

## Incidence and Associated Risk Factors of Contrast Induced Nephropathy in Diabetes and Non Diabetic Patients

Mohammed Rashed Anwar<sup>1</sup>, KAM Mahbub Hasan<sup>2</sup>, Asraful Hoque<sup>3</sup>, Babrul Alam<sup>4</sup>,  
Dilip Kumar Debnath<sup>5</sup>, Md. Anwarul Hoque Faraji<sup>6</sup>

<sup>1</sup>Assistant Professor, Department of Nephrology, National Institute of Kidney Diseases & urology, Dhaka, Bangladesh; <sup>2</sup>Medical Officer, Department of Cardiology, National Institute of Cardiovascular Diseases, Dhaka, Bangladesh; <sup>3</sup>Resident Surgeon, Department of Cardiac Surgery, National Institute of Cardiovascular Diseases, Dhaka, Bangladesh; <sup>4</sup>Associate Professor, Department of Nephrology, National Institute of Kidney Diseases & urology, Dhaka, Bangladesh; <sup>5</sup>Assistant Professor, Department of Nephrology, National Institute of Kidney Diseases & urology, Dhaka, Bangladesh; <sup>6</sup>Assistant Professor, Department of Nephrology, National Institute of Kidney Diseases & Urology, Dhaka, Bangladesh

[Received: 21 August 2016; Revised: 6 September 2016; Accepted: 11 December 2016; Published: 1 January 2017]

### Abstract

**Background:** Contrast-induced nephropathy (CIN) is the third leading cause of hospital-acquired acute renal failure. **Objective:** The purpose of the present study was to compare the incidence and associated risk factors of contrast induced nephropathy in diabetes and non-diabetic patients. **Methodology:** This was cross-sectional study performed in the Department of Nephrology at National Institute of Kidney Diseases and Urology, Sher-E-Bangla Nagar, Dhaka and Department of Cardiology, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh from January 2016 to July 2016. Contrast induced nephropathy (CIN) is defined as increase in serum creatinine of  $\geq 25\%$  from baseline value and/ or an absolute increase of  $\geq 0.5$  mg/dl in serum creatinine from baseline. Patients were divided in to two groups Group A (Patients with Diabetes mellitus) and Group B (Patients without Diabetes mellitus). To identify independent characteristics associated with CIN, multivariable logistic regression analysis was used through SPSS version 23. Results of this model were presented as Odds Ratio (OR). P value was calculated to see the significance of various risk factors in diabetes and non-diabetes patients. **Results:** The difference in baseline creatinine serum creatinine was found statistically significant ( $P < 0.001$ ). In group A 57 patients (50.9%) had eGFR  $< 60$  ml/min/1.73m<sup>2</sup>, 55 patients (49.1%) had eGFR  $\geq 60$  ml/min. The difference in estimated GFR was found statistically significant ( $P < 0.001$ ). Left ventricular ejection fraction  $< 40\%$  was present in 6 (5.4%), 7 (5.1%) in group A and B respectively,  $\geq 40\%$  in 106 (94.6%), 131 (94.9%) in group A and B respectively. CIN developed in 21 (18.80%) patients in group A and 2 (1.4%) patients in group B (CIN was defined by increased in serum creatinine  $\geq 25\%$  of baseline or  $\geq 44$   $\mu$ mol/L). All belonged to group A, 16 (19%) of the diabetic patients out of 86 developed CIN. Diabetic patients who had eGFR  $< 60$  ml/min ( $n=30$ ), 13 (43.3%) developed CIN. Among all patients ( $n=250$ ), 23 developed CIN. Overall incidence was 9.2%. **Conclusion:** CIN was significant developed in diabetes group than non diabetes. Left ventricular ejection fraction and total volume of contrast media used was significantly higher in diabetes group than non-diabetes group B patients. [Journal of National Institute of Neurosciences Bangladesh, 2017;3(1): 29-36]

**Keywords:** Incidence; risk factors; contrast induced nephropathy; diabetes; non diabetic patients

**Correspondence:** Dr. Mohammed Rashed Anwar, Assistant Professor, Department of Nephrology, National Institute of Kidney Diseases & Urology, Sher-E-Bangla Nagar, Dhaka, Bangladesh; Email: [rashedanwar.68@gmail.com](mailto:rashedanwar.68@gmail.com); Cell no: +8801711368756

**Conflict of Interest:** There is no conflict of interest to any of the authors of this article.

**Contributions to Authors:** MR Anwar, MAH Faraji & B Alam have involved from protocol preparation, data collection, data analysis and report writing. KAMM Hasan, A Hoque & DK Debnath have revised the manuscript. All the authors have read and approved the final version of the manuscript.

