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Frequency and Risk Factors of Contrast-Induced Nephropathy after Contrast Procedures in a Nigerian Tertiary Centre

Fréquence et Facteurs de risque de néphropathie induite par les produits de contraste après examens dans un Centre Tertiaire du Nigeria

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ABSTRACT

Contrast-induced nephropathy (CIN) is a significant yet underestimated problem in clinical practice. The increasing use of contrast media in diagnostic and interventional procedures over the last 30years has resulted in CIN becoming the third leading cause of hospital-acquired acute renal failure (ARF) in developed countries. Despite this, there is still a paucity of data on the incidence of CIN following intravenous contrast media especially in developing countries.

The goals of this study were to determine the frequency and risk factors of CIN amongst patients receiving intravenous contrast in a tertiary health institution.

This is a hospital-based prospective observational study. One hundred and eighty (180) consenting patients were recruited consecutively over a 6-month period. Venous blood and urine were collected for haematocrit, serum urea, electrolytes and creatinine estimation and urinalysis, before contrast exposure and up to 72 hours post-exposure.

The frequency of CIN was 35.9% and one patient required haemodialysis. Baseline renal insufficiency, anaemia and age >55 years were significant risk factors for CIN and predictive of CIN in univariate but not multivariate analysis. WAJM 2013; 32(1): 19–25.

Keywords: **Contrast-induced Nephropathy, risk factors, frequency, contrast media.**

RÉSUMÉ

La néphropathie Induite par les produits de Contrate (NIC) est significative mais toutefois, elle demeure un problème sous estimé dans la pratique clinique. L'accroissement de l'usage de produits de contraste à visée diagnostique et interventionnelle au cours des 30 dernières années a positionné les NIC comme troisième cause d'hospitalisation pour insuffisance rénale aigue (IRA) acquise à l'hôpital dans les pays développés. En dépit de ce constat, il demeure une rareté de données sur l'incidence de NIC spécialement dans les pays en développement.

Le but de cette étude étaient de déterminer la fréquence et les facteurs de risque des NIC chez des patients recevant un produit de contraste par voie intraveineuse dans une structure de santé tertiaire.

Il s'agit d'une étude d'observation prospective centrée sur un hôpital. Cent quatre vingt patients concentant étaient successivement recrutés sur une période de 6 mois. Des échantillons de sang veineux ont été collectés pour doser l'hématocrite, l'azotémie, la créatininémie, l'ionogramme sanguin et des examens d'urines ont été faits avant l'exposition au produit de contraste et jusqu'à 72 heures aprés l'exposition.

La fréquence de NIC était de 35.9% et un patient avait nécessité une hémodialyse. Une insuffisance rénale pré existante, une anémie et un âge>55 ans étaient des facteurs de risque significatifs et prédicteurs de NIC en analyse univariée et non en analyse multivariée. **WAJM 2013; 32(1): 19–25.**

Mots Clés: Néphropathie Induite par un Produit de Contraste, facteurs de risque, fréquence, produit de contraste

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Abbreviations: ARF, ACUTE RENAL FAILURE; **BMI,** BODY MASS INDEX; **CCF,** CONGESTIVE CARDIAC FAILURE; **CIN,** Contrast-induced nephropathy; **CIN(+),** Contrast-induced nephropathy present; **CIN(-),** Contrast-induced nephropathy absent; **CM,** Contrast media; **CT,** Computerised tomography; **EDTA,** Ethylene diamine tetraacetic acid; **ESRD,** End stage renal disease; **ESUR,** European Society of Urogenital Radiology; **GFR,** Glomerular filtration rate; **HTN,** Hypertension; **IVU,** Intravenous urography; **JNC,** Joint national committee; **OR,** Odds ratio; **PCV,** Packed cell volume; **SD,** Standard deviation; **UBTH,** University of Benin Teaching Hospital; **WC,** Waist Circumference; **WHR,** Waist hip ratio.

