropathy was observed with gender and age group. No significant association with respect to diagnosis and Gender was observed when patient's age was ≤60 years. While in patients age > 60, no significant association was found with gender.

Published studies reported different incidence rate ranging from 6.6% to 13.3% frequently.[7,8,9] A recent metanalysis concluded that CIN incidence is not low and is associated with age and comorbidities like diabetes, hypertension, prior MI or poor GFR.[7]

Another study with reported a relatively lower incidence of CIN and concluded that pre intervention inter arm systolic blood pressure difference predicts CIN.[8]

In another study around 9% subjects developed CIN and while comparing different prediction scorings they found Mehran Risk Score predicts CIN better than others. 9Other authors supported PRECISE-DAPT score.[12]

Diabetes and IHD enhances the risk of development of CIN.[13] Another study suggested that determining pre intervention red cell distribution width can help in predicting CIN.[14] A study reported 13% incidence and found that both diabetes and amount of contrast are the determining factors. Another study reported 15% incidence and found that in MI patients CIN is a predictor of mortality.[15]

Two Pakistani studies reported low incidence when compared to present study and concluded that this is a non-frequent complication. Studies suggested to be more cautious in cardiac failure patients and in those where kidneys functions are compromised.[9,16]

Timings for reporting post intervention creatinine level and cut off varies in the studies. Some studies measured levels at 24 hours some at 72 hours. That may affect reporting. In the present study our cut off was >1.2gm/dl whereas studies used 0.5% increase from baseline.[9,16] So a little variation in results may be due to difference in ways of interpretation.

Another study suggested that lifesaving intervention must be carried and initial risk assessment will guide for preventive measures. Hydration is suggested as a preventive measure in such cases.[17,18]

It is unclear whether our findings may be extrapolated nationally and internationally due to the limited sample size, the study's confinement to a single centre, and the study's focus on local participants. Regarding association with age, it was not possible to detect any cases older than 70 years due to the age restrictions of our study age group; therefore, extensive data sets with greater age limits are required to find precise results in different age

groups. A further disadvantage is that males predominated in our investigation. The study focused on the urban environment.

This research revealed that patients with a baseline increase in blood Cr-concentration were more likely to suffer acute renal failure following PCI and were more likely to develop renal illness in-hospital. Recognizing the risk burden of renal dysfunction is critical for risk stratification and may aid in the creation of management strategies customized to enhance outcomes, such as the appropriate use of cardiovascular diagnostic tests and medications utilized in current cardiovascular care.

CONCLUSION

According to the findings of our investigation, nephropathy was identified in around 15% of patients following cardiac intervention.

LIMITATIONS

This study contains a relatively small number of participants.

SUGGESTIONS / RECOMMENDATIONS

Studies at larger sample should be conducted in future on this topic.