**Funding:** This research project was not funded by any group or any institute on.

**How to cite this article:** Anwar MR, Hasan KAMM, Hoque A, Alam B, Debnath DK, Faraji MAH. Incidence and Associated Risk Factors of Contrast Induced Nephropathy in Diabetes and Non Diabetic Patients. J Natl Inst Neurosci Bangladesh. 2017;3(1): 29-36

**Copyright:** ©2017 Anwar et al. Published by Journal of National Institute of Neurosciences Bangladesh. This article is published under the Creative Commons CC BY-NC License (<https://creativecommons.org/licenses/by-nc/4.0/>). This license permits use, distribution and reproduction in any medium, provided the original work is properly cited, and is not used for commercial purposes.

## Introduction

Diabetes mellitus is the most widespread affection of mankind. Diabetes is a syndrome characterized by chronic hyper-glycemia and disturbance of carbohydrate, fat and protein metabolism associated with absolute or relative deficiencies in insulin secretion and/or insulin action<sup>1</sup>. Contrast-induced nephropathy (CIN) is the third leading cause of hospital-acquired acute renal failure, accounting for 10.0% of all cases of hospital-acquired renal failure<sup>2</sup>. Two of the most important risk factors are baseline impaired renal function or estimated glomerular filtration rate (eGFR) <60 ml/min/1.73m<sup>2</sup> and diabetes mellitus.

Contrast media volume is the most important modifiable risk factor. Diabetic patients represent a significant proportion of those undergoing contrast exposures due to high prevalence of diabetes in the general population and the ability of the disease to cause a broad spectrum of cardiovascular diseases that require radiological procedures using CM. The incidence of CIN in diabetic patients varies from 5.7 to 29.4%. Importantly, in diabetic patients with preserved renal function and the absence of other risk factors, the rate of CIN are usually comparable to those of a non diabetic population, while clinically important CIN usually occurs in a subset of diabetics with underlying renal insufficiency. In one study CIN occurred in 27% of diabetic patients with baseline serum creatinine 2.0–4.0 mg/dL and in 81.0% of those with serum creatinine >4mg/dl. In another study, CIN occurred in post percutaneous coronary intervention 15.1% of patients without chronic kidney disease vs 27.4% in those with chronic kidney disease<sup>3</sup>.

Irrespective of cause, preexisting renal impairment appears to be the most important risk factor, patients with creatinine levels greater than 1.5 mg/dL were identified as being under a higher risk. The chance of developing CIN may be up to 7 times greater in patients with CKD<sup>4</sup>. Davidson et al<sup>5</sup> in a series of 1,144 patients undergoing cardiac catheterization, found a low risk of contrast induced nephropathy in patients with normal renal function, but a higher risk in those with preexisting azotemia (serum creatinine level >1.2 mg/dl). The risk increased exponentially with serum creatinine concentration like 20.0% incidence in those with a serum creatinine levels of 2 mg/dL (177 pmol/L) From the above discussion, it can be categorically stated that CIN may occur not only in high risk patients but also in general population. In high risk patients, it could be fatal. So these groups of patients need to be

evaluated with regard to the incidence of risk factors for and outcome of CIN. Therefore this present study was undertaken to compare the incidence and associated risk factors of contrast induced nephropathy in diabetes and non-diabetic patients.

## Methodology

This was a prospective, observational study was carried out in the Department of Cardiology, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh. Patients who underwent elective coronary angiographic evaluation at the department of cardiology, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh, during the study period who fulfilled the inclusion criteria were selected. All patients 18 years and above who underwent coronary angiography with or without percutaneous Transluminal coronary Angioplasty with normal or impaired renal function, with or without Diabetes Mellitus or hypertension were included in the study. Age below 18 years, patients with preexisting end stage renal disease requiring dialysis, history of contrast allergy, patients who developed shock after the procedure, patients underwent other contrast exposure within one week from the index procedure were excluded in this study. Patients were divided in to two groups group A (patients with diabetes mellitus) and group B (Patients without diabetes mellitus). Demographic profile, clinical examination and relevant investigation reports and procedural factors of all patients were recorded in pre-designed data collection sheet. The anti-ischaemic, anti-hypertensive, lipid lowering, platelet inhibitors, and oral hypoglycemic agents (except metformin) if taking were continued. 3, Low osmolar, non-ionic radiocontrast agent iopamidol (Lopamir 370) was used for all patients. Base line serum creatinine was estimated before procedure. Post procedure serum creatinine was estimated at 48 hours after coronary angiogram. For estimation of serum creatinine 2 samples of venous blood (one pre-procedure, 1 post-procedure) 3 cc each were collected and send immediately to laboratory. Sample analyzed by automated clinical chemistry analyzer ABX Pentra 400 of HORIBA ABX, France. Estimated GFR (eGFR) was calculated from MDRD formula both pre and 48 hour post procedure. Study population was divided into two groups. Group A-presence of diabetes mellitus and/or impaired renal function (estimated GFR<60ml/min/1.73m<sup>2</sup>, MDRD prediction equation) and group B absence of diabetes mellitus and estimated GFR ≥60ml/min/1.73m<sup>2</sup>). Incidence of CIN in these

groups were compared. We tried to analyze whether there is relationship between the incidence of CIN with renal impairment, diabetes mellitus, contrast volume, hypertension, Dyslipidemia left ventricular ejection fraction <40%. Statistical analysis was conducted using SPSS 23.0 for windows software. Categorical data were expressed as frequencies and corresponding percentages. Parametric data were expressed in mean±SD. Parametric data were evaluated by independent sample "t" test, categorical data were evaluated by Chi-square test as needed. A multivariable logistic regression model were applied including all the potential confounding variables. Level of significance for all analytical test were set at 0.05 and p value <0.05 is considered significant.