INTRODUCTION

Contrast-induced nephropathy (CIN) is a significant yet underestimated problem in clinical practice. The increasing use of contrast media in diagnostic and interventional procedures over the last 30 years has resulted in CIN becoming the third leading cause of hospital-acquired acute renal failure (ARF) after hypotension, and surgery.[1–3] It accounts for 12% of all cases of hospital-acquired ARF in the developed countries.[3] The contrast media guideline of the European Society of Urogenital Radiology (ESUR) defines CIN as impairment in renal function (an increase in serum creatinine by > 25% or 44.2μmol/L) within 3 days after intravascular administration of contrast medium, without an alternative aetiology.[4] In most cases of CIN there is an asymptomatic, non-oliguric rise of serum creatinine within 24hours; however, in more severe cases, the creatinine concentration may not peak until 5–10 days and the increase may be associated with oliguria.[5] It may occasionally progress to end-stage renal failure.

The reported incidence of CIN varies widely across the literature, depending on the patient population studied and the baseline risk factors.[6] An overall incidence of CIN following intra-arterial contrast injection in the general population was reported to be 0.6–2.3% in 1997.[7] However, the frequency of CIN is said to have decreased over the past decade from a general incidence of about15% to 7%[8] owing to a greater awareness of the problem, better risk prevention measures and improved iodinated contrast media with less toxicity.[9]

Although the incidence of CIN is low in patients with normal renal function, its prevalence can be much higher in several patient subsets e.g. diabetics, patients with existing renal insufficiency.[6, 10–12] The rate of CIN reported in studies that included patients with pre-existing renal dysfunction or diabetes mellitus in whom a standard hydration protocol was not administered is between 12% and 26%.[1,6,13–15] Lodhia et al reported an incidence of 25% among patients with decompensated liver cirrhosis.[12] In patients with multiple risk markers, the incidence of CIN can rise to 50% or more.[16]

There are no comprehensive data on the incidence of CIN following intravenous administration of contrast agents (CT imaging and similar procedures).[17] Majority of existing studies are on patients undergoing interventional cardiology procedures and findings for coronary intervention patients cannot suffice for the other population undergoing CT imaging and other procedures. More recently, a study by Mitchell, *et al* reported an incidence of 11% among outpatients who undergo contrast enhanced CT,[18] and a high mortality rate among patients with CIN compared to those without.

There is generally a paucity of data on the incidence of CIN in Nigeria and indeed so in Africa. In Nigeria, Unuigbe et al in 2007 reported a case of acute renal failure following use of intravenous CM (diatrizoate derivative) in an elderly hypertensive diabetic within 24 hours of undergoing intravenous urography.[19] This report confirmed that CIN does occur among our patients in the developing world and more research data in that field was needed.

This study was aimed at determining the frequency and risk factors of CIN amongst patients receiving intravenous contrast in a tertiary health institution.

SUBJECTS

The study population consisted of inpatients and outpatients aged 18years and above, who were referred to the UBTH Radiology Department for contrast-enhanced computer tomography or intravenous urography (IVU). A total of 180 consenting subjects who met the inclusion criteria were recruited consecutively and studied.

The exclusion criteria included subjects below 18 years, failure to obtain consent, subjects with cardiogenic shock, ESRD/ on maintenance haemodialysis, uncontrolled hyperthyroidism/ thyroid malignancies, New-York Heart Association class IV Congestive Cardiac Failure (CCF), history of hypersensitivity to contrast in the past or exposure to contrast in the last 24–48 hours, nursing/ pregnant subjects, subjects in whom decision was made to withhold contrast injection during the procedure e.g patients with intracranial haemorrhage.

SUBJECTS, MATERIALS AND METHODS

The study was conducted at the University of Benin Teaching Hospital (UBTH), a tertiary hospital in the South-South region of Nigeria, serving as the main referral hospital in Edo, Delta, Kogi, and Ondo states. It was a prospective observational study spanning a period of 6 months, September 2009 to March 2010. Ethical clearance was obtained from the Ethics and Research committee of the hospital for the study.

After obtaining informed consent from eligible patients or their relatives, information on their socio-demographic characteristics, clinical history, blood pressure (mmHg) and anthropometric measurements were obtained and results of laboratory investigations were collated.

