JPSIM Journal of Pakistan Society of Internal Medicine

Original Article

Frequency of Contrast-Induced Nephropathy in Type 2 Diabetes Mellitus Patients Underwent Percutaneous Coronary Intervention for Acute Coronary Syndrome

Waqar Ahmad, Anas Sajjad, Farwa Abbas, Amna Riaz, Rae Hasnain Shehzad, Iqra Waheed

Mayo Hospital Lahore

Abstract

Objective: The purpose of this study was to determine the incidence of contrast-induced nephropathy in patients with type 2 diabetes mellitus who had percutaneous coronary intervention for acute coronary syndrome.

Methods: This descriptive study was undertaken in Department of Cardiology, Mayo Hospital, Lahore during July 2018 to January 2019. Total 165 patents fulfilling selection criteria were enrolled in the study. Then all patients underwent PCI. After that, patients were sent to cardiology wards and monitored for the next two days. Serum creatinine levels were measured after 48 hours of blood collection. If creatinine>25% from baseline, then CIN was labelled.

Results: The mean age of patients was 55.75±6.05 years. There were 125(75.8%) males and 40(24.2%) females. The mean duration of diabetes mellitus was 7.97±0.98 years. The mean duration of ACS was 10.84±5.95hours. There were 57(34.54%) patients who were diagnosed with CIN.

Conclusion: The frequency of CIN in type 2 diabetes mellitus patients underwent PCI for ACS was 34.54%.

Key words: Contrast-induced nephropathy, type 2 diabetes mellitus, percutaneous coronary intervention

How to cite this:

Ahmad W, Sajjad A, Abbas F, Riaz A, Shehzad RH, Waheed I. Frequency of Contrast-Induced Nephropathy in Type 2 Diabetes Mellitus Patients Underwent Percutaneous Coronary Intervention for acute Coronary Syndrome. J Pak Soc Intern Med. 2023;4(2): 125-129

Corresponding Author: Dr. Waqar Ahmad

Email: doc_waqar10@yahoo.com

DOI: https://doi.org/10.70302/jpsim.v4i2.2326

Introduction

A common side effect of angiographic procedures like PCI is contrast-induced nephropathy (CIN), which affects the kidneys. These procedures are often done on patients with acute coronary syndrome (ACS).[1,2] In individuals with renal functional impairment, type II diabetes mellitus is a major risk factor for CIN.[2,3] Individual risk assessments for preventative actions during contrast media treatments are necessary due to the high risk of CIN formation and its prognostic importance in individuals with Type 2 diabetes mellitus.[4,5]

In the absence of other possible causes of nephropathy, such as nephrotoxins, hypotension, urinary obstruction, or atheromatous emboli, a serum creatinine elevation of >25% or >0.5mg/dl (44mol/l) from baseline within 48h is considered to be clinically significant and indicative of a nephrotic syndrome. Serum creatinine levels typically peak between days three and five and go back to normal between days ten and fourteen.[1,6,7]

Diabetes patients with mild to moderate chronic renal insufficiency have an incidence of 9-40%, whereas those with severe chronic renal insufficiency have an incidence of 50-90%.[8] Based on the criteria of CIN, whereby the blood creatinine concentration must rise by 25% over its initial value, the incidence of CIN would be 0.88% among the whole patient population (9.33%).[9] Another research found that following PCI, the incidence of CIN in individuals with type II diabetes was 21.5%.[10] One research, however, found that even after PCI, 40.4% of diabetic individuals still had CIN.[11]

The purpose of this research was to determine how often CIN occurred in individuals with Type 2 diabetes mellitus who had PCI for ACS. The incidence of CIN differs among people with Type 2 diabetes mellitus, according to the available literature. But not much work has been done in this regard and the above mentioned studies contain controversy regarding the extent of CIN after PCI among diabetics. So we wanted to conduct this study to get the local evidence to get the extent of problem in local population. So that, in future, among diabe-

tics, we can plan preventive strategies to avert development of CIN or manage the patients earlier to prevent permanent loss of renal function. The purpose of this study was to determine the incidence of contrast-induced nephropathy in patients with type 2 diabetes mellitus who had percutaneous coronary intervention for acute coronary syndrome.