### Results

Total of 250 patients who underwent elective coronary angiographic evaluation at the department of cardiology, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh, during the study period who fulfilled the inclusion criteria were selected. Demographic and baseline characteristics of the study population. The mean age of patient in group A was 55.83±9.00 years whereas the mean age of patients in group B was 50.44±11.63 years. There were statistically significant difference between two groups (P<0.001). Out of 112 patients in group A and 138 patients in group B 87(77.7%) and 119 (86.2%) were male and 25 (22.3%) and 19 (13.8%) were female respectively. Mean (±SD) body mass index (BMI) were 23.74±2.82 and 23.17±2.19 kg/m<sup>2</sup> of group A and group B patients, respectively. Systolic blood pressure of group A and group B patients (mean±SD) were 127.45±14.43 and 126.01±11.87 mmHg, and diastolic blood pressure was 63.13±9.35 and 83.59±9.00mmHg, respectively. Serum creatinine concentration in group A was significantly higher (P<0.001) than group B (mean±SD:107.57±25.48 and 87.01±12.88 µmol/L). In Group A 86 (76.8%) and none in Group B had diabetes mellitus. Left ventricular ejection fraction in group A and group B patients were 58.72±10.40% and 57.64±11.01%, respectively. Total volume of contrast media used was significantly higher in group A than group B patients (mean ± SD 81.03 ± 36.53ml and 59.41 ± 20.89 ml, P<0.001) (Table 1).

Presence of risk factors in group A and group B patients. In group A and group B, impaired renal function (eGFR <60 ml/min/1.73 m<sup>2</sup>) was present in 57(50.9%) and none (0%), diabetes mellitus was present in 86(76.8%) and none (0%), hypertension in

53(47.3%) and 52(37.7%), dyslipidemia in 57(50.9%) and 3(2.2%) and history of myocardial infarction (MI) in 27(24.1%) and 17(12.3%) patients respectively. Statistically hypertension showed no significant variation between the two study groups. However, presence of other risk factors was significantly high among group A patients, impaired renal function (P<0.001, diabetes mellitus (P<0.001), dyslipidemia (P<0.001), and previous myocardial infarction (P<0.05) (Table 2).

Table 1: Demographic and Baseline Characteristics of the Study Patients

Parameters	Group A (n=112)	Group B (n=138)	P Value
Age (years)	55.83±9.00	50.44±11.63	0.0001***
Sex			0.077ns
• Male	87(77.7%)	119(86.2%)	
• Female	25(22.3%)	19(13.8%)	
Body mass index (kg/m <sup>2</sup> )	23.74±2.82	23.17±2.19	0.071ns
Systolic blood pressure (mmHg)	127.45±14.43	126.01±11.87	0.390ns
Diastolic blood pressure (mmHg)	63.13±9.35	83.59±9.00	0.692ns
Serum creatinine Concentration (µmol/L)	107.57±25.48	87.01±12.88	0.0001***
Diabetes mellitus			
• Present	86 (76.8%)	0(0.0%)	0.0001***
• Absent	26 (23.2%)	138(100%)	
Left ventricular ejection Fraction (%)	58.72±10.40	57.64±11.01	0.427ns
Left ventricular ejection Contrast media used (ml)	81.03±36.53	59.41±20.89	0.0001***

Group A: Diabetes mellitus and/or eGFR <60 ml/mim/1.73 m<sup>2</sup>; Group B: Non-diabetic and/or eGFR ≤60 ml/mim/1.73 m<sup>2</sup>; Statistical analysis done by Chi-square test/Unpaired Student's 't' test; Plus-minus values are mean±SD for continuous variables; Values = Number (percent) for other variables; Ns = Not significant \*\*\*= Significant at P<0.001

Distribution of study subjects on the basis of the procedure followed in group A and group B patients. Coronary angiogram was done in 90 (80.4%) and in 134 (97.1%), and coronary angiogram plus percutaneous transluminal coronary angioplasty in 22 (19.6%) and 4 (2.9%) patients of group A and B, respectively (P<0.001). Distribution of procedures in between groups were found statistically significant (P<0.001) (Table 3).