Five mls of venous blood was collected by venepuncture (before, 24, 48 and 72 hours after exposure to contrast) into lithium heparin bottles for serum electrolyte, urea and creatinine estimation. Creatinine estimation was done using the modified Jaffe's method.[20] Two mls of blood was also collected into EDTA bottles and plain bottles for haematocrit and serum albumin estimation respectively. Blood sample for packed cell volume was centrifuged by haematocrit centrifuge (Hawksley) and readings were obtained using micro-haematocrit reader (Hawksley micro-haematocrit reader). Random urine samples were collected into plain bottles and tested using the 10 parameter multistix (Medi-Test Combi 10® SGL by Macherey-Nagel) for urinary abnormalities before and up to 72 hours after exposure to contrast for evidence of urinary abnormalities such as proteinuria, haematuria.

Eighty milliliters of iopamidol, a low osmolar non-ionic iodinated CM was injected intravenously for all patients who had CT scans while 50mls of diatrizoate (urograffin), a high osmolar CM was used for IVU.

The following definitions and criteria were used to evaluate the physical and biochemical parameters:

- CIN was regarded as elevation of serum creatinine by ≥25%, 24–72hrs after exposure to contrast.
- Hypertension (HTN) – reported history of HTN, HTN medication usage or repeated blood pressure reading of ³ 140/90 mmHg as measured with a mercury sphygmomanometer, according to JNC VII guidelines.[21]
- Hypotension – repeated systolic blood pressure reading of <100mmHg.
- Smoking status – reported history of daily smoking was regarded as a current smoker.[22] Overall consumption was then evaluated in pack years.
- Cardiac failure was defined as self report of diagnosis of CCF by a doctor or other health personnel.
- Diabetes was defined as self report of diagnosis by a doctor or other health personnel, or use of oral glucose lowering agent/insulin.
- Obesity was regarded as BMI >30kg/m^2, waist circumference >102cm in males and 88cm in females (WHR > 0.7 in females and > 0.9 in males).
- Anaemia was regarded as PCV< 30%.[23]
- Hypoalbuminaemia was regarded as serum albumin < 3.5mg/dl.
- Renal insufficiency was regarded as baseline serum creatinine of ≥ 1.5mg/dl or GFR <60mls/min.
- Abnormal serum urea was defined as serum urea concentration > 40mg/dl.

Data entry and management were performed using statistical software package version 16(SPSS, inc., Chicago, IL). The socio-demographic characteristics, health status and biochemical measurements (serum electrolytes, protein and creatinine; urine abnormalities, haematocrit) of the study population are presented as tables. Data are presented as mean ± SD for continuous variables and as frequency and percentages for categorical variables. The main statistical analysis involved the estimation of the incidence of CIN for the study population; the incidence rates of CIN according to age, sex and risk factors such as hypotension, anaemia, diabetes mellitus, renal impairment, hypoalbuminaemia and exposure to specific drugs were analysed.

For nominal data, the Chi-square test was used to determine the difference between groups while for numerical data the Student t-test was used. Yates correction was used in cases where a cell had value less than 5 and Fishers exact test was used when the expected value was less than 10 and marked as †. All p value < 0.05 was regarded as significant and marked with asterix within tables for ease of recognition. The unadjusted odds ratio (OR) between baseline serum creatinine and the outcome of CIN was determined by simple logistic regression analysis.

RESULTS

One hundred and eighty patients were recruited for the study, however blood samples for more than one day was obtained in 142 patients. Of the 142 patients 17 (12%) were outpatients while 125(88.0%) were inpatients /emergency patients. One hundred and thirty three patients were referred for CT scan while only 9 had IVU. Figure 1 shows the various indications for CT scan among the patients studied.

Age of subjects ranged between 18–85 years. There were more males than females with a sex ratio of 1:1.6 (F: M).9.9% of subjects were known hypertensives, 7.0% known diabetics, 22.5% and 4.2% respectively had family history of hypertension and diabetes; 2.1% had a history of diagnosed renal disease, 26.1% used alcohol while 6.3% smoked.

