A COMPARSION OF CONTRAST INDUCED NEPHROPATHY BETWEEN NORMAL AND HIGH RISK POPULATION UNDERGOING CORONARY ANGIOGRAPHY

Mir Waqas Baloch, Rehana Khadim, Samra Rehmat, Mohammad Asad, Sohail Aziz, Tahir Iqbal, Imran Fazal, Tariq Hussain Khattak, Azhar Ali Chaudary, Hasnain Iqbal

Armed Forces Institute of Cardiology/National University of Medical Sciences (NUMS) Rawalpindi Pakistan

ABSTRACT

Objective: To determine the frequency of contrast induced nephropathy in patients undergoing coronary angiography and compare frequency of contrast induced nephropathy in low and high risk patients.
Study Design: A comparative cross-sectional study.
Place and Duration of study: Cardiac cath department, Armed forces institute of cardiology and National institute of heart diseases, Rawalpindi from Jul 2017 to Dec 2017.
Material and Methods: Patients undergoing elective percutaneous coronary angiography with co-morbids (diabetes mellitus and hypertention) were included. Diabetics who are on oral hypoglycemic agents i.e. metformin was stopped 48 hours prior to the procedure. Patients were enrolled into two groups; one with normal baseline creatinine level and the second group with abnormal baseline i.e. creatinine >1.5, diabetes and hypertention which was pre-hydrated. All patients were administered intravenous fluid (normal saline) before the procedure.
Results: Total 115 patients were included according to the inclusion criteria of the study. Mean age was 45.11 ± 7.09 years while there were 60 (52.2%) male and 55 (47.8%) female patients in the study. Frequency of contrast induced nephropathy in patients undergoing coronary angiography was 11 (9.3%), whereas frequency of contrast induced nephropathy in low and high risk patients was 8 (72.7%) and 3 (27.3%) respectively which was statistically not significant (*p*-value 0.980).
Conclusion: The study showed that occurrence of contrast induced nephropathy in patients undergoing coronary angiography was although not statistically significant but more studies should be done in order to contribute in the existing research through validation of risk factors (predictors) for contrast induced nephropathy in diabetic and hypertensive patients.

Keywords: Contrast Induced Nephropathy, Coronary angiography, High risk patients, low risk patients.

INTRODUCTION

The use of radio contrast media has increased greatly from the past decades for diagnostic radiography and interventional procedures and it is estimated that approximately 60 million people in the world are administered radio contrast medium each year[1]. Contrast induced nephropathy (CIN) has gained increased attention in the clinical setting, particularly during cardiac intervention but also in many other radiological procedures in which iodinated contrast media are used[2]. There is at present good clinical evidence from well-controlled randomized studies that CIN is a common cause of acute renal dysfunction[3]. After radiographic contrast procedures 12 to 14 percent of patients suffer from acute renal insufficiency during hospitalization[4]. CIN is the third primary cause of hospital-acquired acute renal failure[5,6].

Contrast induced nephropathy is the acute deterioration of renal function after parenteral administration of radiocontrast media in the absence of other causes. Contrast Induced Nephropathy is generally defined as an increase in serum creatinine concentration of >0.5 mg/dl or 25% above baseline within 48 hours after contrast administration[5,7]. Although the exact mechanisms of CIN have yet to be fully elucidated, several causes have been described. Increased adenosine, endothelia, and free radical–induced vasoconstriction and reduced