Table 2: Distribution of Study Subjects By Risk Factors

Risk Factors	Group A (n=112)	Group B (n=138)	P Value
Impaired renal function (eGFR <60 ml/min/1.73m <sup>2</sup> )			
Present	57(50.9)	0(0.0%)	0.0001***
Absent	55 (49.1%)	138(100.0%)	
Diabetes mellitus			
Present	86(76.8%)	0(0.0%)	0.0001***
Absent	26(23.2%)	138(100.0%)	
Hypertension			
Present	53(47.3%)	52(37.7%)	0.125ns
Absent	59(52.7%)	86(62.3%)	
Dyslipidemia			
Present	57(50.9%)	3(2.2%)	0.0001***
Absent	55(49.1%)	135(97.8%)	
Previous myocardial Infarction			
Present	27(24.1%)	17(12.3%)	0.015*
Absent	85(75.9%)	121(87.7%)	

Statistical analysis done by Chi-square test; Ns = Not significant; \* = Significant at P<0.05; \*\*\* = Significant at P<0.001

Table 3: Distribution of study subject on the basis of procedure followed in this study

Procedure	Group A (n=112)	Group B (n=138)	P Value
Coronary angiogram	90(80.4%)	134(97.1%)	0.0001***
Coronary angiogram & PTCA	22(19.6%)	4(2.9%)	

PTCA=Percutaneous transluminal Coronary angioplasty; Statistical analysis done by Chi-square test; \*\*\* = Significant at P<0.001

Table 3: Distribution of study subject on the basis of procedure followed in this study

Investigations	Group A (n=112)	Group B (n=138)	Total (n=250)	P Value
Baseline serum Creatinine Concentration (µmol/L)				
≤140 (61-140)	101(90.2%)	138(100.0%)	239(95.6%)	0.0001***
(61-114)	55(49.11%)	0(0.0%)	55	
(115-140)	46(41.09%)	0(0.0%)	46	
>140 (141-223)	11(9.8%)	0(0.0%)	11(4.4%)	
Estimated GFR (MDRD formula) (ml/min/1.73 m <sup>2</sup> )				
>60 (34-59)	57(50.9%)	0(0.0%)	57(22.8%)	0.0001***
≥60 (60-111)	55(49.1%)	138(100.0%)	193(77.2%)	
Left ventricular Ejection fraction (%)				
<40	6(5.4%)	7(5.1%)	13(5.2%)	0.920 <sup>ns</sup>
≥40	106(94.6%)	131(94.9%)		

Statistical analysis done by Chi by Chi-square test; Ns = Not significant; \*\*\* = Significant at P<0.001

Pre-procedure selected investigations done of the study subjects. In group A (n=112) 101 patients (90.2%) had baseline serum creatinine ≤140µmol/L. In group B (n=138), all patients had baseline serum creatinine >140µmol/L (100%). The difference in baseline creatinine serum creatinine was found statistically significant (P<0.001). In group A 57 patients (50.9%) had eGFR <60ml/min/1.73m<sup>2</sup>, 55 patients (49.1%) had eGFR ≥60ml/min. The difference in estimated GFR was found statistically significant (P<0.001). Left ventricular ejection fraction <40% was present in 6 (5.4%), 7 (5.1%) in group A and B respectively, ≥40% in 106 (94.6%), 131 (94.9%) in group A and B respectively, were found non-significant (Table 4).

Pre- and post-procedure serum creatinine concentration and estimated GFR in the two study groups. Both pre-procedure (107.57±25.48 and 87.01±12.88 µmol/L) and post-procedure (123.23±35.81 and 95.71±14.93 µmol/L) mean (±SD) serum creatinine concentration was significantly higher (P<0.001) in group A patients compared to group B patients. In both group A and group B, post-procedure serum creatinine concentration was significantly higher (P<0.001) compared to pre-procedure. Similarly, both pre-procedure (66.74±19.74 and 84.52±14.44 ml/min/1.73 m<sup>2</sup>) and post-procedure (57.63±14.18 and 76.18±13.46 ml/min/1.73 m<sup>2</sup>) mean (±SD) estimated GFR was significantly higher (P<0.001) in group B patients compared to group A patients. In both group A and group B, post-procedure estimated GFR was significantly lower (P<0.001) compared to pre-procedure (Table 5).



Table 5: Comparison of pre-and post-procedure serum creatinine concentration and estimated GFR (Mean±SD)

Investigation	Group A (n=112)	Group B (n=138)	P Value
Serum creatinine Concentration (µmol/L)			
Pre-procedure	107.57±25.48	48.01±12.88	0.0001***
Post-procedure (at 48 hour)	123.23±35.81	95.71±14.93	0.0001***
P Value <sup>b</sup>	0.0001***	0.0001***	
Estimated GFR (ml/min/1.73m <sup>2</sup> )			
Pre-procedure	66.74±19.74	48.52±14.44	0.0001***
Post-procedure (at 48 hour)	57.63±17.18	76.18±13.46	0.0001***
P value <sup>b</sup>	0.0001***	0.0001***	

Statistical analysis done by aUnpaired Student's 't' test bPaired Student's 't' test; \*\*\* = Significant at P<0.001

The mean (±SD) peak increase in serum creatinine concentration at 48-hour post-procedure from pre-procedure values. The mean (±SD) peak increase in group A compared to group B was significantly higher (P<0.001) (15.66±15.44 and 8.69±9.81µmol/L) (Table 6).

