

FREQUENCY OF ACUTE RENAL FAILURE AFTER CARDIAC CATHETERIZATION AND PERCUTANEOUS INTERVENTION

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ABSTRACT

Objectives: To observe the frequency of acute renal failure after percutaneous coronary intervention and cardiac catheterization.

Patients and Methods: This is a retrospective study, comprising 200 patients undergoing cardiac catheterization and percutaneous coronary intervention at Aga Khan University Hospital. Patient aged above 18 years and not on regular dialysis was included in the study. Proper history and physical examination was carried out on every patient and properly hydrated with 50 ml/hr of normal saline except in-patient with congestive cardiac failure. Serum creatinine and blood urea nitrogen was checked before procedure and 24 hrs after procedure. Amount and type of contrast media was noted.

Main Outcome Measures: Serum creatinine after 24 hrs is compared with base line creatinine. Rise in serum creatinine of > 0.5 mg/dl, is labeled as contrast induced nephropathy.

Result: Out of a total 199 patients (1 was excluded as he was on regular dialysis) 16 (8%) were found to have contrast induced nephropathy. Congestive cardiac failure and renal insufficiency were found to have significant association for development of contrast induced nephropathy.

Conclusion: Contrast induced nephropathy is not a frequent complication. Contrast (ionic or nonionic) is safe to use in patients undergoing percutaneous coronary intervention and cardiac catheterization even in presence of risk factors. However caution should be used in patient with congestive cardiac failure and antecedent renal insufficiency.

KEY WORDS: Contrast induced nephropathy, coronary angiography.

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INTRODUCTION

Contrast nephropathy (CN) is an important cause of iatrogenic acute renal failure and carries significant risk for affected patients. CN is the third leading cause of acute renal failure in hospitalized patients.¹

Cardiac catheterization and per cutaneous coronary intervention is a relatively safe procedure but has a well-defined risk of morbidity and mortality. The use of contrast media in this procedure has been documented to be responsible for some risk.²

Although acute CN requiring dialysis occurs in less than 1% of per cutaneous coronary intervention population, lesser degrees of renal dysfunction occur in approximately 15 %.¹ Despite advances in the per cutaneous care, including stents and advanced anti thrombotics, the risk of CN remains unchanged over last decade. CN was defined as an increase in the serum creatinine level of at least 44 micmol/lit (0.5 mg/dl) above the baseline value with in 48 hrs of exposure to contrast agent.³ Serum creatinine generally

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peaks at 3–5 days and return to baseline value by 7–10 days.⁴⁻⁶ The acute renal failure is non-oliguric in most cases.⁷⁻⁸ Urinalysis often reveals granular cast, tubular epithelial cell, and minimal proteinuria, but in many cases may be entirely bland. Most, but not all inpatients exhibit low fractional excretion of sodium.

CN appear to be the result of a synergistic combination of direct renal tubular epithelial cell toxicity and renal medullary ischemia.⁹ Direct cytotoxicity in CN is suggested by histologic changes of cell injury and enzymuria after contrast administration.¹⁰ The nature of the contrast, associated ions, concentration, and concomitant hypoxia are all important to the degree of cellular damage, while the osmolality of the solution seems to be of secondary importance.¹¹

Various agents have been used for prevention of CN. Barrette and Carlisle performed a meta analysis of all the randomized trials available before the end of 1991, comparing the nephrotoxicity of high and low osmolality contrast in humans by serial measurements of GFR or serum creatinine. Pooling the p value from the trials suggested a reduction in nephrotoxicity with low osmolality media, which was of borderline statistical significance (P=0.02). The administration of intravenous fluid has long been used to reduce the likelihood of CN for high-risk patients. The rationale for this is that giving fluids before the study may counter an osmotic diuresis result from the contrast. Some benefits of this approach have been suggested by uncontrolled and retrospective studies,^{12,13} and also by a randomized, controlled trial of deliberate hydration versus no intervention for the prevention of CN. In a recent study by Solomon et al.¹⁴ showed that 11% of patient with chronic renal failure developed CN despite saline administration before hand. Most recently, Stevens et al.¹⁵ reported the result of a randomized trial in which high risk patients undergoing cardiac catheterization were treated with a combination of fluid therapy, furosemide, mannitol and low dose dopamine and

compared with a control group treated with hydration alone. Although the authors concluded that this regimen of forced diuresis provided a modest benefit in preventing CN, there is no statistical difference in the mean rise in serum creatinine at 48 hours between the groups. There is accumulating evidence that reactive oxygen species have a role in the renal damage caused by contrast agents.¹⁶⁻¹⁸

The objective of this study was to determine the frequency of acute renal failure after cardiac catheterization and per-cutaneous intervention. This study will be helpful in establishing the proportion of patient having acute renal failure after cardiac catheterization and intervention in our community.