Frequency of CIN among Patients Studied

Fifty-one patients (35.9%) developed CIN and of this only one patient required haemodialysis with renal function returning to normal baseline within 2 weeks. One of the 9(11.1%) patients who had IVU and 50 out of 133 (37.5%) who had CT scan developed CIN (p=0.150†). Out of the 17 outpatients 3(17.6%) developed CIN, while 48 of 125 (38.4%) inpatients had CIN (p=0.160).

There were no statistically significant difference between the anthropometric measurements of CIN+ and CI-patients (Table 1).

Serum creatinine levels of subjects ranged between 0.30mg- 4.60mg/dl, with mean of 0.88±0.46mg/dl. The frequency of CIN increased significantly with increasing baseline serum creatinine levels (Table 2).

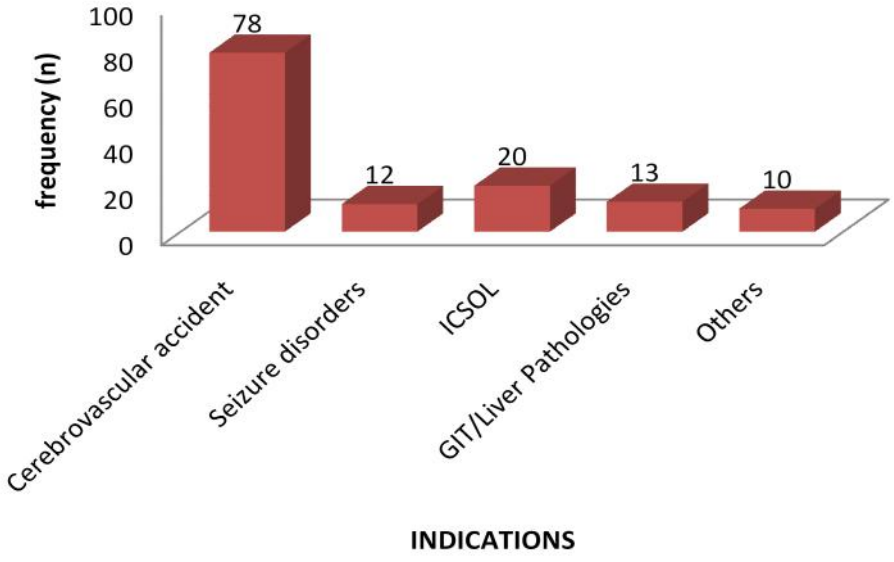

Fig. 1: Indications for contrast CT Scan

ICSOL, Intracranial space occupying lesion; GIT, Gastrointestinal; Others, dizziness, lung carcinoma, toxoplasmosis, hydrocephalus, uterine fibroid

Table 1: Comparison of Anthropometric Indices for CIN (+) and CIN (-) Patients

Parameters	ALLMean ± SD	CIN(+)Mean ± SD	CIN(-)Mean ±SD	Mean Difference 95% CI	P value
BMI (kg/m^2)	26.40 ± 2.69	26.23 ± 2.37	26.49 ± 2.86	–0.3(–1.1,2.2)	0.576
Weight (kg)	74.76 ± 8.49	74.27 ± 7.41	75.04 ± 9.05	–0.7(–3.7,0.7)	0.606
WC(cm)	79.18 ± 12.95	79.10 ± 10.4	78.9 ± 15.0	0.2 (–4.5, 4.9)	0.934
WHR	0.87 ± 0.04	0.88 ± 0.04	0.87 ± 0.04	0.0 (–0.0,0.0)	0.346

CI, Confidence interval; CIN, Contrast-induced nephropathy; SD, Standard deviation; WC, Waist circumference; WHR, Waist hip ratio.