Distribution of patients showing CIN. CIN developed in 21 (18.80%) patients in group A and 2 (1.4%) patients in group B. CIN was defined by increased in serum creatinine ≥25% of baseline or ≥44µmol/L. The result was statistically significant, P<0.001. Among 57 patients who had eGFR <60ml/min 19 (33.3%) developed CIN, all belonged to group A, 16 (19%) of the diabetic patients out of 86 developed CIN. Diabetic patients who had eGFR <60ml/min (n=30), 13 (43.3%)

Table 6: Peak increase in the serum creatinine concentration from baseline to 48 hour

Number of Group	Increase in serum creatinine Concentration (µmol/L)			P Value
	Mean±SD	Median	Range (Min-Max)	
Group A (n=112)	15.66±15.44	9.06	-3.99-65.00	0.0001***
Group B (n=138)	8.69±9.81	7.36	-3.72-97.00	

Statistical analysis done by unpaired Student's 't' test; \*\*\* = Significant at P<0.001

Table 7: Distribution of Patients Showing CIN Following Coronary Angiogram

Subgroups	Group A	Group B	Total	P Value
	No. (%)	No. (%)	No. (%)	
All patients	(n = 112)	(n=138)	(n=250)	0.0001***
CIN	21 (18.8)	2 (1.4)	23 (9.2)	
No CIN	91 (81.2)	136 (98.36)	227 (90.8)	
Patients with eGFR <60ml/min/1.73m <sup>2</sup>	(n = 57)	(n=0)	(n=57)	
CIN	19 (33.3)	0	19 (33.3)	
No CIN	38 (66.7)	0	38 (66.7)	
Patients with diabetes mellitus	(n = 86)	(n=0)	(n=86)	0.494ns
CIN	16 (19.0)	0	16 (19.0)	
No CIN	70 (81.0)	0	70 (81)	
Patients eGFR <60ml/min/1.73 m <sup>2</sup> Plus diabetes mellitus	(n = 30)	(n=0)	(n=30)	
CIN	13 (43.3)	0	13 (43.3)	
No CIN	17 (56.7)	0	17 (56.7)	

Statistical analysis done by Chi-square test; Ns = Not significant; \*\*\* = Significant at P<0.001; Analysis by patient subgroup

developed CIN. Among all patients (n=250), 23 developed CIN. Overall incidence was 9.2%. (Table 7)

### Discussion

This study demonstrates that CIN is a frequent complication after coronary angiogram and percutaneous coronary intervention and the incidence of CIN is higher especially in patient with selected risk factors in diabetes patients. In present study showed the mean age of the study patients in group A was 55.83±9.00 years, where as in group B it was 50.44±11.63 years. Highest number of patients (33.5%) was in the age group of 50-60 years. The mean age difference between two groups were found statistically significant ( $p<0.05$ ). A total of 206 patients were male (82.4%) and remaining 44 patients (17.6%) were female, male to female ratio was 4.68:1 In study of Mishima et al<sup>6</sup> observed that the mean values for age was found 62.3±12.2 years. Alrawahi et al<sup>7</sup> study showed 98(45.6%) patients were male in case and 121(33.8%) in control group.

Present study showed the mean body mass index (BMI) of the studied subjects were 23.74±2.82 and 23.17±2.19 kg/m<sup>2</sup> in group A and group B respectively, the mean body mass index were similar in two groups. Mishima et al<sup>6</sup> study observed the mean BMI was found 26.1±5.5 kg/m<sup>2</sup>. Present study revealed that the mean systolic blood pressure was 127.45±14.43 mmHg and 126.01±11.87 mmHg in group A and B respectively. Diastolic blood pressure was 63.13±9.35 mmHg in group A and B respectively. Systolic and diastolic blood pressure between two groups was found statistically not significant. The mean baseline serum creatinine was 107.57±25.48 µmol/L and 87.01±12.88 µmol/L in group A and B respectively. This difference was statistically significant ( $p<0.001$ ). Sharma et al<sup>8</sup> study showed the incidence of CIN in patients with preexisting impairment of renal function (baseline creatinine clearance <60 ml/min) was 45.45% vs. 4.04% in patients with baseline creatinine clearance ≥60 ml/min ( $p<0.001$ ). There was no difference regarding the amount of contrast agent administered between patients with different baseline creatinine clearance. Rihal et al<sup>4</sup> 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.

The mean volume of contrast media administered was 81.03±36.53 ml and 59.41±20.89 ml in group A and B respectively, difference was statistically significant ( $p<0.001$ ). Chao et al<sup>9</sup> study observed the volume of administered contrast medium can be another important factor regarding the risk of contrast-induced

AKI. Multiple studies have identified that the mean contrast volume is an independent predictor of CIN<sup>10-11</sup>. Circumstantial evidence has pointed out that intra-arterial injection of contrast medium carries a higher risk of contrast-induced AKI than intravenous use<sup>11</sup>. However, no mechanisms have been provided to explain this phenomenon<sup>12</sup>.

