Assessment of Estimated GFR and Clinical Predictors of Contrast Induced Nephropathy among Diabetic Patients Undergoing Cardiac Catheterization

Thesis

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List of Abbreviations

Abb.	Full term
ACC	American college of cardiology
AHA	American heart association
ANP	Atrial natriuretic peptide
ARF	Acute renal failure
BL	Baseline value
CA	Coronary angiography
Ca^{2+}	Calcium ion
CHF	Congestive heart failure
CI	Confidence interval
CIN	Contrast-induced nephropathy
Cm	Centimeter
CO_2	Carbon dioxide gas
Cr Cl	Creatinine clearance level
CT	Computed tomography
DBP	Diastolic blood pressure
dL	Deciliter
DM	Diabetes mellitus
ECG	Electrocardiogram
eGFR	Estimated glomerular filtration rate
GFR	Glomerular filtration rate
Grp	Group
HOCM	High osmolar contrast medium
HS	Highly significant

Ht	Height
IABP	Intra-aortic balloon pump counterpulsion
in	Inch
IOCM	Iso osmolar contrast medium
kg	Kilogram
LD_{50}	Lethal dose 50
LOCM	Low osmolar contrast medium
mg	Milligram
mmHg	Millimeter mercury
Na	Sodium
NAC	N-Acetyl cysteine
NO	Nitric oxide
NS	Non-significant
NSAIDs	Non steroidal anti-inflammatory drugs
O_2	Oxygen
p	Probability of chance
PCI	Percutaneous coronary intervention
S.Cr	Serum creatinine level
SBP	Systolic blood pressure
SD	Standard deviation
VOL	Volume of contrast media administered
Wt	Weight
%	Percentage
µmol	Micro-mole

INTRODUCTION

Contrast-induced nephropathy is a leading cause of morbidity and mortality in high-risk patients undergoing any procedure involving the use of radiographic contrast media ***(Cavusoglu et al., 2004)***.

Subjects who develop this complication have higher rates of mortality, longer hospital stays and worse long-term outcomes ***(Mehran et al., 2004)***.

The occurrence of contrast-induced nephropathy is related to the number of the patients' co-existing clinical risk factors. Among the many risk factors, pre-existing renal impairment, advancing age, the presence of diabetes mellitus as well as the volume and type of contrast agent administered are the most important ***(Cavusoglu et al., 2004)***.

The precise pathophysiologic mechanisms responsible for the development of contrast-induced nephropathy are complex and incompletely understood. At present, the only available tool for reducing the risk of developing contrast-induced nephropathy is prevention. This can be achieved by means of adequate peri-procedural hydration, using N-acetyl cysteine as well as the selection of low osmolar or iso-osmolar contrast agents in the least amount possible. Other agents are still being tested for this purpose as well ***(Harjai et al., 2008)***.

AIM OF THE WORK

To study different risk predictors of contrast induced nephropathy, among diabetic patients with normal serum creatinine undergoing cardiac catheterization.

To asses the volume of contrast in relation to eGFR as a predictor of CIN and the cut off value that can be used as a risk predictor for occurrence of CIN.

To follow up the occurrence of major adverse cardiac events (mortality, reinfarction, stroke, target vessel revascularization) during one month of hospital discharge.

Chapter (1)

CONTRAST-INDUCED NEPHROPATHY

Historical background

Nearly seventy years ago, Osborne et al first reported the imaging of the urinary tract using iodinated contrast material ***(Osborne et al., 1983)***. Over the past 30 years, there has been a marked increase in diagnostic and interventional procedures in which iodinated contrast was used ***(Gleeson and Bulugahapitiya, 2004)***.

The structure of radiocontrast agents has been modified over the last several decades, yielding compounds with significantly less chemotoxicity. Unfortunately, the administration of even the newest radiocontrast agents may cause nephrotoxicity ***(Gleeson and Bulugahapitiya, 2004)***.

Contrast-induced nephropathy has become a significant source of hospital morbidity and mortality with the ever-increasing use of iodinated contrast media in diagnostic imaging and interventional procedures such as coronary angiography. It ranks third amongst the causes of hospital-acquired acute renal failure, after surgery and hypotension ***(Barrett, 1994)***.

Unfortunately, it is frequently the high risk patients; particularly those with preexisting renal insufficiency and diabetes mellitus; which are encountered by the cardiovascular and interventional radiologist ***(Gleeson and Bulugahapitiya, 2004)***.

Definitions

Defining contrast-induced nephropathy has proven to be quite challenging and many studies have put forward various suggestions ***(Barrett, 1994)***.

Lautin et al. (1991) used six separate definitions with criteria ranging from an increase in serum creatinine level of more than 0.3 mg/dL to an increase of 2.0 mg/dL or more and found that the more restrictive higher cut-off point to be less sensitive for predicting incidences of contrast-related renal dysfunction.

A new definition of contrast nephropathy in patients undergoing percutaneous coronary intervention was recently proposed by ***Harjai et al. (2008).*** This tripartite definition classifies contrast nephropathy as:

Grade 0 (serum creatinine increase <25% above baseline and <0.5 mg/dL above baseline).

Grade 1 (serum creatinine increase ≥25% above baseline and <0.5 mg/dL above baseline).

Grade 2 (serum creatinine increase ≥0.5 mg/dL above baseline).

This classification is prognostic of long-term outcomes of patients after percutaneous coronary intervention. Patients with grade 2 nephropathy had the worst outcome while those with grade 0 nephropathy had the best outcome on long-term follow-up ***(Harjai et al., 2008).***

Hence contrast-induced nephropathy has become most commonly defined as "impairment of renal function occurring within 48 hours after administration of radiographic contrast media which is maintained for 2 to 5 days. It is manifested by an absolute increase in the serum creatinine level of at least 0.5mg/dL (44.2 μmol/L), or by a relative increase of at least 25% over the baseline value in the absence of another cause ***(Kolonko et al., 1998)***.

This definition may in part account for the large number of cases reported showing only transient elevations of serum creatinine levels or at least elevations that do not require dialysis. Although this large number has led to questioning of the clinical relevance of such rises, these subtle changes have been shown to be associated with significant morbidity rates and, in addition, may help to identify those with borderline renal function who may be at risk of developing fulminant renal failure in the future ***(Lautin et al., 1991)***.

Ideally, the impairment of renal function should be measured by serial creatinine clearance, but because this step may be neither practical nor cost-effective in many centers,