INCIDENCE OF CONTRAST-INDUCED NEPHROPATHY AND ITS CONTRIBUTING FACTORS AFTER CARDIAC CATHETERIZATION IN TYPE II DIABETIC PATIENTS

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ABSTRACT

Objective: To evaluate the incidence of contrast-induced nephropathy (CIN) and its contributing factors after cardiac catheterization in type II diabetic patients.

Study Design: Cross sectional study.

Place and Duration of Study: Choudhary Pervaiz Ellahi Institute of Cardiology (CPEIC), Multan for six months, from Aug 2016 to Mar 2017.

Material and Methods: This cross sectional study was conducted and completed in the department of cardiology Choudhary Pervaiz Ellahi Institute of Cardiology (CPEIC), Multan for six months (August 2016 to March 2017). Before start of the study ethical approval was obtained from hospital ethical board/committee; informed consent was taken from patients and their attendants after complete information and they were also ensured about their confidentiality. All collected data was aligned and entered in a computer software SPSS version 23.1 and data was analyzed. Mean ± SD values were calculated and presented for quantitative data variables like age, similarly frequency (percentages) were calculated and presented for qualitative variable data like gender. After stratification of data, student chi square test was used to see effect modification. A *p*-value ≤0.05 was considered as significant.

Results: A total number of 255 patients were included in the study. All patients were admitted for cardiac catheterization. Participants of the study were divided into two groups on the basis of CIN presence. Group A consisted of 210 patients who didn't develop contrast-induced nephropathy (CIN) after catheterization of coronary artery. Group B consisted of 45 patients of CIN after catheterization of coronary artery. It was found that incidence of CIN in diabetic patients with micro-albuminuria was 17.64% (n=45).

Conclusion: Observation of our study found that diabetic patients either with normal baseline creatinine are at an increased risk of developing CIN after angiography of coronary artery.

Keywords: Cardiac catheterization, Incidence, Diabetes mellitus type-II.

INTRODUCTION

Coronary angiography and percutaneous coronary intervention performed by using a contrast media causes nephropathy to named contrast induced nephropathy (CIN)[1]. Literature reported that incidence of this event is increasing rapidly. CIN can be defined as 25% increase in serum creatinine from baseline values within twenty four hours of procedure. CIN may lead the patient to renal dialysis or morbidity and mortality[2]. Usually this renal impairment resolves within three weeks but in some cases it may be permanent and patient may need permanent dialysis or chronic renal failure[3]. Incidence of CIN and its prolong effects can be reduced by using preventive measures such as pre procedural evaluation of sick patients and its management[4].

Many guidelines and scoring system have been designed to reduce the incidence of CIN and these guidelines are also applied in different cardiac centers[5]. Before procedure serum creatinine and glomerular filtration rate must be investigated because it's the main risk factor of CIN[6]. Many prophylactic therapies have been suggested like hemodialysis, N-acetylcysteine in high dose and good hydration with saline[7].

Diabetic patients are at higher risk of renal insult because their kidneys are predisposed to

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raised creatinine. Cardiac catheterization in diabetic patients is a challenge for cardiologists because these patients have higher chances of CIN. Some studies investigated the independent role of albuminuria as a predictor of CIN[8]. Causes of CIN after catheterization include prior renal failure, diabetes, age of patient, heart failure, hemodynamic changes during procedure and dose or volume of contrast. Prognosis after this event is very poor without dependency on predisposing factors[9,10].

The aim of this study was to investigate the frequency and risk factors for prognosis of CIN in diabetic type-II patients undergoing cardiac catheterization, and to give a prevention measure for this complication.

MATERIAL AND METHODS

This cross sectional study was conducted and completed in the department of cardiology Choudhary Pervaiz Elahi Institute of Cardiology (CPEIC), Multan for six months (August 2016 to March 2017). Before start of the study ethical approval was obtained from hospital ethical board/committee; informed consent was taken from patients and their attendants after complete information and they were also assured about their confidentiality. Sample size was calculated with WHO sample size calculator using following figures: CI 95%, absolute precision required is 0.05 and proportion of outcome variable (p) 21%. Sampling technique used was non probability consecutive sampling. Cardiac catheterization or coronary angiography was performed according to set protocol, serum creatinine and other baseline investigations were obtained before two weeks before and 24 hours after coronary angiography. Patients with creatinine level more than 1.5 mg/dl, previous MI, history of contrast exposure, heart failure and cardiogenic shock were excluded from the study. History about any nephrotoxic medicine like Angiotensin converting enzyme inhibitors, Angiotensin receptors blockers, acyclovir, NSAIDs and sulfa antibiotics was obtained and recorded. Patients BMI, pulse, blood pressure and peripheral pulses were assessed. Creatinine clearance was also calculated with following formula:

Cr Clearance = (140-Age) x Body weight (kg) x 0.86 (If female)/(72xCr mg/dl)

Angiography was done with standard protocol; femoral artery access was obtained by Seldinger's method. Prophylaxis of intravenous CIN given and all patients were preloaded with N/S solution 1ml/kg/h before 6-12 hours of catheterization and 12-24 hours after catheterization. Intravenous fluid was given unless contraindicated such as fluid overload. All patients received 300 mg aspirin before PCI and continue at dose of 100 mg per day. Contrast was given during PCI (ultravist non-ionic). Oral antihyperglycemic drugs (metformin) were stopped before and after two days of PCI and serum creatinine level was monitored till second day of post PCI and patients were labelled as group A no CIN (in which CIN was absent) and group B CIN (in which CIN developed).