Correspondence: Dr Mir Waqas Baloch, Armed Forces Institute of Cardiology/NHID Rawalpindi Pakistan.
Email:mirwaqas8@gmail.com)

nitric oxide and prostaglandin-induced vasodilatation have been observed[5]. Contrast agents also have direct toxic effect on renal tubular cells causing vacuolization and altered mitochondrial function[2,6]. According to minnesota registry of interventional cardiac procedures, RCIN was found in 22 percent of patients with serum creatinine >2 mg/ dl and in 30 percent of patients with serum creatinine >3 mg/dl[2]. CIN in patients undergoing percutaneous coronary intervention has been reported to have 30 percent mortality rate with risk factors such as dehydration, diabetes mellitus, nephrotoxic drugs and quantity of contrast dye used[3]. The incidence of CIN in the general population has been calculated to be less than 3 percent[4]. In high-risk patients, i.e., patients with preexisting renal dysfunction, diabetes mellitus, congestive heart failure, and older age; the incidence has been calculated to vary extensively from 12 to 50 percent[4]. The occurrence of CIN in diabetic patients ranges from 5.2 to 35.7 percent in recent studies[7]. The chances of progressive deterioration can rise to 42 percent in patients with abnormal baseline renal function[2] Eleven (7.10%) out of 155 patients undergoing coronary angiography experienced contrast induced nephropathy[7]. CIN has been associated with increased morbidity, extended length of hospital stay[5] and increased costs[8].

This study aims to investigate the occurrence of CIN in patients undergoing coronary angiography as there is inevitable clinical need for it. As suggested by literature CIN is more common in patients with diabetes mellitus and hypertension which is why the present study would verify the extent of CIN in diabetic and hypertensive patients undergoing coronary angiography. Moreover, there is a lack of research regarding this complication, hence local data would be available. Thus, the study would contribute in the existing research through validation of risk factors (predictors) for CIN in diabetic and hypertensive patients undergoing coronary angiography and help undertake preventive measures.

MATERIALS AND METHOD

Permission was sought from hospital ethical committee. A descriptive cross sectional study conducted in Armed forces institute of cardiology and National institute of heart disease, Rawalpindi with duration of study was 6 month from Jul 2017 to Dec 2017. Non probability consecutive samplying techniques was used as a samplying technique. Contrast induced nephropathy was taken as greater than 25% increase of serum creatinine or an absolute increase in serum creatinine of 0.5mg/dl after using iodine contrast agent in 48 hours without another clear cause for kidney injury. High risk patients included any one of following parameters; diabetes mellitus documented H/o diabetes mellitus >2 years, hypertension documented H/o hypertension >2 years and baseline-creatinine level >1.5-2.0. Low risk patients included any one of the following parameters; Diabetes mellitus not documented of diabetes mellitus, hypertension not documented of hypertensionand baseline-creatinine level <1.5. Patients undergoing Elective PCI, gender (both male & female) with ages between 20-80 years and Low risk and high risk patients were considered as inclusion criteria of the study whreas patients with chronic kidney disease-V, baseline creatinine >2, prior history of coronary artery by-pass graft surgery, cardiogenic shock, left ventricular ejection fraction less than 30 percent and History of exposure to contrast with in previous 6 months, were considered as exclusion criteria of the study. Particulars of all the patients who meet the inclusion and exclusion criteria was recorded in the Performa. Patients undergoing elective percutaneous coronary angiography with co-morbids (diabetes mellitus and hypertention) were included. Diabetics who are on oral hypoglycemic agents i.e. metformin was stopped 48 hours prior to the procedure. Patients were enrolled into two groups; one with normal baseline creatinine level and the other with abnormal baseline i.e. creatinine >1.5, diabetes and hypertention which was prehydrated. All patients were administered

intravenous fluid (normal saline) before the procedure. Intravenous hydration consisted of 1ml normal saline per kilogram of body weight per hour which was started 12 hours before contrast agent injection and continued for 12 hours after the injection. renal function tests after the procedure was done. Followed by 24 hours and 48 hours interval to observe any late changes in renal functions. Both high and low risk groups were hydrated and limited amount of dye was used i.e. 40-60 cc. Statistical analysis was performed using SPSS version 23. Mean and standard deviation was calculated for quantitative variable like age. Frequency and percentage was calculated for qualitative variable like gender, contrast induced nephropathy, low and high-risk patients. Low and high risk was compared for contrast induced nephropathy by applying chi-square test. A *p*-value <0.05 was taken as significant.

Table-I: Descriptive statistics of Age (years) of patients.

