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Original Article

Incidence of contrast induced acute kidney injury in patients undergoing percutaneous coronary intervention in North Indian population

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ABSTRACT

Objectives: The aim of the study was to assess the incidence,clinical predictors and outcome of Contrast induced nephropathy (CIN) after percutaneous coronary interventions (PCI) in coronary artery disease patients in overburdened north india tertiary care center
Background: CIN incidence varies in different centers because of several risk factors.CIN is associated with high morbidity and mortality.PCI in CAD patients is now a day care procedure, so in overburdened tertiary care centers in india,CIN development remain undetected because of short duration of hospital stay.
Methods: In 300 consequtive coronary artery diseased patients treated with coronary angioplasty in our catheterization laboratory.we measured serum creatinine and GFR at baseline and serum creatnine at 48 h and at 7 day.CIN was defined as a rise in serum creatinine level more than 0.5 mg/dl or 25% of baseline at 48 h.
Results: CIN developed in 32 patients(10.66%).in univariative analysis 10 factors were significant for development of CIN.in multivariate analysis contrast dose (odds ratio 1.048,95% CI),number of stents, reduced ejection fraction,diabetes mellitus,and use of ace inhibitors were significantly associated with CIN.
Conclusion: In the era of PCI for CAD patients, CIN is a frequent complication, even in patients with normal renal function, and is associated with a more complicated in-hospital course. In overburdened Indian hospitals, CIN is frequently underreported because of day care procedures and early discharge. We found a new factor for development of CIN.CIN was associated with $\geq$3 number of stents deployment, contrast volume more than 250 ml, EF $\leq$30% and in diabetic patients. There was no effect of isoosmolar contrast from low osmolar contrast.

1. Introduction

Contrast-induced nephropathy (CIN) is a possible complication of coronary diagnostic and interventional procedures. CIN is responsible for approximately 10% of all iatrogenic renal insufficiency and is the third most common cause of hospital-acquired acute renal failure, after impaired renal perfusion and nephrotoxic medications.[1] Its development has been associated with increased in hospital and long-term morbidity and mortality, prolonged hospitalization, and long-term renal impairment. [1–7] Chronic renal insufficiency, diabetes mellitus, congestive heart failure, intravascular volume depletion, and the use of a large amount of contrast agent are considered important predisposing factors.[5,7,8] Multiple strategies have been demonstrated to be successful for prophylaxis of CIN, including adequate hydration, minimization of contrast dose, and the use of iso-osmolar or certain low osmolar contrast media. [9–10] Several conditions may contribute to renal injury in this setting. Among them, hypotension or even shock, a large volume of contrast media, and the impossibility of starting a renal prophylactic therapy are the factors most likely involved. Renal insufficiency and AMI represent a high-risk combination for development of CIN in patient undergoing PCI. [11] Moreover, other clinical observations have shown that renal dysfunction is an independent risk factor for death in CAD patients. [12–14]

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The real incidence of CIN, in unselected population is less than 3% but in high risk patients like renal impaired patient,diabetics, the incidence is as high as 50% [15]. Generally, CIN follows a benign course, and persistent renal impairment and dialysis dependency are rare. [16] Hence,it seems that Incidence of CIN varies in different population due to different underlying risk factors. The purpose of this prospective study was to determine the incidence, the clinical predictors, and the clinical consequences of CIN development in an unselected population of consecutive patients undergoing PCI in Coronary Artery Disease patients.

1.1. Study population

Three hundred consecutive, eligible patients scheduled for coronary angioplasty at the SMS Hospital between July 2014 to june 2015 were considered for enrolment for the study. The exclusion criteria for study were a glomerular filtration rate (GFR) < 30 ml/min, patients on regular dialysis, a history of disseminated intravascular coagulation, coagulopathy, bleeding disorders, or acute renal failure secondary to other pathologies such as obstruction. Histories of multiple myeloma or pulmonary edema, pregnancy, administration of dopamine, mannitol or fenoldopam, and recent exposure to radiographic contrast media (within 2 days of the study) were also considered as exclusion criteria. In addition, patients who denied research access to their medical records were excluded as well.

The sample size was calculated to be 225 subjects at 95% confidence interval and 4% absolute allowable error, assuming incidence of CIN among patient undergoing PCI to be 10%,hence for study purpose 300 subjects were taken.

