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From Contrast Exposure to Renal Recovery: Incidence, Predictors, and Early Outcomes in A Resource-Limited Setting

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Abstract **Original Research Article**

Background: Contrast-induced nephropathy (CIN) is a major cause of acute kidney injury (AKI) worldwide, with variable prevalence across centres. New biomarkers, such as serum cystatin C, have been introduced for earlier AKI detection, but data from resource-limited settings remain scarce. This study assessed the incidence, predictors, and short-term renal outcomes of CIN using cystatin C and creatinine in patients undergoing contrast-enhanced procedures at the University of Maiduguri Teaching Hospital (UMTH), Nigeria. ***Methods*:** In this prospective study, 150 consenting adults (≥18 years) receiving contrast media were enrolled. Sociodemographic data and baseline laboratory measurements, including cystatin C, creatinine, and estimated glomerular filtration rate (eGFR), were obtained. CIN was defined as a ≥0.5 mg/dL or ≥25% rise in serum creatinine within 48–72 hours post-contrast. Logistic regression identified predictors of CIN, and renal outcomes were assessed over three months. ***Results:*** CIN prevalence was 30% using creatinine at 48 hours and 49.3% using cystatin C at 24 hours. Independent predictors included older age (OR = 1.346, p = 0.009), higher contrast volume (OR = 2.037, p = 0.001), elevated baseline creatinine (OR = 1.601, p = 0.006), and lower baseline eGFR (OR = 1.767, p = 0.003). Cystatin C sensitivity and specificity ranged from 51.1–68% and 52.4–58.1%, respectively, across 24–72 hours, without superiority over creatinine. Of CIN cases, 73.3% recovered within two weeks; 17.9% had persistent dysfunction, and 4.6% required dialysis. At three months, 62.5% of persistent cases recovered, 25% had ongoing impairment, and 12.5% remained on dialysis. ***Conclusion:*** CIN is common in UMTH, with significant short-term renal sequelae. Key risk factors include age, contrast volume, and pre-existing renal impairment. Cystatin C did not outperform creatinine in CIN detection in this cohort.

Keywords: Contrast-induced nephropathy, acute kidney injury, cystatin C, creatinine.

INTRODUCTION

Contrast-induced nephropathy (CIN) is defined as an acute elevation of serum creatinine greater than 0.5 mg/dL above baseline or an increase exceeding 25% within 48–72 hours following administration of contrast media (CM) [1,2]. Serum creatinine remains the conventional diagnostic standard; however, its delayed rise after contrast exposure limits early detection of renal injury [2]. Cystatin C, an endogenous cysteine proteinase inhibitor, has been proposed as a more reliable biomarker because it is stable, freely filtered by the glomerulus, and reflects renal function earlier often within 24 hours of injury—while being less influenced by age, sex, race, or muscle mass [2,3].

In developed countries, CIN is the third leading cause of hospital-acquired acute kidney injury (AKI), after hypovolemia and surgical procedures [2,6]. Although its course may be benign in many patients, CIN is associated with increased morbidity, mortality, prolonged hospitalization, and, in some cases, the need for dialysis [4,5]. The global shift toward greater use of

advanced radiological imaging often requiring contrast agents has heightened CIN's clinical relevance, particularly in resource-limited settings [6].

In Nigeria, the growing demand for diagnostic and interventional radiological procedures using low, iso, and high-osmolar contrast media is likely to increase CIN incidence, along with the burden of AKI requiring dialysis [6]. While data from developed settings report CIN in 12% of hospital-acquired AKI cases, local studies suggest even higher rates [6,7]. For instance, Okoye *et al.,* [6]. reported a 35.9% incidence in Benin City, with risk factors including pre-existing renal impairment, diabetes mellitus, and concomitant nephrotoxic drug use.

The pathogenesis of CIN is multifactorial, involving a combination of ischemic and toxic injury to renal tubular cells. However, not all patients exposed to contrast agents develop CIN, suggesting that certain protective or predisposing factors influence individual susceptibility. Identifying these factors in our population is critical to preventing CIN and reducing the associated AKI burden [8-10].

Despite the increasing recognition of CIN, limited research in our setting has evaluated its prevalence, risk factors, and outcomes using both traditional (creatinine) and newer (cystatin C) biomarkers. This prospective study was designed to determine the prevalence of CIN in patients receiving contrast at the University of Maiduguri Teaching Hospital (UMTH), assess short-term renal outcomes over two weeks and three months, and identify predictors of CIN in this population.