Methods

This descriptive study was undertaken in Department of Cardiology, Mayo Hospital, Lahore during July 2018 to January 2019. A sample size of 165 cases was computed by using a 95% confidence level, a 4.5% margin of error, and the estimated proportion of CIN, i.e. 9.33%, in patients with type - II diabetes undergoing percutaneous coronary intervention,

Sampling technique: Non-probability, consecutive sampling

Inclusion and Exclusion Criteria: Patients of age 40-65 years of either gender presenting as Type II diabetes mellitus (as per operational definition) planned to undergo PCI were enrolled for acute coronary syndrome. Diabetes was defined as BSR>186mg/dl for >1 year and patient taking anti-glycaemic medication. Acute coronary syndrome was defined as Unstable angina (chest pain>12hours, dyspnoea, no ST elevations on ECG, troponin<100), STEMI (chest pain>12 hours, dyspnoea, troponin>100, ST elevation>1mm on ECG) or NSTEMI (chest pain>12 hours, dyspnoea, troponin >100, no ST elevation). Patients with renal problem or on dialysis, already had previous angiography or PCI were excluded from the study.

Data collection procedure: Informed consent was obtained. Demographic details was also obtained. Serum creatinine levels were determined after an initial blood draw using a 3cc BD syringe and a hospital laboratory. Then, a single senior cardiologist with at least 4 years of residency performed PCI on all of the patients. After that, patients were transferred to cardiology wards and monitored for the next two days. A 3cc BD syringe was used to draw blood at 48 hours post-injury, and the sample was transferred to the hospital lab for serum creatinine analysis. If creatinine>>25% from baseline, then CIN was labelled. Performa was used to collect the data.

Data analysis: All the data was analysed using SPSS version 21. CIN was presented as frequency and percentage.

Results

The mean age of the patients was 55.75 ± 6.05 years. The mean duration of diabetes mellitus was 7.97 ± 0.98 years. There were 125 (75.8%) males and 40 (24.2%) females in our study. There were 58 (35.15%) patients in our study who were under weight, 57 (34.54%) were normal and 50 (30.30%) were overweight. The mean duration of ACS was 10.84 ± 5.95 hours. The mean creatinine level at baseline was 0.95±0.059 mg/dl. The mean creatinine level after 48 hours was 1.17 ± 0.18 mg/dl. There were 49 (29.69%) patients with unstable angina, 57 (34.54%) with ST-elevation myocardial infarction and 59 (35.75%) with Non-ST elevation myocardial infarction. Table 1

There were 57 (34.54%) patients who were diagnosed with CIN while 108 (65.45%) were not having CIN. Fig 1.

There was no significant association between CIN and age groups of the patients as the p-value was not significant (p-value = 0.77). There was no significant association between CIN and gender as the p-value was insignificant (p-value = 0.282). There was significant association between CIN and BMI of the patients as the p-value was significant (p-value = 0.039). There is significant association between CIN and Duration of diabetes mellitus as the p-value was significant (P-value = 0.014). There was insignificant association between CIN and Duration of ACS as the p-value was insignificant (p-value = 0.149) Table 2

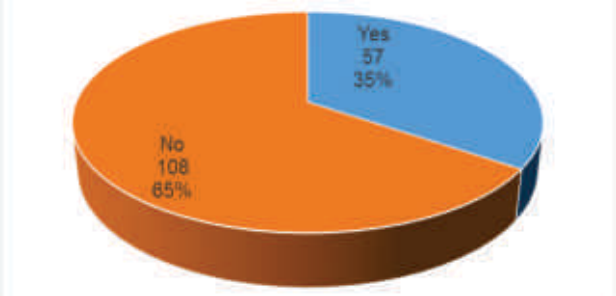

Fig 1: *Frequency of Distribution*

Table 1: *Basic information of enrolled patients*

	Mean
n	**165**
Age of Patients	55.75 ± 6.07
Gender	
Male	125 (75.76%)
Female	40 (24.24%)
BMI	
Under weight	58 (35.15%)
Normal weight	57 (34.54%)
Overweight	50 (30.30%)
Type II Diabetes mellitus	7.97 ± 0.98
ACS	10.84 ± 5.95
Creatinine at baseline	0.96 ± 0.06
Creatinine after 48 hours	1.17 ± 0.158
Type of ACS	
Unstable angina	49 (29.69%)
ST-elevation myocardial infarction	57 (34.54%)
Non-ST-elevation myocardial infarction	59 (35.75%)

