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ORIGINAL ARTICLE

Predicting contrast induced nephropathy post coronary intervention: A prospective cohort study

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KEYWORDS

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Abstract *Objective:* The purpose of our study was to assess the incidence and predictors of contrast induced nephropathy (CIN) in unselected patients undergoing coronary intervention either coronary angiography (CA) or percutaneous coronary interventions (PCI), at Assiut university hospitals.
Background: CIN is a frequent, potentially lethal complication after coronary intervention. It is the 3rd most common cause of hospital-acquired acute renal failure.
Patients and methods: This is an observational prospective cohort study. Two hundred consecutive patients between December 2011 and August 2012 underwent CA and PCI were enrolled in the study. Blood samples were collected at baseline and 3 days after interventions. All patients were followed up for 2 weeks for major adverse events.
Results: CIN was observed in 23 (11.5%) patients. According to Mehran risk score, 84.5% of our patients had low risk for CIN, 15.5% had moderate risk for CIN, and no one had high risk score. Multivariate logistic regression analysis of predictors for CIN, showed that the use of high osmolar contrast media (CM) (Telebrix) was associated with 4 times higher incidence of CIN than the use of low osmolar CM (Ultravest) (OR = 4.07; 95% CI = 1.1–15.1). None of our patients had clinical signs or symptoms of acute renal failure, or required haemodialysis at 2 weeks of follow up.
Conclusion: Although most of our study population was at low risk, the incidence of CIN was relatively high due to the use of high osmolar CM. Further studies are needed for cost effectiveness in light of negligible clinical impact.

1. Introduction

Contrast induced nephropathy (CIN) represents a significant adverse event of contrast media (CM) administration, leading to acute kidney impairment and, subsequently, to increased hospital morbidity and mortality. [1] In the general population, the incidence of CIN has been reported to be <2%, but it can

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reach 20–30% in high-risk groups such as the elderly patients and patients with diabetes mellitus (DM), chronic kidney disease (CKD), or congestive heart failure (CHF).[2]

Reduction in renal perfusion and toxic effects on the tubular cells caused by the direct and indirect effects of CM on the kidneys are generally recognized as important mechanisms for the development of CIN. Furthermore, contrast exposure causes a certain degree of imbalance between increased renal vasoconstriction and decreased vasodilatation; this leads to a decrease in renal blood flow and contraction of the afferent glomerular arteriole, as well as renal ischemia and cell necrosis.[3] Oxygen radicals released by the ischemia–reperfusion contribute to not only renal damage but also the apoptosis of the renal tubular epithelial cells.

In the majority of studies, the term CIN is widely defined as an absolute rise in the serum creatinine (SCr) level by at least 0.5 mg/dL (44 mmol/L) or an increase in SCr level of >25% over baseline within 3 days following intravascular CM exposure.[4] The clinical presentation varies from asymptomatic to symptomatic renal failure and death. Thus, it is important to identify risk factors prior to the administration of a contrast agent.

Many individual risk factors have been reported for the development of CIN. Although the combination of two or more risk factors is rather common in daily practice, the cumulative risk of several variables on renal function was still unknown.[5–7] This showed the need for global assessment of the impact of these variables on the development of CIN by using a simple risk scoring system that can be easily conducted clinically.[8]

The purpose of our study was to assess the incidence and predictors of CIN in unselected patients undergoing coronary intervention either coronary angiography (CA) or percutaneous coronary interventions (PCI), at Assiut university hospitals (AUH).

2. Methods

2.1. Study design and setting

We conducted a prospective cohort study at the catheterization laboratory of AUH between December 2011 and August 2012. The study protocol was reviewed and approved by the institutional review committee, and all patients granted their informed consent to be included in the study.

2.2. Study population

Two hundred consecutive unselected patients scheduled to undergo a coronary procedure either CA or PCI were enrolled in the study. Patients were excluded from the study if they had end stage renal disease and on regular dialysis and those with other contrast exposure within one week or less from the index procedure.

The CM used was left to the discretion of the interventional cardiologist. Main CM used in our laboratory is Telebrix® [produced by Guerbet Asia Pacific and composed of Meglumine; it is a High-Osmolar, Ionic CM (HOCM)]. Ultravist® [produced by Bayer Healthcare and composed of iopromide; it is a Low Osmolar, Non-Ionic CM (LOCM)] is another option used only in selected high risk patients mainly due to financial constrain.

2.3. Data collection and measurements

The demographic and clinical data were prospectively collected by our research team using a standardized ''peri-procedural datasheet''. The patients were interviewed on the day of the procedure and 2 weeks after.

2.3.1. Baseline characteristics

Demographic data (age, sex, length, and weight), risk factors, previous treatment and type and indication for intervention were collected. Body mass index (BMI) was calculated based on height and weight (kg/m^2).

2.3.2. Cardiac catheterization

CA and PCI were performed in accordance with established clinical practice using standard diagnostic and guide catheters, wires, balloon catheters, and stents via the femoral approach. The amount of contrast media administered was decided by the interventional cardiologist. Patients were treated in accordance with the guidelines of the American College of Cardiology/American Heart Association.[9]

2.3.3. Risk factors

All patients were admitted to the hospital one day before cardiac catheterization. Risk stratification for development of CIN was calculated for all patients using the Mehran risk score.[7] That risk score includes hypotension (5 points, if systolic blood pressure <80 mm Hg for at least 1 h requiring inotropic support), use of intra-aortic balloon pump (5 points), congestive heart failure (5 points, if class III/IV by New York Heart Association classification or history of pulmonary edema), age (4 points, if >75 years), anemia (3 points, if hematocrit <39% for men and <36% for women), diabetes mellitus (3 points), contrast media volume (1 point per 100 mL), estimated glomerular filtration rate (GFR; GFR in mL/min per 1.73 m^2; 2 points, if GFR 60–40; 4 points, if GFR 40–20; 6 points, if GFR < 20). A risk score of <6, 6–10, 11–16, and >16 indicates a risk for CIN of 7.5%, 14%, 26%, and 57%, respectively [7].

2.3.4. CIN prophylaxis

We also used the ''peri-procedural datasheet'' to assess whether or not patients were instructed to increase oral fluid intake, discontinue potential nephrotoxic medication/metformin or received prophylactic intravenous hydration in accordance with the hospital CIN prevention protocol. That protocol indicates that patients who need prophylactic intravenous hydration should receive 0.9% sodium chloride (NaCl), 3–4 ml/kg/h for four hours before and after intervention. In patients with severe kidney disease or congestive heart failure administration of 1 ml/kg/h for 12 h is recommended before and after intervention. The final decision to actually apply prevention measures using intravenous hydration in patients at risk was left to the discretion of the treating physician.

2.3.5. Kidney function

Serum creatinine concentrations and eGFR were determined at hospital admission (prior to the procedure), and on days 1, 2, and 3 after the procedure. The changes of serum