In different studies throughout the world shown number of risk factors for the development of CIN. Out of those pre existing impairment of renal function (eGFR < 60 ml/min/1.73m<sup>2</sup> BSA), diabetes mellitus, dyslipidemia and myocardial were identified as significant risk factors. Kim et al<sup>13</sup> report left ventricular ejection fraction less than 40%, GFR less than 60 ml/min/1.73 m<sup>2</sup>, serum reactive protein C more than 0.5 mg/dl and contrast volume consumption more than 250 cc as CIN's independent risk factors. Basal Scr level, shock, female gender, DM were CIN's risk factors in report of Ghani et al<sup>14</sup>. Renal underlying disease, hemodynamic instability, dyslipidemia, hypotension after angiography were risk factors for CIN in Valente et al<sup>15</sup> research.

In our study selected common risk factor revealed hypertension (42.0%) was the commonest followed by diabetes mellitus (34.4%), impaired renal function (22.8%). Among male and female distribution of risk factors were not significant. One hundred one (90.2%) patients had base line serum creatinine ≤140 µmol/L in group A and 138 (100%) in group B. Fifty seven (50.9%) patients had estimated GFR (<60 ml/min/1.73m<sup>2</sup> body surface Area) in group A and none in group B. The difference were statistically significant ( $p<0.001$ ). Six (5.4%) patients had low LVEF (<40%) in group A and 7(5.1%) in group. LVEF was not statistically significant ( $p=0.920$ ). Assareh et al<sup>16</sup> study observed CIN occurs in 4(2.2%) patients with GFR measured by 24-h Clcr method ≥ 60ml/min/1.73m<sup>2</sup> and in 23(32.4%) patients with GFR measured by 24-h Clcr method <60, also in 11(5.6%) cases with GFR estimated by CG equation ≥60 and in 16(27.1%) cases with GFR estimated by CG equation <60 (P values were <0.001 in both. Banda et al<sup>17</sup> 97% of study participants had normal baseline renal function based on eGFR, with the remaining having eGFR ranging from 42 to 59 mL/min/1.73 m<sup>2</sup> with no significant association with CIN. In a cohort study of 80.0% study participants with eGFR >60 mL/min/1.73 m<sup>2</sup>, Selistre et al<sup>18</sup> also reported no association between baseline eGFR and risk for CIN.

In present study observed pre procedure, mean serum creatinine concentration was 107.57±25.48 µmol/L in group A and 87.01±12.88 µmol/L in group B. Post procedure, mean serum creatinine concentration was

123.23±35.81  $\mu\text{mol/L}$  in group A and 95.71±14.93  $\mu\text{mol/L}$  in group B. The difference were statistically significant ( $p<0.001$ ). Mean serum creatinine concentration at post procedure was statistically significant ( $p<0.001$ ) within the group A compare with pre procedure. Mean serum creatinine concentration at post procedure was statistically significant ( $p<0.001$ ) within the group B compare with pre procedure. Shukla et al<sup>19</sup> the GFR calculated by sCr based formula was at baseline 45.77 (mL/min per 1.73m<sup>2</sup>), at 24 h 46.10 (mL/min per 1.73 m<sup>2</sup>) and at 48 h 25.17 (mL/ min per 1.73 m<sup>2</sup>). There was no significant difference between the baseline and 24 h but there is statistical difference between baseline and 48 h.

In current pre procedure, mean eGFR was 66.74±16.74 ml/min/1.73m<sup>2</sup> in group A and 84.52±14.44 ml/min/1.73m<sup>2</sup> in group B. Post procedure, mean eGFR was 57.63±17.18 ml/min/1.73m<sup>2</sup> in group A and 76.18±13.46 ml/min/1.73m<sup>2</sup> in group B. The difference were statistically significant ( $p<0.001$ ). Mean eGFR at post procedure was statistically significant ( $p<0.001$ ) within the group A compare with pre procedure. Mean eGFR at post procedure was statistically significant ( $p<0.001$ ) within the group B compare with pre procedure. Shukla et al<sup>19</sup> study showed the mean GFR calculated by sCyC based formula was significantly ( $p = 0.0026$ ) lower at 24 h after CM exposure. Similarly, the GFR calculated by the combined equation of sCyC and sCr was 36.73 and 26.37 ml at baseline and 24 h respectively showing a statistically significant difference.

In this study the peak mean increase in the serum creatinine from baseline after 48 hours of contrast administration was 15.66±15.44  $\mu\text{mol/L}$  in group A and 8.69±9.81  $\mu\text{mol/L}$  in group B. The peak increase in the serum creatinine concentration from baseline was found statistically significant ( $p=0.001$ ). This results implicates that patients having low eGFR (<60 ml/min/1.73 m<sup>2</sup>) with or without diabetes mellitus are vulnerable to develop renal impairment following radio contrast exposure. Among 250 patients total 23 patients developed CIN. Overall incidence was 9.2%. Out of 23, 21(18.8%) in group A and 2 (1.4%) in group B developed CIN. Difference of incidence of CIN in two groups was statistically significant ( $p=0.001$ ). Sub group analysis showed 19(33.3%) of the patients out of 57 who had eGFR <60 ml/min/1.73m<sup>2</sup> developed CIN. On the other hand 16(19%) of the diabetic patient out of total 86 developed CIN. Total 30 patients had both diabetes mellitus and impaired renal function 13 (43.3%) of them developed CIN. In a retrospective study Taliercio et al<sup>20</sup> reported 23% incidence of CIN (defined as rise