1.2. Study protocol

The study protocol was approved by the local Ethics Committee and written informed consent was obtained from all the patients. Patient, disease, and procedural data were recorded prospectively, and a careful history and examination were done to assess comorbid conditions such as diabetes mellitus and hypertension, previous exposure to contrast media and drugs. Procedural variables were also recorded, including the number of diseased coronary vessels, treated coronary vessels, number of stents used, type of contrast agent and contrast dye amount. The study participants received the nonionic, low-osmolar monomeric agent iohexol(omnipaque) or the nonionic, iso-osmolar dimeric contrast medium iodixanol (Visipaque) for coronary angioplasty. Iodixanol was used in patients in which baseline serum creatinine was more than 1.5 miligram per deciliter. All information was recorded in a coded data collection form. Some patients, who were having baseline serum creatinine more than 1.5 were hydrated with IV fluids (normal saline) before the procedure, except for patients with congestive cardiac failure. Intravenous hydration consisted of 1 ml normal saline per kilogram of body weight per hour, which was started 3–12 h before contrast agent injection and continued for 12 h after the PCI. All routine investigations were done before PCI including serum creatinine. Serum creatinine was measured the day before and 48 h after and 7 day after contrast agent administration. At baseline, estimated GFR was calculated by cockgraft–Gault formula. Left ventricular ejection fraction was estimated by echocardiography, if available, and the type and amount of the contrast medium were recorded. Choice of the contrast medium depended on the patient's comorbidity and on the serum creatinine levels before the procedure. The end point of the study was the peak increase of serum creatinine as a measure of CIN during hospitalization for angioplasty or Acute renal failure requiring emergency renal replacement therapy (hemofiltration or hemodialysis).

According to our clinical protocol, emergency renal replacement therapy (hemofiltration or hemodialysis) was performed if there was oligouria for more than 48 h, despite the administration of more than 1 g of intravenous furosemide per 24 h. Blood transfusion was initiated in case of hemoglobin reduction below 8.0 g/l.

1.3. Definitions

CIN was defined by an increase in creatinine of >0.5 mg/dl or 25% of the initial value at 48 hours [1]. Pre-existing renal disease was defined as previous history of renal artery stenosis, acute renal failure, glomerulonephritis, obstruction, nephrotic syndrome, or nephrectomy irrespective of baseline creatinine levels or glomerular filtration rate; anemia, as hematocrit value <39% for men and < 36% for women; hypotension, as systolic blood pressure <90 mm Hg requiring inotropic support with medications; significant blood loss, as haemoglobin reduction below 8.0 g/dl, blood transfusion, or significant hematoma/arterial pseudoaneurysm requiring intervention.

The creatinine clearance was estimated using the Cockcroft–Gault method: 140-age (years) × weight (kg)/72 × serum creatinine (mg/dl) { × 0.85 for female subjects}.[17] Renal function was then categorized according to the stages set by the National Kidney Foundation with creatinine clearance ≥90 ml/min considered normal, 60–89 ml/min considered mildly impaired, and <60 ml/min considered at least moderately impaired. [18]

1.4. Stastical analysis

The results were reported as the means ± standard deviation for the quantitative variables and percentages for the categorical variables. The groups were compared using the χ 2 test if required for the categorical variables. Unpaired student *t*-test was used for analysis of quantative variables. Predictors exhibiting a statistically significant relation with CIN in univariate analyses were taken for multivariate logistic regression analysis to investigate their independence as predictors. Odds Ratios and 95% Confidence Interval were calculated. p values of ≤ 0.05 were considered statistically significant. All statistical analyses were performed using SPSS version 13.0 (SPSS Inc., Chicago, Ill., USA).

2. Results

2.1. Patients

CIN at 48 h post-procedure occurred in 10.66% of our derivation study population (32 0f 300 patients). Baseline demographic, clinical and angiographic characteristics, as well as main procedural data are listed in Table 1. Overall the mean age was 57.96 years and 9.3% were more than 70 year of age and there were 10% females. There was an increasing trend of development of CIN in older populations. In younger age group (<50year) no one developed CIN. However, the results were probably due to 95% patients in this age group were with normal serum creatinine. 13.34% had LVEF < 30%. Diabetes Mellitus was found in 20% cases (60 patients).24.6% were hypertensive. 70% had myocardial infraction in form of NSTEMI and STEMI however there was no difference between these patients .The mean baseline serum creatinine level was 1.16 mg/dl (SD 0.36 mg/dl) non CIN patients and 1.94 mg/dl in CIN patients (SD 0.25 mg/dl) whereas 72 patient (24%) out of 300 of study population had baseline serum creatinine levels >1.5 mg/dl. 56 of these 72 patients had no history of pre-existingrenal insufficiency; this suggests that the increased Cr value was likely due to changes in renal hemodynamics associated

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