Table 2: *Comparison of CIN in different groups*

		CIN		Chi-square	P-value
		Yes	No		
Age group	45-51	18 (36.0%)	32 (64.0%)	0.51	0.77
	52-58	20 (37.0%)	34 (63.0%)		
	59-65	19 (31.1%)	42 (68.9%)		
Gender	Male	46 (36.8%)	79 (63.2%)	1.159	0.282
	Female	11 (27.5%)	29 (72.5%)		
BMI	Normal	19 (32.8%)	39 (67.2%)	9.69	0.036
	Overweight	15 (26.3%)	42 (73.7%)		
	Obese	23 (46.0%)	27 (54.0%)		
Diabetes Mellitus	5-6	4 (25.0%)	12 (75.0%)	11.83	0.014
	7-8	27 (30.0%)	63 (70.0%)		
	9-10	26 (44.1%)	33 (55.9%)		
ACS (hours)	1-12	31 (40.3%)	46 (59.7%)	2.085	0.149
	13-24	26 (29.5%)	62 (70.5%)		

Discussion

When patients are admitted to hospitals, CIN is a major contributor to the development of acute renal damage. Twelve percent to twenty six percent of patients with myocardial infarction who had PCI were found to have CIN in prior research.[12] Studies have shown a connection between CIN and a number of other risk factors. Factors including diabetes, chronic renal failure and volume of contrast utilized in the surgery all increase the likelihood of complications.[13]

According to Kumar et al 2017 there were, 69.6% were males and 30.4% were females in their study and the mean age of patients was 56.6 ±12.5 years.[14] Whereas in our study there were 55.15% were males and 44.85% were females while the mean age of the patients was 55.75±6.05 years.

According to Sany et al., overall incidence of CIN in type - II diabetics was 21.5% whereas in our study the frequency of CIN was 34.53% which is a little bit higher than the study mentioned above.[10] According to another study the baseline serum was less than 1.29 mg/dl which is similar to our findings while the creatinine after 48 hours was greater than 1.29 mg/dl which was also similar to the findings of our study.[10]

According to Toprak et al., the frequency of CIN was 20% among the diabetic patients this is also a little bit less as compare to our study.[15] Clinically significant associations between CIN and end-organ damage were seen for cardiogenic shocks, volume depletion, low ejection fraction & reduced cardiac functioning, after myocardial infarction. The global frequency of CIN is still significant, at 15–7%, despite a drop over the last decade.[16] Prophylaxis is frequently suggested before percutaneous coronary intervention in patients with diabetic nephropathy due to their very high risk of acquiring CIN during the procedure. [17]

There is insufficient evidence to recommend regular CIN prevention for those with diabetes mellitus and normal renal function.[18] This study found that despite having normal pre-procedure serum creatinine levels, patients with diabetes who have albuminuria remain at a high risk for CIN. The incidence of CIN was 21.5% overall among diabetic patients, 17% among patients with microalbuminuria, and 26% among patients with macro-albuminuria.[18]

The presence of diabetes mellitus was shown to have a real and substantial link with the probability of CIN development in relation to risk factors of cardiovascular disease. Certain odd parameters found to have a substantial correlation with the probability of acquiring CIN based on statistical examination of their baseline features. As expected, diabetes mellitus and diuretic usage topped the list, but none of the other medication classes included in our research exhibited any significance in avoiding or generating CIN.[19]