PATIENTS AND METHODS

This is a retrospective study. Two hundred patients above the age of 18 years admitted at Aga Khan University and Hospital and underwent diagnostic or therapeutic cardiac catheterization with minimum stay of one day was enrolled in the study, out of which one

Table-I: Relationship of Contrast Induced Nephropathy and Risk Factors. (N = 199)

<i>Risk Factors</i>	<i>Total #</i>	<i>Rise in Creatinine <0.5 Mg/Dl</i>	<i>Rise in Creatinine 0.5 Mg/Dl</i>
Diabetes Mellitus	80(40.20%)	69(86.25%)	11(13.75%)
Hypertension	93(46.73%)	85(91.39%)	8(8.60%)
Proteinuria	32(16.08%)	25(78.12%)	7(21.87%)
Congestive Cardiac Failure	9(4.52%)	4(44.44%)	5(55.55%)
Renal Insufficiency	27(13.56%)	21(77.77%)	6(22.22%)
Ionic Contrast	43(21.60%)	41(95.34%)	2(4.65%)
Nonionic Contrast	156(78.39%)	142(91.02%)	14(8.97%)
Stable Angina			
Unstable Angina			
Angina NSTEMI	149(74.87%)	141(94.36%)	8(5.36%)
ST elevated MI	50(25.12%)	42(84.0%)	8(16.0%)
Dose of Contrast <150 MI	124(62.31%)	115(92.74%)	9(7.25%)
Dose of Contrast >150 MI	75(37.68%)	68(90.66%)	7(9.33%)
Angiotensin Converting Enzyme Inhibitor	47(23.61%)	42(89.36%)	5(10.63%)

was excluded because he was on regular dialysis. Other exclusion criteria included patients having DIC, coagulopathy, bleeding disorder and patients having ARF secondary to other pathologies (obstruction). Careful history and examination was done to assess co-morbid condition such as diabetes mellitus, hypertension, hyperlipidemia, previous exposure to contrast, drugs and hydration status. Most of the patients were started on I/ V fluids for at least 3-4 hrs before procedure except for patients in congestive cardiac failure. Base line serum creatinine and blood urea nitrogen was also checked along with serum creatinine after 24 hours and at 48 hours if any rise was detected in serum creatinine at 24 hours. Type and amount of contrast was also noted. All the information was recorded in a coded data collection form. Contrast nephropathy was defined as an increase in the serum creatinine level of at least 0.5mg/dl above the base line value at 24 or 48 hrs after exposure to contrast agent.

RESULTS

One hundred ninety nine patients were included in this study. One hundred forty two (71.35%) were male and 57(28.6%) were female. The age range in the study was between 31 years to 87 years with mean of 57 years \pm 10 years. (Table-I). Rise in serum creatinine of ≥ 0.5 mg/dl (CN) after contrast exposure was found in 16 patients (8%). Rise in serum creatinine of $<0.5 - >0.2$ mg/dl after contrast exposure was found in 16 patient (8%). 61 patient (30.7%) had < 0.2 rise in serum creatinine. No rise in serum creatinine was observed in 106 patients (53.5%). (Table-II) (Fig-I)

Table-II: Frequency of Rise in Creatinine

	Frequency	Percent	Cumulative Percent
No rise in creatinine	106	53.3 %	53.3 %
Rise of 0.1 to 0.2	61	30.7 %	83.9 %
Rise of >0.2 but <0.5	16	8.0 %	92.0 %
Rise of 0.5 and >0.5	16	8.0 %	100.0 %
Total	199	100%	