Table 2: Distribution of Baseline Serum Creatinine Concentrations among CIN (+) and CIN (–) Subjects

Serum Creatinine (mg/dl)	All n(%)	CIN(+)n(%)	CIN (–)n(%)
0.30 – 0.89	81 (100.0)	24 (29.6)	57 (62.6)
0.90 – 1.49	53 (100.0)	21 (39.6)	32 (35.2)
1.50 – 2.09	6 (100.0)	4 (66.6)	2 (2.2)
>2.10	2 (100.0)	2 (100.0)	0 (0.0)
Total	**142 (100.0)**	**51 (35.9)**	**91 (64.1)**

¡^{2} = *8.174, df=3, p=0.043*

Table 3: The Association between Presumptive Risk Factors and CIN

Risk Factors	Total	CIN(+)n(%)	CIN(-)n(%)	P Value or Fishers Exact
Sex				
Female	55 (100.0)	20 (36.3)	35 (63.7)	0.929
Male	87 (100.0)	31 (35.6)	56 (64.4)	
Age				
<55yrs	84 (100.0)	24 (28.6)	60 (71.4)	0.028*
>55yrs	58 (100.0)	27 (46.5)	31 (53.5)	
SBP				
> 100mmHg	135 (100.0)	48 (35.5)	87 (64.5)	0.702†
< 100mmHg	7 (100.0)	3 (42.8)	4 (57.2)	
Anaemia				
Yes	25 (100.0)	14 (54.5)	11 (45.5)	0.021*
No	117 (100.0)	37 (32.5)	80 (67.5)	
Diabetes				
Yes	10 (100.0)	5 (50.0)	5 (50.0)	
No	132 (100.0)	46 (34.8)	86 (65.2)	0.499 †
Creatinine				
≥ 1.5mg/dl	8 (100.0)	6 (75.0)	2 (25.0)	0.025*†
< 1.5 mg/dl	134 (100.0)	45 (33.5)	89 (66.5)	
GFR				
< 60ml/minGFR	12 (100.0)	8 (66.7)	4 (33.3)	0.028*†
≥ 60ml/min	130 (100.0)	43 (33.1)	87 (66.9)	
Serum albumin				
<3.5g/dl	70 (100.0)	28 (40.0)	42 (60.0)	0.317
>3.5g/dl	72 (100.0)	23 (31.9)	49 (68.1)	
Serum urea				
>40mg/dl	50 (100.0)	23 (46.0)	27 (54.0)	0.065
<40mg/dl	92 (100.0)	28 (30.4)	64 (69.6)	

*† = Fishers exact test; *= Significant p value*

Table 4: Predictors of CIN

Risk Factors	OR	CI (95%)	P	OR	CI (95%)	P
Simple Logistic Regression				**Multiple Logistic Regression**		
GFR ≥60ml/min	4.05	1.15 – 14.19	0.029	0.52	0.12 – 2.30	0.390
Cr ≥ 1.5mg/dl	5.90	1.15 – 30.50	0.033	4.13	0.67 – 25.40	0.126
PCV ≥ 30%	2.75	1.14 – 6.64	0.024	0.92	0.31 – 2.71	0.884
Age ≥ 55years	2.18	1.08 – 4.37	0.029	0.49	0.24 – 1.03	0.060

OR= odds ratio, CI= confidence interval, GFR=glomerular filtration rate, Cr= serum creatinine, PCV= packed cell volume.

Risk Factors of Contrast-induced Nephropathy

Table 3 shows the association between the presumptive risk factors and CIN. A higher proportion of females than males had CIN. Diabetics and hypoalbuminaemic patients had a higher occurrence of CIN compared to those without these conditions. These differences were however not statistically significant. There were a significantly higher proportion of patients with serum creatinine ≥ 1.5mg/dl or GFR<60ml/min developing CIN compared to those with values < 1.5mg/dl or GFR >60ml/min; similarly anaemic and patients aged >55yrs had significantly increased risk of CIN. Baseline creatinine, GFR<60ml/min, anaemia and age ≥55yrs were predictive of CIN in univariate analysis but not in multivariate analysis (Table 4).