of serum creatinine >1 mg/dl) in azotemic (scr  $\geq$  2 mg/dl) patients, rate was 50% in diabetic-azotemic patients. McCullough et al<sup>12</sup> reported an incidence of CIN after PCI of 14.7%. Rudnick et al<sup>21</sup> reported that the incidence of CIN in diabetes with normal renal function was 9%, and in diabetes with pre existing renal impairment it was 19.7%, and 16% in patients with only preexisting renal impairment. Dangas et al<sup>22</sup> demonstrated that incidence of CIN following PCI was 13.1% and 19.2% in patients without CKD and with CKD respectively. Rihal et al<sup>4</sup> in a retrospective study demonstrate incidence of 3.3% (serum creatinine increase  $\geq$ 25% within 48 hours) following coronary intervention. Iakovou et al<sup>23</sup> reported incidence of 16.5% following PCI (serum creatinine increases  $\geq$  25% within 48 hours). Swartz et al<sup>24</sup> found incidence of 12% following coronary angiography. McCullough and Sandberg<sup>25</sup> found overall incidence of CIN was 15%.

In this study, in individuals of eGFR <60ml/min/1.73m<sup>2</sup> 19(33.3%) patients out of 57 developed CIN compared to 4(2.1%) out of 193 patient those who had eGFR  $\geq$ 60ml/min/1.73m<sup>2</sup> BSA. Taliercio et al<sup>20</sup> observed that patients with impaired renal function (eGFR<60ml/min/1.73m<sup>2</sup> BSA) undergoing CAG developed CIN in 23%. Gruberg et al<sup>26</sup> found that despite of giving pre procedure hydration CIN occur in 1/3 patient who underwent PCI and serum creatinine  $\geq$ 1.8 mg/dL, another study by same group in 2000 found that 37.0% patients developed CIN rise of serum creatinine  $\geq$ 25% who had impaired renal function.

Those incidence is almost consistent with our study, 33.3% incidence in patients with preexisting impaired renal function. In our study we detect diabetes mellitus as a statistically significant risk factor. 16 diabetic patient (18.6%) developed CIN. MuCullough et al<sup>12</sup> found incidence of CIN 19.5% among diabetics patients. Weisberg et al<sup>27</sup> demonstrated 16% incidence in diabetes. Incidence of CIN with DM varied from 5.7%<sup>28</sup> to 29.4%<sup>3</sup>. This present study revealed percentage of CIN is higher when diabetes mellitus and impaired renal function (eGFR < 60ml/min/1.73m<sup>2</sup>) exist ether. 13 patients out of 30(43.3%) developed CIN in those who has both diabetes mellitus and impaired renal function. Weisberg et al<sup>27</sup> demonstrated 43.0% rate of CIN in Azolemic-diabetics. Lautine et al<sup>29</sup> showed that incidence of CIN was 10% for nonazotemic patient vs 30.0% for azotemic patient, 16.0% for diabetic non azotemic patients as 38% for patients who were both diabetic and azotemic.

## Conclusion

CIN was significant developed in diabetes group than non-diabetes. Left ventricular ejection fraction and total volume of contrast media used was significantly higher in diabetes group than non diabetes group B patients.