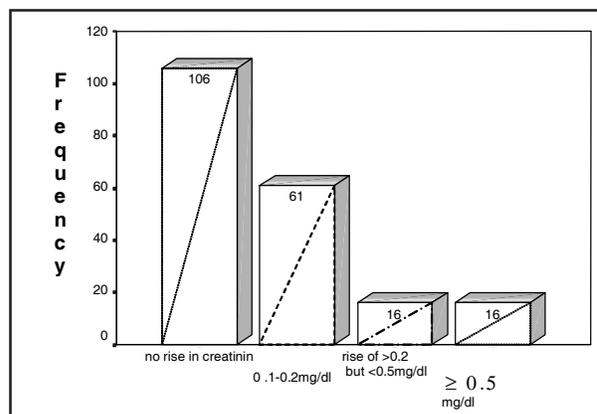


Fig-1: Frequency of Rise in Creatinine (N=199)

Congestive cardiac failure was present in 9 patients and CN was observed in 5 patients (55.55%). Diabetes mellitus was found in 80 patients and CN was observed in 11 patients (13.75%). Hypertension was found in 93 patients and CN was observed in 8 patients (8.60%).

Nonionic contrast was given in 156 patients and 14 had CN (8.95 %). 43 patients had ionic contrast and CN was observed in 2 (4.65 %). Amount of contrast used was between 30 ml and 500 ml. 75 patients had ≥ 150 ml and 7 patients (9.33 %) had CN. One hundred seventy two patients had SCr <1.3 mg/dl pre-exposure and two had CN (1.16%). Renal insufficiency (SCr ≥ 1.3 mg/dl) was present in 27 patients and 6 had CN (22.22 %). Fifty patients subjected to coronary angiography/ interventional had ST segment elevated myocardial infarction and rest had stable angina, unstable angina or non ST elevation myocardial infarction (NSTEMI). 16 % of patients had CN in former group vs. 5.36 % in later group. Out of 16 patients having CN, six patients (37.5%) had ≥ 3 risk factors for development of CN, seven patients (43.75%) had two risk factors and three patients (18.75%) had no risk factors. None of the patient having CN had oliguria and maximum post exposure S.Cr was 5.1mg/dl at 48hrs.

DISCUSSION

The influence of changing the serum creatinine criteria for inclusion into the nephrotoxicity ranges can be appreciated from

the report of Parfery et al who stated that, with use of an increase of 50% from base line, the incidence of nephrotoxicity in a group of patients with renal impairment was 4%. Where as reducing the limit to an increase of >25% in serum creatinine level tripled the incidence to 12.9%.¹⁹ A recent epidemiological study reported a rate of 14.5% in series of approximately 1800 consecutive patients undergoing cardiac procedure.¹⁷

Once contrast induced nephropathy occurs, it is associated with a markedly higher in hospital and long term mortality following PCI. This was also stated by Rihal et al from the Mayo clinic database, reviewing predictors of death after PCI. Various international studies have identified risk factors for contrast nephropathy, which include pre existing renal impairment, diabetes mellitus, congestive cardiac failure, dehydration, ionic contrast, high dose of contrast, concurrent use of nephrotoxic medication, non steroidal anti inflammatory drugs and angiotensin converting enzyme inhibitors. In our study population diabetes, Hypertension, use of angiotensin converting enzyme inhibitor and high dose of contrast did not appear to be associated with increased frequency of contrast nephropathy. Congestive cardiac failure and preexisting renal impairment were associated with increase frequency of contrast induced nephropathy. Increase frequency of CN occur in patients receiving non-ionic contrast medium (9%) Vs. patients receiving ionic contrast medium (4%). In our catheterization lab osmolar load of non-ionic is high than the ionic contrast medium that is why we didn't appreciate any beneficial effect of non-ionic contrast agent. ST – segment elevation MI was associated with comparatively high frequency of CN comparing with rest of ischemic heart disease spectrum. This probably is the reflection of more hemodynamic disturbance associated with former. Presence of two or more risk factors was associated with high frequency of contrast nephropathy which again emphasises the fact that addition of another risk factor in

presence of one risk factor greatly enhance the risk of CN. None of the patient required dialysis for contrast nephropathy. Our data on 199 consecutive patients who had radio contrast studies indicate that acute renal failure is not a frequent complication, and our result is comparable to previous prospective studies.

CONCLUSION

Contrast induced nephropathy is not a frequent complication. Contrast (ionic or nonionic) is safe to use in patients undergoing percutaneous coronary intervention and cardiac catheterization even in presence of risk factors. However caution should be used in patient with congestive cardiac failure and antecedent renal insufficiency.

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