Aims and Objectives

Primary Objective

- To evaluate short-term renal outcomes, including recovery, non-recovery, and dialysis dependency, following contrast-induced nephropathy (CIN), and to determine the usefulness of serum cystatin C compared with serum creatinine in monitoring these outcomes among patients undergoing contrast-enhanced imaging at UMTH, Maiduguri.

Secondary Objectives

1. To determine the prevalence of CIN in the study population.
2. To identify patient-related and procedural risk factors associated with CIN.
3. To assess the relationship between identified risk factors/co-morbidities and short-term renal outcomes.

METHODOLOGY

Study Design and Setting

This was a prospective observational study conducted at the University of Maiduguri Teaching Hospital (UMTH), Maiduguri, Nigeria, a major tertiary referral center serving the North-Eastern region.

STUDY POPULATION

The study enrolled adult patients (≥18 years) scheduled for contrast-enhanced imaging procedures between [insert month/year] and [insert month/year].

Inclusion Criteria

- Adults undergoing intravascular administration of iodinated contrast medium for radiological investigations.
- Baseline serum creatinine and/or cystatin C available within 24 hours before contrast exposure.

Exclusion Criteria

- Pre-existing end-stage kidney disease on maintenance dialysis.
- Refusal to give informed consent.
- Incomplete follow-up data within the defined short-term period.

Definition of Contrast-Induced Nephropathy

CIN was defined as an absolute increase in serum creatinine of ≥0.5 mg/dL (44 µmol/L) or a relative increase of ≥25% from baseline within 48–72 hours after contrast administration, in the absence of other identifiable causes.

Data Collection

Demographic data, comorbid conditions (e.g., diabetes, hypertension, heart failure), indication for imaging, type and volume of contrast used, baseline renal function, and concomitant nephrotoxic medications were recorded using a structured proforma.

Laboratory Assessment

- Serum creatinine and serum cystatin C were measured at baseline and at 48–72 hours post-contrast exposure.
- Renal outcomes were classified as complete recovery, partial recovery, persistent renal dysfunction, or dialysis dependency at 14 days post-exposure.

Outcome Measures

Primary outcomes:

- Incidence of CIN.
- Distribution of short-term renal outcomes (recovery vs non-recovery vs dialysis dependency).

Secondary outcomes:

- Predictors of CIN.
- Association between baseline cystatin C levels and renal outcomes.