Typically, patients are selected for renoprotective treatments and post-procedural careful monitoring of creatinine levels based on their creatinine levels. Creatinine levels are also monitored closely. Still, the fact that 8.6% individuals with normal blood creatinine level, developed CIN even though they had creatinine clearance about 560 mL/min/1.73 m² shows how important it is for all diabetic patients to find out their creatinine clearance before being exposed to contrast medium.[10]

While cardiac causes accounted for 44% of deaths in this study, myocardial infarction or death in 6.3% patients

who had balloon angioplasty, and in 3.3% individuals who received medical care alone.[20] Although there was a significant reduction in angina symptoms in both groups three months after randomization, the medical group had 16.5% absolute excess of grade II or severe angina.

Throughout the course of the study, 23% of those assigned to the physician group needed revascularization. After treatment, 7.9% of patients in the angioplasty group and 5.8% of patients in the medically managed group needed bypass surgery. Those who participated in RITA-II either had no symptoms or just minor symptoms, but the majority of them had severe anatomic CAD: 62% had multi-vessel disease, and 34% had substantial proximal left anterior descending artery disease.

The AVERT study found that after 18 months, 13% of the people who were treated with medicine and 21% people who had angioplasty had ischaemic events. These results suggest that low-risk patients with stable coronary artery disease may be able to avoid ischemic events just as well with intensive lipid-lowering medication as with balloon angioplasty.[21] Three hundred and forty-one individuals with stable coronary artery disease symptoms were randomly randomized to receive either balloon angioplasty or atorvastatin treatment.

Data from randomized studies compared medical therapy versus balloon angioplasty showed that medical treatment should be regarded as the first care option for in most of the cases who have "Canadian Cardiovascular Society Classification class I & II symptoms." Those with more severe symptoms and ischemia should be the priority for percutaneous or surgical revascularization.

The COURAGE experiment showed that if medical treatment alone can manage angina symptoms in individuals with modest, stable angina & coronary artery stenosis, then this might be a suitable approach.[22] The study compared intensive pharmacologic treatment with angioplasty versus intense pharmacologic therapy alone, with mortality from any cause and nonfatal MI as the outcomes, with average follow-up around five years.

Conclusion

Patients with type 2 diabetes mellitus who had PCI for ACS had a frequency of CIN of 34.54%. There is currently no cure for CIN, making it all the more important to identify those at high risk and take measures to reduce the disease's prevalence.

Conflict of Interest: *None*

Funding Source: *None*

References

1. Mohammed NM, Mahfouz A, Achkar K, Rafie IM, Hajar R. Contrast-induced nephropathy. Heart views: Official J Gulf Heart Assoc. 2013;14(3):106.
2. Barbieri L, Verdoia M, Schaffer A, Cassetti E, Di Giovine G, Marino P, et al. Pre-diabetes and the risk of contrast induced nephropathy in patients undergoing coronary angiography or percutaneous intervention. Diab Res Clin Prac. 2014;106(3):458-64.
3. Heyman SN, Rosenberger C, Rosen S, Khamaisi M. Why is diabetes mellitus a risk factor for contrast-induced nephropathy? BioMed Res Int. 2013; https:// doi. org/ 10.1155/2013/123589.
4. Han Y, Zhu G, Han L, Hou F, Huang W, Liu H, et al. Short-term rosuvastatin therapy for prevention of contrast-induced acute kidney injury in patients with diabetes and chronic kidney disease. J Am Coll Cardiol. 2014; 63(1):62-70.
5. Deng J, Wu G, Yang C, Li Y, Jing Q, Han Y. Rosuvastatin attenuates contrast-induced nephropathy through modulation of nitric oxide, inflammatory responses, oxidative stress and apoptosis in diabetic male rats. J Translational Med. 2015;13(1):53.
6. Tsai M-L, Mao C-T, Chen D-Y, Hsieh I-C, Wen M-S, Chen T-H. Short-and long-term major cardiovascular adverse events in carotid artery interventions: a nationwide population-based cohort study in Taiwan. PloS one. 2015;10(3):e0121016.
7. Jin B, Wu B, Zhang J, Luo X, Shi H. Preventive effect of reduced glutathione on contrast-induced nephropathy in elderly patients undergoing coronary angiography or intervention: a randomized, controlled trial. Brazilian J Med Biolog Res. 2015;48(9):839-42.
8. Gleeson TG, Bulugahapitiya S. Contrast-induced nephropathy. Am J Roentgenol. 2004;183(6):1673-89.
9. Zhang M-M, Lv Q-Z, Li X-Y. Drug effects and clinical investigations for contrast-induced nephropathy after coronary angiography or percutaneous coronary intervention in patients with diabetes. Am J Therapeut. 2017; 24(4):e423-e30.
10. Sany D, Refaat H, Elshahawy Y, Mohab A, Ezzat H. Frequency and risk factors of contrast-induced nephropathy after cardiac catheterization in type II diabetic patients: a study among Egyptian patients. Renal Failure. 2014;36(2):191-7.
11. Shamkhalova M, Zaĭtseva N, Kurumova K, Shestakova M, Deev A, Matskeplishvili S, et al. Contrast-induced nephropathy in coronarography of patients with type 2 diabetes mellitus: risk factors, prognostic significance, prophylactic approaches. Terapevticheskii Arkhiv. 2009; 81(8):36-42.
12. Mehran R, Aymong ED, Nikolsky E, Lasic Z, Iakovou I, Fahy M, et al. A simple risk score for prediction of contrast-induced nephropathy after percutaneous coronary intervention: development and initial validation. J Am Coll Cardiol. 2004;44(7):1393-9.