CONFLICT OF INTEREST / DISCLOSURE

None

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REFERENCES

1. Kusirisin P, Chattipakorn SC, Chattipakorn N. Contrast-induced nephropathy and oxidative stress: mechanistic insights for better interventional approaches. Journal of Translational Medicine. 2020 Dec;18(1):1-35.
2. Lopez-Ruiz A, Chandrashekar K, Juncos LA. Changing paradigms in contrast nephropathy. J Am Soc Nephrol. 2017;28(2):397–9.
3. Ehrmann S, Aronson D, Hinson JS. Contrast-associated acute kidney injury is a myth: yes. Intensive Care Med. 2018;44(1):104–6.
4. Pattharanitima P, Tasanarong A. Pharmacological strategies to prevent contrast-induced acute kidney injury. Biomed Res Int. 2014;2014:236930.
5. Rudnick MR, Leonberg-Yoo AK, Litt HI, Cohen RM, Hilton S, Reese PP. The controversy of contrast-induced nephropathy with intravenous contrast: what is the risk?. American Journal of Kidney Diseases. 2020 Jan 1;75(1):105-13.
6. Shams E, Mayrovitz HN. Contrast-induced nephropathy: a review of mechanisms and risks. Cureus. 2021 May 4;13(5).
7. He H, Chen XR, Chen YQ, Niu TS, Liao YM. Prevalence and Predictors of Contrast-Induced Nephropathy (CIN) in Patients with ST-Segment Elevation Myocardial Infarction (STEMI) Undergoing Percutaneous Coronary Intervention (PCI): A Meta-Analysis. J Interv Cardiol. 2019 Aug 25;2019:2750173.
8. Simsek Z, Zehir R, Kalkan S, Ceneli D, Alizade E, Bayam E, et al. Inter-arm blood pressure difference is associated with contrast-induced nephropathy in patients with ST-segment elevation myocardial infarction who underwent primary percutaneous coronary intervention. Clin Exp Hypertens. 2022 Apr 3;44(3):258-262.
9. Kumar R, Ahmed Khan K, Rai L, Ahmed Solangi B, Ammar A, Nauman Khan M, Ahmed I, et al. Comparative analysis of four established risk scores for predicting contrast induced acute kidney injury after primary percutaneous coronary interventions. Int J Cardiol Heart Vasc. 2021 Oct 29;37:100905.
10. Zhang F, Lu Z, Wang F. Advances in the pathogenesis and prevention of contrast-induced nephropathy. Life sciences. 2020 Oct 15;259:118379.
11. Kelesoglu S, Yilmaz Y, Elcık D, Çetınkaya Z, Inanc MT, Dogan A,et al. Systemic immune inflammation index: a novel predictor of contrast-induced nephropathy in patients with non-ST segment elevation myocardial infarction. Angiology. 2021 Oct;72(9):889-95.
12. Çınar T, Tanık VO, Aruğaslan E, Karabağ Y, Çağdaş M, Rencüzoğulları İ, et al. The association of PRECISE-DAPT score with development of contrast-induced nephropathy in patients with ST-elevation myocardial infarction undergoing primary percutaneous coronary intervention. Cardiovasc Interv Ther. 2019 Jul;34(3):207-215.
13. Adil M, Khan I, Hassan Z, Habib SA, Jibran MS, Nawaz T Sr. One Year Outcomes After Percutaneous Coronary Intervention in Diabetics With Stable Ischemic Heart Disease: A Single-Center Comparative Study. Cureus. 2021 Jan 16;13(1):e12731.
14. Latif A, Ahsan MJ, Lateef N, Kapoor V, Fazeel HM, Razzaq F, et al. A. Prognostic Impact of Red Cell Distribution Width on the Development of Contrast-Induced Nephropathy, Major Adverse Cardiac Events, and Mortality in Coronary Artery Disease Patients Undergoing Percutaneous Coronary Intervention. Curr Cardiol Rev. 2021;17(6):e051121191160.
15. Kumar D, Liaquat H, Sial JA, Saghir T, Kumari R, Kumar H, etal. Risk Factors Associated With Contrast-Induced Nephropathy after Primary Percutaneous Coronary Intervention. Cureus. 2020 Aug 13;12(8):e9721.
16. Soofi MA. Frequency of acute renal failure after cardiac catheterization and percutaneous intervention. Pak J Med Sci. 2006;22:446-50.
17. de Laforcade L, Bobot M, Bellin MF, Clément O, Grangé S, Grenier N, Wynckel A, Guerrot D. Kidney and contrast media: Common viewpoint of the French Nephrology societies (SFNDT, FIRN, CJN) and the French Radiological Society (SFR) following ESUR guidelines. Diagnostic and Interventional Imaging. 2021 Mar 1;102(3):131-9.
18. Faucon AL, Bobrie G, Clément O. Nephrotoxicity of iodinated contrast media: From pathophysiology to prevention strategies. European Journal of Radiology. 2019 Jul 1;116:231-41.