All collected data was aligned and entered in a computer software SPSS version 23.1 and data was analyzed. Mean ± SD values were calculated and presented for quantitative data variables like age, urea, creatinine, GFR, EF and LVMI, similarly frequencies (percentages) were calculated and presented for qualitative variable data like gender and albumin/Cr. Independent t-test was applied in demographic characteristics while paired t-test was applied in outcome variables. After stratification of data student chi square test was used to see effect modification. A *p*-value ≤0.05 was considered as significant.

RESULTS

A total number of 255 (100%) patients were included in the study. All patients were admitted for cardiac catheterization. Participants of the study were divided into two groups on the basis of presence of CIN: Group A consists of 210 patients who didn't develop contrast-induced nephropathy (CIN) after catheterization of coronary artery. Group B consist of 45 patients of CIN after catheterization of coronary artery. It

was found that incidence of CIN in diabetic patients with micro-albuminuria 17.64% (n=45).

The mean age, blood urea, creatinine (pre), GFR (pre), Alb/Cr, EF and LVMI in group A was 55.40 ± 3.17 years, 30.12 ± 2.92 (mg/dL), 1.01 ± 0.198 (mg/dL), 90.35 ± 5.42 (mL/min/1.73m^2), 320.34 ± 107.30 (mg/g), 55.47 ± 5.70 and 130.35 ± 5.45 (g/m^2) respectively, while the mean age, blood urea, creatinine (pre), GFR (pre), Alb/Cr, EF and LVMI in group B was 61.22 ± 5.48 years, 36.42 ± 4.28 (mg/dL), 1.52 ± 0.12 (mg/dL), 80.88 ± 4.64 (mL/min/1.73m^2), 614.76 ± 218.20 (mg/g), 39.91 ± 3.36 and 1125 ± 7.13 (g/m^2) respectively. There were 77.1% (n=162) males and 22.9% (n=48) females in group A, and 66.7% (n=30) males and 33.3% (n=15) females in group B. No association was found between gender in groups (X^2 = 2.186 DF = 1, *p*-value=0.139). The patients, who developed CIN, used ACEI/ARBs more frequently 97.8% (n=44) vs 42.4% (n=89). Association was found between ACEI/ARBs in groups (X^2 = 45.575 DF = 1, *p*-value=0.001) (table-I, II, III & IV).

Albumin in urine was noted in 51.4% (n=108) patients, 22.9% (n=48) patients, 21.4% (n=45) and 4.3% (n=9) patients respectively in group A. While Albumin in urine was noted as 37.8% (n=17) patients, 20% (n=9) patients, 17.8% (n=8) and 24.4% (n=11) patients in group B. Association was found between ACEI/ARBs in groups (X^2 = 20.98 DF = 1, *p*-value=0.000) (table-IV).

There was significant difference regarding to age (*p*=0.000), blood urea (*p*=0.000), creatinine (pre) (*p*=0.000), GFR (pre) (*p*=0.000), Alb/Cr (*p*=0.000), EF (*p*=0.000) and LVMI (*p*=0.000) among CIN versus no CIN group (table-I, II, II & IV). It was also observed that pre- and post-procedure serum blood urea, creatinine and

Table-I: Demographic variables in both groups (CIN and No CIN).

Variable	Group A No CIN (n=210)	Group B CIN (n=45)	Test of Sig.
Gender	M=77.1%, F=22.9%	M=66.7%, F=33.3%	X^2 = 2.186 *p* = 0.139
Age	55.40 ± 3.17 years	61.22 ± 5.48 years	T = -9.623 *P* = 0.001
ACEI OR ARBS	Used = 57.6%, Un-used = 42.4%	Used = 97.8% Un-used = 2.2%	X^2 = 45.575 *p* = 0.001

Table-II: Serum urea, creatinine and GFR in both groups.

Variable	Group A No CIN (n=210)	Group B CIN (n=45)	Test of Sig.
Urea (pre)	30.12 ± 2.92 (mg/dL)	36.42 ± 4.28 (mg/dL)	t=11.98 *p*=0.001
Serum Creatinine (pre)	1.01 ± 0.198 (mg/dL)	1.52 ± 0.12 (mg/dL)	t=16.60 *p*=0.001
GFR (pre)	90.35 ± 5.42 (mL/min/1.73m^2)	80.88 ± 4.64 (mL/min/1.73m^2)	t=10.88 *p*=0.001

Table-III: Ejection fraction and LVMI in both groups.