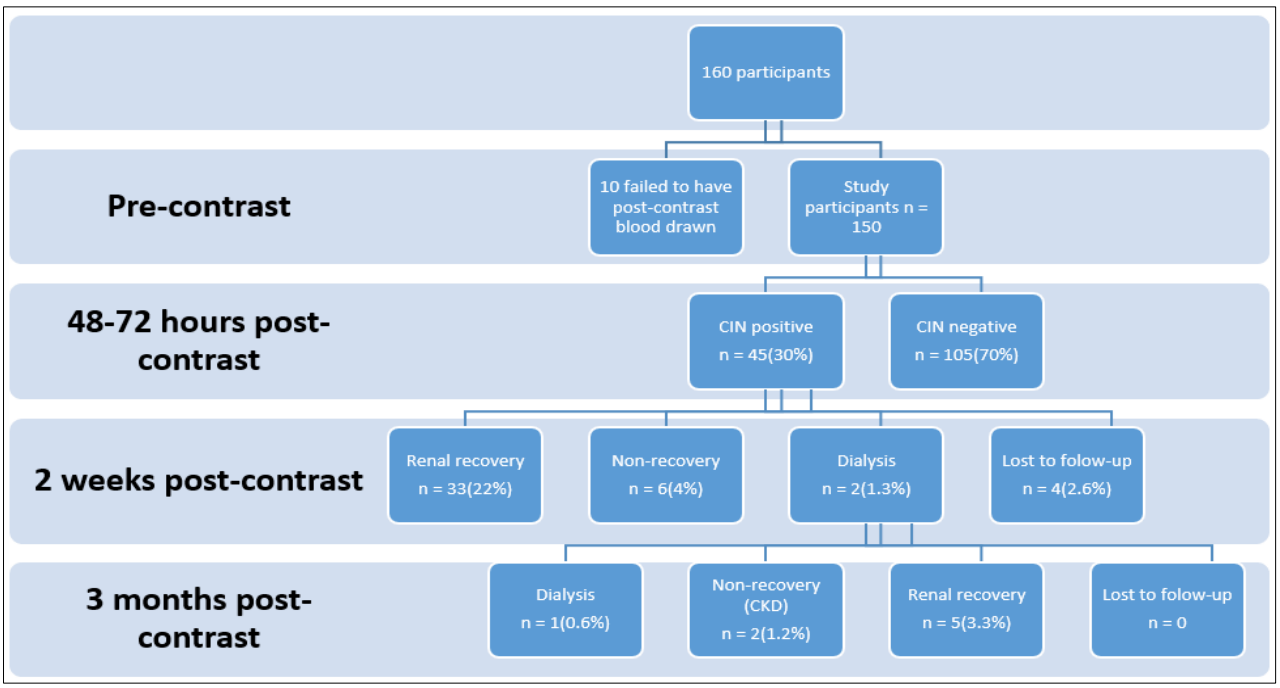
Statistical Analysis

Data were analysed using IBM-SPSS (International Business Machines-Statistical Package for

the Social Sciences) Statistics for Windows, Version 21.0. Continuous variables were expressed as mean ± standard deviation or median (IQR) and compared using Student's t-test or Mann–Whitney U test as appropriate. Categorical variables were expressed as proportions and compared using the Chi-square test. Logistic regression was used to identify independent predictors of CIN. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 160 subjects who satisfied the inclusion criteria were enrolled into the study. These subjects presented to the Radiology Department for various radiological investigations requiring the use of CM.



Sociodemographic Characteristics

The mean age of participants was 49.2 ± 15.4 years (range: 23–75), with the 50–59-year age group representing the largest proportion (33.3%), followed by 60–69 years (20.7%). Males predominated (61.3%), giving a male-to-female ratio of 1.58:1. Most had tertiary education (44.6%), while 26.7% had no formal education. Kanuri ethnicity was the most common (32.0%), followed by Babur (15.3%), Marghi (18.0%), Hausa/Fulani (11.3%), and Shuwa (6%); Yoruba and Igbo each constituted 6%, with other minority tribes making up 20%. Most participants were married (74.7%), while 12% were widowed, 10.7% single, and 2% separated. (Table 1)

Table 1: Socio-demographic Characteristics of Study Participants

Variable	Number of subjects (%)	Mean age ± SD (years)
Sex		
Male	92(61.3)	55.5 ± 10.7
Female	58 (38.7)	45.5 ± 13.3
Age Group (years)		
18-29	9(6.0)	
30-39	22 (14.7)	
40-49	27 (18.0)	
50-59	50(33.3)	
60-69	31(20.7)	
70-79	11(7.3)	
Marital Status		
Single	16 (10.7)	
Married	112 (74.7)	
Separated/Divorced	3 (2.0)	
Widowed	19 (12.7)	
Ethnicity		
Kanuri	48 (32.0)	

Babur Marghi Shuwa	23(15.3) 18 (12.0) 9(6.0)	
Hausa/Fulani	17(11.3)	
Igbo Yoruba	6 (4.0) 6 (4.0)	
Others	20 (13.3)	
Educational Status		
None Secondary Tertiary	40 (26.7) 22(14.7) 67(44.6)	
Islamic	21 (14.0)	