DISCUSSION

This study has shown interestingly that the frequency of CIN is not as low aspreviously perceived. The frequency of 35.9% is much higher when compared to some previously recorded data like 3.3% by Richal, *et al*[24] 1.4% by Mueller *et al*[25] and 6.6% by Barret; *et al*[26] though these studies investigated CIN after intra-arterial contrast procedures. The frequency of 17.6% obtained among the outpatient subset in our study however compares with the reported incidence of 11% by Mitchell et al from their study of CIN after contrast enhanced CT in the outpatient setting.[18]

Clinical studies on CIN usually measure a surrogate endpoint for acute renal failure since the incidence of patient outcome events is so low.[17] There is generally a lack of uniformity of definitions of surrogate CIN and thus comparison between different CIN trials is difficult. There are variations on the definition of CIN with regard to both the increase in serum creatinine (for example increases of ≥25%, ≥20%, ≥50%, ≥100% or absolute increases e.g ≥0.5mg/dl, ≥0.3mg/dl ≥1.0mg/dl have been used) and the time frame, which uses measurements of the increase in serum creatinine within 48, at 48, at 72, between 48 and 72 hrs, etc.[17, 27] The incidence of surrogate CIN varies greatly within the same patient groups, depending on the definition employed. A lower incidence is usually found when definition including the stricter absolute increase of ≥0.5mg/dl is applied. The incidence can increase substantially if the definition of relative increase of ≥25% is applied and even more when a combination of definitions is used.[17] Overall, the acute rise in serum creatinine of 0.5mg/dl or a 25% increase from baseline widely used in defining CIN, describes specific changes in renal function in keeping with the RIFLE classification.[9]

Various factors are responsible for the disparity in incidence rates obtained in studies. Mccullogh *et al*[28] reported an incidence of 3.9% and 14.5% when CIN was defined using serum creatinine rise ≥ 0.5mg/dl and ≥ 25% respectively. The high incidence obtained in our study may be due to the use of ≥25% rise in baseline serum creatinine to define CIN. Solomon *et al* recently suggested that an absolute rise of 0.3mg/dl (regardless of 25% rise in creatinine) may be a more sensitive threshold for CIN and related complications.[27]

Other factors that may generally influence the incidence rate of CIN include the baseline renal function of the study population (normal, reduced or mixed); contrast volume and presence or not of preventive interventions e.g. pre-hydration, use of sodium bicarbonate or N-acetylcysteine.[4] Though majority of the patients studied had normal or highnormal baseline serum creatinine, the drawbacks of using serum creatinine as a measure of renal function is well documented.

The frequency of CIN was higher among in-patients (38.4%) than out-patients (17.6%). Majority of subjects studied were ill emergency/inpatients who had other comorbid conditions commonest being cerebrovascular disease. In the study by Mitchell *et al*[18] majority of patients were stable out-patients referred for pelvic CT scan. Emergency patients are at increased risk for CIN[9] because of the lack of time to hydrate them adequately for the procedure. Again there was no specific pre-hydration protocol done in this study. More than 50% of patients had stroke, this meant that such patients had underlying vascular disease which is a risk factor for CIN. Again there is the regular practice of using mannitol for a subset of these patients, who had cerebral oedema. Mannitol may be harmful if used in patients undergoing contrast procedures as it causes osmotic diuresis and induces adenosine secretion[9] further increasing the risk for CIN.

The incidence of CIN in patients with underlying CKD is extremely high, ranging from 14.8%–55%[29, 30]. Hall *et al*[31] showed that the risk of CIN rises as baseline serum creatinine rises; risk of CIN was 2%, 10.4% and 62% respectively in patients with serum creatinine of ≤1.2mg/dl, 1.4–1.9mg/dland ≥ 2mg/dl. Similarly, in our study the risk of CIN significantly increased with increasing baseline serum creatinine; 29.6%, 39.6%, 66% and 100% respectively in patients

with baseline serum creatinine <0.9, 0.9–1.49, 1.5–2.0 and >2.0mg/dl. Only 5.6% (n=8) of the patients studied had serum creatinine ≥1.5mg/dl however 75.0% (n=6) of these developed CIN confirming the increased risk of CIN in patients with renal insufficiency.