Age (Mean ± SD)		45.11± 7.09 years
		n (%)
Gender	**Male**	60 (52.2%)
	Female	55 (47.8%)

Table-II: Comparsion of high and low risk patients with Contrast-Induced Nephropathy.

	Contrast Induced Nephropathy		**Total**	***p*-value***
	YES	**No**		
High risk	08 (72.7%)	76 (73.1%)	84 (73.0%)	0.980
Low risk	03 (27.3%)	28 (26.9%)	31 (27.0%)	
Total	11 (9.3%)	104 (90.4%)		

*A *p*-value<0.05 was taken as significance.

RESULTS

Total 115 patients were included according to the inclusion criteria of the study. Mean age of the patients was 45.11 ± 7.09 years while there were 60 (52.2%) male and 55 (47.8%) female patients as shown in table-I. Frequency of contrast induced nephropathy in patients undergoing coronary angiography was 11 (9.3%), followed by high and low risk patients in undergoing coronary angiography was 84 (73.0%) and 31 (27.0%) respectively. Frequency of contrast induced nephropathy in low and high risk patients was 8 (72.7%) and 3 (27.3%) respectively which was statistically not significant (*p*-value 0.980), as shown in table-II.

DISCUSSION

Contrast-induced nephropathy (CIN), also known as contrast-induced acute kidney injury, is an iatrogenic renal injury that follows intravascular administration of radio-opaque contrast media (CM) in susceptible individuals[9]. CIN was first described during the 1950s in case reports of fatal acute renal failure that had occurred following intravenous pyelography in patients with renal disease arising from multiple myeloma. Despite technological advances, CIN remains responsible for a third of all hospital-acquired acute kidney injury (AKI) and affects between 1% and 2% of the general population and up to 50% of high-risk subgroups following coronary angiography (CA) or percutaneous coronary intervention (PCI)[6,8]. The serum creatinine levels begin to rise within 24-48 hours, peak at 2-3 days and return to the baseline values within 2 weeks. The most commonly used definition of CIN in the literature is either a relative increase in serum creatinine of 25% or an absolute increase of 0.5 mg/dL from a baseline value within 48 to 72 hours after contrast exposure[10]. The proliferation of imaging methods and interventional procedures involving administration of intravascular CM in both non-cardiac modalities (e.g. vascular CT angiography and interventional vascular angiography)[11] and in established (e.g. CA and PCI) and emerging

cardiac modalities (e.g. CT coronary angiography (CTCA) and transcatheter aortic valve implantation (TAVI)) has significantly increased the number of patients exposed to CM and thus the number at risk of CIN. The widespread adoption of primary PCI for the treatment of acute myocardial infarction (AMI), despite significantly improving cardiovascular outcomes, has increased the incidence of CIN due to the inherent difficulties in rapidly assessing CIN risk, instigating prophylactic measures, attendant haemodynamic compromise and higher contrast volumes, all known risk factors for the development of CIN[12,13]. Despite several therapeutic approaches, the rising age and incidence of comorbidity within the broad cohort of cardiac patients receiving CM has ensured that the prevention of CIN remains a significant clinical challenge[14,15]. In a study by Sajjad et al[7], mean age in years was 58.4 ± 13.43. Similarly, in our study, mean age (years) in the study was 45.11 ± 7.09.

In our study, the frequency and percentage of males were 60(52.2%) and females were 55 (47.8%). Likewise, in a study conducted in 2016[13], there were 83(53.55%) male and 72 (46.45%) female patients respectively. Study conducted by Sajjad et al[7], CIN developing in diabetics, undergoing coronary angiography was recorded in 11 (7.10%). In the same way, frequency of contrast induced nephropathy in patients undergoing coronary angiography was 11 (9.3%). In our study, frequency of contrast induced nephropathy in high risk patients were 03 (27.3%). Similarly, in a study presented in 2016[13] showed that frequency and percentage of CIN patients with high risk was 19 (67.85%).