## References

1. Agrawal R, Phogawae M, Agrawal RP. Prevalence of Nephropathy and Its Risk Factors in Type -2 Diabetes: A Tertiary Care Hospital Based Study. RUMS Journal of Health Sciences 2016;1(1):20-23
2. Evola S, Lunetta M, Macaione F, Fonte G, Milana G, Corrado E et al. Risk factors for contrast induced nephropathy: A study among Italian patients. Indian heart journal 2012;64:484-491
3. Nikolsky E, Mehran R, Turcot D, Aymong ED, Minhtz GS, Lasic Z. Impact of Chronic Kidney Disease on prognosis of patients with diabetes mellitus treated with percutaneous coronary intervention. Am j Cardial 2004; 94: 300-325
4. 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
5. Davidson CJ, Hlatky M, Morris KG. Cardiovascular and renal toxicity of a non-ionic radiographic contrast agent after cardiac catheterization: a prospective trial. Ann Intern Med 1989; 100: 119-124
6. Mishima T, Motoyama K, Imanishi Y, Hamamoto K, Nagata Y, Yamada S, et al. Decreased cortical thickness, as estimated by a newly developed ultrasound device, as a risk for vertebral fracture in type 2 diabetes mellitus patients with eGFR of less than 60 mL/min/1.73 m<sup>2</sup>. Osteoporos Int 2015;26:229-236
7. Alrawahi AH, Rizvi SGA, Al-Riami D, Al-Anqoodi Z. Prevalence and Risk Factors of Diabetic Nephropathy in Omani Type 2 Diabetics in Al-Dakhiliyah Region. Oman Medical Journal 2012;27(3):212-216
8. Sharma SK, Dubey L, Laudary S. Incidence and predictors of Contrast Induced Nephropathy after coronary intervention at College of Medical Sciences Teaching Hospital, Bharatpur. Nepalese Heart Journal 2014;11(1):3-11
9. Chao CT, Wu VC, Lin YH. Contrast-Induced Nephropathy in Coronary Angiography and Intervention. In What Should We Know About Prevented, Diagnostic, and Interventional Therapy in Coronary Artery Disease 2013. InTech.10
10. Lindsay J, Apple S, Pinnow EE, et al. Percutaneous coronary intervention-associated nephropathy foreshadows increased risk of late adverse events in patients with normal baseline serum creatinine. Catheterization and Cardiovascular Interventions 2003; 59:338-343
11. Mehran R, Aymong ED, Nikolsky E, et al. A simple risk score for prediction of contrast-induced nephropathy after percutaneous coronary intervention: Development and initial validation. Journal of the American College of Cardiology 2004; 44:1393-1399
12. McCullough PA. Contrast-Induced Acute Kidney Injury. J American Coll Cardio 2008; 51:1419-1428
13. Kim U, Kim YJ, Lee WJ, Lee SH, Hong GR, Park JS, et al. The estimated glomerular filtration rate with using the mayo clinic quadratic equation as a new predictor for developing contrast induced nephropathy in patients with angina pectoris. Korean Circ J 2008;38:301-4
14. Ghani AA, Tohamy KY. Risk score for contrast induced nephropathy following percutaneous coronary intervention. Saudi J Kidney Dis Transpl. 2009;20:240-5
15. Valente S, Lazzeri C, Giglioli C, Margheri M, Comeglio M, Nicolaci L, et al. Contrast-induced nephropathy in urgent coronary interventions. J Cardiovasc Med 2006;7:737-41
16. Assareh A, S, Ahmadzadeh A, Yadollahzadeh M, Nasehi N and Haybar H. Defining the at risk patients for contrast induced nephropathy after coronary angiography; 24-h urine creatinine versus Cockcroft-Gault equation or serum creatinine level. J Res Med Sci. 2012;17(9):859-864
17. Banda J, Duarte R, Dickens C, Dix-Peek T, Muteba M, Paget G et al. Risk factors and outcomes of contrast-induced nephropathy in hospitalized South Africans. S Afr Med J 2016;106(7):699-703
18. Selistre LD, Souza VC, Dubourg L, et al. Contrast induced nephropathy after computer tomography. J Bras Nefrol 2015;37(1):27-31
19. Shukla AN, Juneja M, Patel H, Shah KH, Konat A, Thakkar BM, Madan T, Prajapati J. Diagnostic accuracy of serum cystatin C for early recognition of contrast induced nephropathy in Western Indians undergoing cardiac catheterization. Indian Heart Journal 2017;69(3):311-5.
20. Taliercio CP, Vlietstra RE, Fisher LD, Burnett JC. Risks for renal dysfunction with cardiac angiography. Ann Intern Med. 1986;104:501-504
21. Rudnick MR, Goldfarb S, Wexler L, Ludbrook PA, Murphy MJ, Halpern EF, Hill JA, Winniford M, Cohen MB, VanFossen DB. Nephrotoxicity of ionic and nonionic contrast media in 1196 patients: a randomized trial. The Iohexol Cooperative Study. Kidney Int 1995;47:254-261
22. 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;95:13-19
23. Iakovou I, Dangas G, Mehran R, Lansky AJ, Ashby DT, Fahy M, Mintz GS, Kent KM, Pichard AD, Satler LF, Stone GW, Leon MB. Impact of gender on the incidence and outcome of contrast-induced nephropathy after percutaneous coronary intervention. J Invasive Cardiol. 2003;15(1):18-22
24. Swartz RD, Rubin JE, Leeming BW, et al: Renal failure following major angiography. Am J Med 1978;65:31-36
25. McCullough PA, Sandberg KR. Epidemiology of contrast-induced nephropathy. Rev Cardiovasc Med. 2003;4 Suppl 5:S3-9
26. Gruberg I, Mintz GS, Mehran R. The prognostic implications of further renal function deterioration within 48 hours of interventional coronary procedures in patients with preexistent chronic renal insufficiency. J Am Coll Cardiol 2000;36:1452-1548
27. Weisberg LS, Kurnik PB, Kurnick BRC. Risk of radio contrast nephropathy in patients with and without DM. Kidney Int 1994; 45: 259-265
28. Lasser EC, Yon SG, Berry CC. Reports on contrast media reactions: analysis of data from reports to the US food and Drug administration. Radiology 1997;203:605-610
29. Lautine EM, Freeman NJ, Schoenfeld AH. Radiocontrast associated renal dysfunction: Incidence and risk factors. AJR Am J Roengenol 1991;157:49-58