In this study patients with some of the known risk factors for CIN had higher incidence of CIN compared to patients without such risk factors though did not reach statistical significance e.g.5 of the 10 (50%) diabetic patients developed CIN compared to non-diabetics (34.8%) and 46% of those with raised serum urea developed CIN compared to 30.4% among those with normal levels. Among diabetics, the incidence of CIN varies from 5.7–29.4%.[29,30] Lautin *et al* reported a higher incidence in azotaemic (30%) compared to non- azotaemic patients (10%).[32]

It was not surprising that increasing age and anaemia were significant risk factors of CIN. There is generally a decline in renal function with age, though this did not reflect in the overall baseline serum creatinine of the population. One of the limitations of the use of surrogate creatinine as a marker of renal function is that it overestimates renal function in the elderly because of their reduced muscle mass. Other peculiar characteristics of the elderly include atherosclerosis and reduced ability to accommodate oxidative injury[9] both of which increase the risk of CIN. In this study 14 out of 25 anaemic subjects developed CIN. Anaemia as a risk factor has been confirmed by other studies.[33]

In the study by Mitchell, *et al*[18] three presumed risk factors (heart failure, diabetes, and vascular disease) were more common in patients who developed CIN, whereas baseline renal insufficiency and the presence of anemia were not. In their study population, 41% had none of the presumptive risk factors for CIN, and 25% had only one risk factor.In our study however, only 19% of the 142 patients had none of the presumptive risk factors for CIN. This again further explains the higher incidence rate recorded in this study when compared to other studies.The fact that all patients were blacks may also be contributory, as some report have associated black race with increased predisposition to CIN.[35,36]

The correlation between the amount of CM and the risk of CIN is well documented.[6] The volume of contrast used during the procedures was generally minimal, 80mls for CT and 50mls for IVU. It would have been expected that there will be a relatively lower risk of CIN in these patients compared to patients in whom higher volumes of contrast are used though this was not studied. However CIN has been reported in patients in whom < 80mls of contrast media was used.[36] Manske *et al*[37] reported that volumes of low osmolar contrast media (iohexol or iopamidol) greater than 30mL were associated with markedly increased incidence of contrast nephropathy among azotaemic diabetics and for each 5-mL increment, the risk of nephropathy increased by 65%.

Previous literature that examined CIN focused on populations that were undergoing coronary or limb angiography and on patients with moderate to severe renal insufficiency.[38] Several differences in administration technique and patient strata underscore the need for more data on CIN following contrast enhanced CT. First, CT requires an intravenous bolus of 80 to 150ml of contrast material injected within 10 to 20 seconds, whereas organ or limb angiography studies use a series of smaller intra-arterial injections of contrast material, delivered over 10 to 20 min. This may result in important mechanistic differences in the development of CIN compared with intravenous administration. More critical is the increasing use of contrast CT scan in developing nations often times for emergency diagnosis of stroke in unstable patients with co-morbid conditions that may predispose them to CIN.

The frequency of CIN was lower among patients who had IVU(11.1%) compared to those who had CT scan(37.5%). This observation may be due to the lower volume of contrast injected during IVU but more important is the fact that patients who presented for IVU were mostly outpatients who were more clinically stable and had prior renal function test done as a routine practice.

Finally, this study confirms that CIN does occur among patients undergoing contrast-enhanced CT and that baseline renal insufficiency, anaemia and increasing age are the strongest risk factors. With the increasing use of this important diagnostic imaging, it is essential to emphasize that when considering patients for contrast procedure, patients and physicians need to be aware of the potential renal complications and weigh the risks against benefits. The usefulness of adequate hydration prior to and after contrast procedures is still of paramount importance. Additional research is needed to determine the potential for delayed complications in patients who have CIN and do not develop severe renal failure or death in the short term.

Our limitations were the high drop-out rate especially among outpatients due to unavailability for follow up blood draws; and refusal, death or discharge of inpatients. Most patients were not followed up for long enough to determine complete resolution of renal impairment (i.e. return of serum creatinine to baseline values) and to identify patients who may develop complications later. This was due to financial constraints and logistics.

DUALITY OF INTEREST.
declared None.

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