Variable	Group A No CIN (n=210)	Group B CIN (n=45)	Test of Sig.
EF	55.47 ± 5.70	39.91 ± 3.36	t=17.62 *p*=0.001
LVMI	130.35 ± 5.45 (g/m^2)	1125. ± 7.13 (g/m^2)	t=18.70 *p*=0.001

Glomerular Filtration Rate (GFR) were also significantly different in CIN versus no CIN group (table-V).

DISCUSSION

In our study we focused on incidence of contrast induced nephropathy after use of contrast in diabetic patients who required cardiac catheterization. In our study we observe that incidence of CIN in diabetic patients with microalbuminuria is 17.64% (n=45). In another study conducted by Sany et al[11] on incidence of CIN in type II diabetic patients and found 21.5% of contrast induced nephropathy in diabetic patients. Microalbuminuria was found 17% of these patients, 26% patients have micro-albuminuria among CIN patients. In his study he suggested that diabetic patients with normal baseline creatinine are at higher risk of having CIN when coronary angiography was performed, most patients of his study were older age category who were develop high serum creatinine and CIN.

In a study conducted in 2009 by Abe et al [12]) reported that incidence of CIN labeled as an

Table-IV: Urinary albumin and albumin/Cr ratio in both groups.

Variable	Group A No CIN (n=210)	Group B CIN (n=45)	Test of Sig.
Albumin in urine			
0	51.4% (n=108)	37.7% (n=17)	$X^2 = 20.98$ $p = 0.001$
1	22.8% (n=48)	20% (n=9)	
2	21.4% (n=45)	17.7% (n=8)	
3	4.2% (n=9)	24.4% (n=11)	
Alb/Cr	320.34 ± 107.30 (mg/g)	614.76 ± 218.20 (mg/g)	t = 13.43 $p = 0.001$

Table-V: Post procedure group wise comparison of all values.

Variable	Pre (n=255)	Post (n=255)	Test of Sig.
Blood urea (mg/dL)			
No CIN			
Mean ± S.D	31.04 ± 2.67	36.49 ± 2.51	t=-23.40 p=0.001
CIN			
Mean ± S.D	35.95 ± 3.49	66.61 ± 9.85	t=-46.07 p=0.001
Serum creatinine (mg/dL)			
No CIN			
Mean ± S.D	1.19 ± 0.10	1.54 ± 1.07	t=-5.15 p=0.001
CIN			
Mean ± S.D	1.10 ± 0.21	2.05 ± 0.93	t=-15.51 p=0.001
GFR mL/min per 1.73m^2			
No CIN			
Mean ± S.D	89.80 ± 5.53	88.38 ± 5.59	t=2.88 p=0.004
CIN			
Mean ± S.D	45.17 ± 2.11	43.21 ± 3.07	t=10.23 p=0.001

absolute increase in serum creatinine ≥0.5mg/dl, or ≥25% increase from baseline after cardiac catheterization. He conducted study on different populations. He also reported that high volume contrast use can cause CIN especially in female gender, diabetic and underweight patients. In our study we also concluded that high volume contrast can cause nephropathy in diabetic patients.

Nassir et al[13] reported in his study that contrast media can cause marked increase in serum creatinine in type II diabetic patients. In his trial he reported that diabetic patients are three times more prone to develop CIN than non-diabetic patients. Results of his study are also comparable with our findings.

Shoukat et al[14] also conducted similar study and reported that rate of CIN in the general population is about 6%, this ratio is quit less than those patients in which PCI was done and who were diabetic.This study is also comparable with our results.

Rihal et al[15] conducted a similar study and found the incidence of CIN after PCI 3.3% in general population. He reported 20% increase in CIN in patients with cardiac diseases.

Au et al[16] conducted a study on this topic and reported that avoidance from incidence of CIN is meaningfull. Best way to prevent CIN after cardiac catheterization is adequate assessment of renal parameters and preload with isotonic solution before use of contrast and cardiac catheterization.

Trivedi et al[17] reported in his study that, when we concern about development of CIN, fluid resuscitation alone is not sufficient while Solomon et al[18] demonstrated the good efficacy of 0.45% Saline over IV normal saline plus Furosemide in patients of renal insufficiency.

Rahman et al[19] conducted a study on this topic and found 155 (63.3%) diabetic patients, among these diabetic patients 59 (24.08%). He concluded that diabetic patients are at high risk of contrast induced nephropathy after cardiac catheterization. Results of this study are comparable with our results. Similar results were reported by Zaytseva et al[20], Wang et al[21] and Rear et al[22] in their studies. These studies were also in favour of our study.

CONCLUSION

This study reveals that diabetic patients, despite having a normal baseline creatinine are at an increased risk of developing CIN post-coronary angiography.

CONFLICT OF INTEREST

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

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