CONCLUSION

The study showed that occurrence of contrast induced nephropathy in patients undergoing coronary angiography was although not statistically significant but more studies should be done in order to contribute in the existing research through validation of risk factors (predictors) for contrast induced nephropathy in diabetic and hypertensive patients undergoing coronary angiography which will help to undertake preventive measures.

CONFLICT OF INTEREST

This study has no conflict of interest to be declare by any author.

REFERENCES

1. Assareh A, Yazdankhah S, Majidi S, Nasehi N, Mousavi SS. Contrast induced nephropathy among patients with normal renal function undergoing coronary angiography. J Renal Inj Prev 2016; 5(1): 21-4.
2. Hung Y, Lin S, Hung S, Huang W, Wang PY. Preventing radio contrast-induced nephropathy in chronic kidney disease patients undergoing coronary angiography. World J Cardiol 2012; 4(5): 157-72.
3. Kumar V, Dev N, Vasudha KC. Risk of contrast induced nephropathy in patients undergoing primary coronary angioplasty. Int J of Biomed & Adv Res 2012; 3(6): 490-93.
4. Nough H, Eghbal F, Soltani MH, Nejafi F, Falahzadeh H, Fazel H, et al. Incidence and main determinants of contrast induced nephropathy following coronary angiography or subsequent balloon angioplasty. Cardiorenal Med 2013; 3(2): 128-35.
5. Rear R, Bell RM, Hausenloy DJ. Contrast induced nephropathy following angiography and cardiac interventions. Heart 2016;102(8): 638-48.
6. Rezaei Y, Khademvatani K, Rahimi B, Khoshfetrat M, Arjmand N, Seyyed-Mohammadzad MH. Short-term high-dose vitamin e to prevent contrast medium-induced acute kidney injury in patients with chronic kidney disease undergoing elective coronary angiography: a randomized placebo-controlled trial. J Am Heart Assoc. 2016; 5(3): e00219.
7. Sajjad U, Ali I, Akram Z. Frequency of contrast induced nephropathy following exposure to contrast in coronary angiography in diabetics with previously normal renal function. Ann King Edward Med Uni 2016; 22(1): 12-15.
8. Atkuri KR, Mantovani JJ, Herzenberg LA: N-Acetylcysteinea safe antidote for cysteine/glutathione deficiency. Curr Opin Pharmacol 2007; 7(4): 355–9.
9. Thaha M, Widodo, Pranawa W, Yogiantoro M, Tomino Y. Intravenous N-acetylcysteine during hemodialysis reduces asymmetric dimethylarginine level in end-stage renal disease patients. Clin Nephrol 2008; 69(1): 24–32.
10. Klass O, Walker M, Siebach A, Stuber T, Feuerlein S, Juchems M, et al. Prospectively gated axial CT coronary angiography: comparison of image quality and effective radiation dose between 64- and 256-slice CT. Eur Radiol 2010; 20(5): 1124-31.
11. Mehran R, Nikolsky E. Contrast-induced nephropathy: definition, epidemiology, and patients at risk. Kidney Int 2006; 100: s11–15.
12. Maioli M, Toso A, Gallopin M, Leoncini M, Tedeschi D, Micheletti C et al. Preprocedural score for risk of contrast-induced nephropathy in elective coronary angiography and intervention. J Cardiovasc Med 2010; 11(6): 444-9.
13. Solomon R. Contrast-induced acute kidney injury: is there a risk after intravenous contrast? Clin J Am Soc Nephrol 2008; 3(5):1242-43.
14. Romano G, Briguori C, Quintavalle C, Zanca C, Rivera NV, Colombo A, et al. G. Contrast agents and renal cell apoptosis. Eur Heart J 2008; 29(20): 2569-76.
15. Nyman U, Bjork J, Aspelin P, Marenzi G. Contrast medium dose-to-GFR ratio: a measure of systemic exposure to predict contrast-induced nephropathy after percutaneous coronary intervention. Acta Radiol 2008; 49(6): 658–67.