13. Manske CL, Sprafka JM, Strony JT, Wang Y. Contrast nephropathy in azotemic diabetic patients undergoing coronary angiography. Am J Med. 1990;89(5):615-20.
14. Kumar S, Nair RK, Aggarwal N, Abbot A, Muthukrishnan J, Kumar KH. Risk factors for contrast-induced nephropathy after coronary angiography. Saudi J Kidney Dis Transplant. 2017;28(2):318.
15. Toprak O, Cirit M. Risk Factors for Contrast-Induced Nephropathy. Kidney Blood Pressure Res. 2006; 29(2): 84-93.
16. Roy P, Raya V, Okabe T, Slottow TLP, Steinberg DH, Smith K, et al. Incidence, predictors, and outcomes of post-percutaneous coronary intervention nephropathy in patients with diabetes mellitus and normal baseline serum creatinine levels. Am J Cardiol. 2008; 101(11): 1544-9.
17. Bartholomew BA, Harjai KJ, Dukkipati S, Boura JA, Yerkey MW, Glazier S, et al. Impact of nephropathy after percutaneous coronary intervention and a method for risk stratification. Am J Cardiol. 2004; 93(12): 1515-9.
18. Solomon R. The role of osmolality in the incidence of contrast-induced nephropathy: a systematic review of angiographic contrast media in high risk patients. Kidney Int. 2005;68(5):2256-63.
19. LA NEFROPATIA DMDC. La prevenzione della nefropatia da mezzo di contrasto e la gestione del paziente a rischio. G Ital Cardiol. 2009;10(2):88-96.
20. Sculpher M, Smith D, Clayton T, Henderson R, Buxton M, Pocock S, et al. Coronary angioplasty versus medical therapy for angina. Health service costs based on the second Randomized Intervention Treatment of Angina (RITA-2) trial. Eu Heart J. 2002;23(16):1291-300.
21. Pitt B, Waters D, Brown WV, van Boven AJ, Schwartz L, Title LM, et al. Aggressive lipid-lowering therapy compared with angioplasty in stable coronary artery disease. New Eng J Med. 1999;341(2):70-6.
22. Teo KK, Sedlis SP, Boden WE, O'Rourke RA, Maron DJ, Hartigan PM, et al. Optimal medical therapy with or without percutaneous coronary intervention in older patients with stable coronary disease: a pre-specified subset analysis of the COURAGE (Clinical Outcomes Utilizing Revascularization and Aggressive druG Evaluation) trial. J Am Coll Cardiol. 2009;54(14):1303-8.