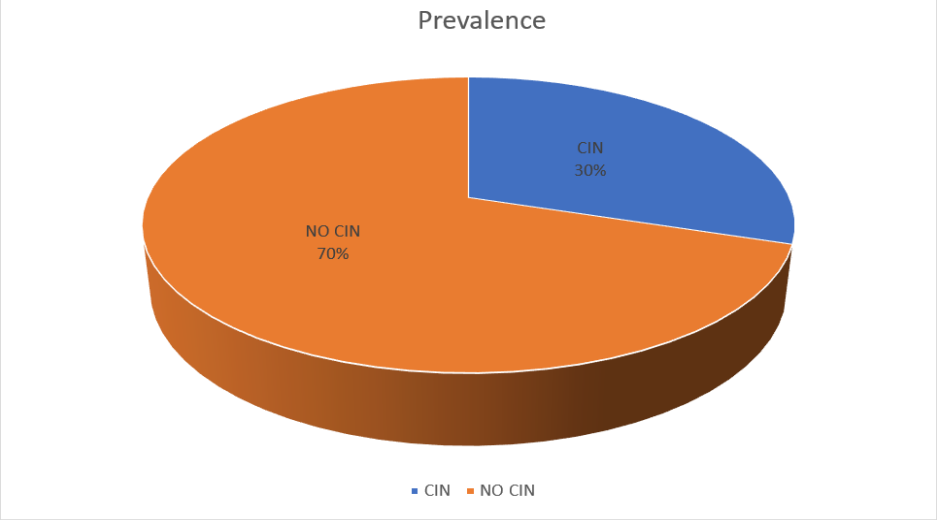
INCIDENCE

The incidnece of CIN was found to be 30% (45 subjects) based on rise in serum creatinine by 44.2µmol/l and/or ≥ 25% at the baseline value at 48 and 72 hours post contrast (Table 2)

Table 2: Incidenceof CIN

Criteria (rise in serum creatinine from baseline)	Normal n(%)	Number of study subjects with CIN n(%)
≥ 44.2 (µmol/48hrs)	128(85.3)	22(14.7)
≥44.2 (µmol/72hrs)	122(81.3)	28(18.7)
≥ 25% (µmol/48hrs)	118(78.7)	32(21.3)
≥ 25% (µmol/72hrs)	111(74.0)	39(26.0)
44.2µmol/l and ≥ 25%	105(70.0)	45(30.0)

Abbreviations: CIN (contrast-induced nephropathy)



Pie Chart showing incidence of CIN based on serum creatinine rise at 48 and 72 hours

Predictors

The predictors for the development of CIN after exposure to Contrast media included the following: advancing age (p=0.019), high volume of contrast (p=0.001), high creatinine at baseline (p=0.006), and low eGFR at baseline (p=0.003) (Table 3)

Table 3: Predictors of CIN

Predictors	P Value	OR	CI (95%)
Advancing Age	0.009	1.346	1.006 – 2.990
High Volume of contrast	0.001	2.037	1.015 – 9.060
High Creatinine at baseline	0.006	1.601	1.043 – 6.010
Low eGFR at baseline	0.003	1.767	1.510– 7.980
Cystatin C at baseline	0.002	1.052	1.012- 1.053

Abbreviations: OR (odds ratio), eGFR (estimated glomerular filtration rate), CI (confidence interval)

Short term outcomes

Forty-five subjects developed CIN after exposure to CM. Of these, 33 had serum creatinine value return to baseline, 6 subjects had non recovery of renal function, two subjects were on haemodialysis. Four subjects were lost to follow up.

At three months post exposure to ICAs, one subject was still having twice weekly haemodialysis, five out of the six subjects (who had non-renal recovery at 2 weeks post-contrast) had their serum creatinine return to baseline. Two of the subjects had persistent renal non-recovery but had not commenced renal replacement therapy. (Table 4,5)

Table 4: Outcome 2 weeks post-CIN

Category of outcome	Number of subjects (%)
Renal recovery	33(73.3)
Non-recovery	6(13.3)
Renal replacement Lost to follow-up	2(4.6) 4(8.8)
Death	0(0.0)
Total	45(100)

Abbreviation: CIN (contrast induced nephropathy)

Table 5: Outcome Three Months Post-CIN

Category of outcome	Number of subjects(%)
Renal recovery	38(84.4)
Non-recovery	2(4.4)
Renal replacement Lost to follow up	1(2.2) 4(8.8)
Death	0(0.0)
Total	45(100)

Abbreviation: CIN (contrast induced nephropathy)

DISCUSSION

This study demonstrates that the prevalence of contrast-induced nephropathy (CIN) in our setting is considerably higher than many earlier global reports. Depending on the definition applied, the prevalence ranged from 14.7% to 26.0% when assessed using either an absolute rise in serum creatinine of 44.2 µmol/L (0.5 mg/dL) or a ≥25% increase from baseline, with cystatin C yielding even higher frequencies. When both criteria were combined, the overall prevalence reached 30%, a finding comparable to that reported by Okoye *et al.,* [6]. in Nigeria. The higher prevalence observed in this study compared to those of Richal *et al.,* [47], Mueller [48] *et al.,* and Barrett *et al.,* [49], may partly be due to the absence of routine pre- and post-procedure prophylactic interventions such as vasodilators, hydration protocols, and nephroprotective agents, which are more commonly implemented in high-resource settings. Additionally, it is possible that Black African populations may have a higher inherent susceptibility to CIN, as suggested by similarities between our findings and those from other African studies.

Our results further underscore the variability in CIN incidence based on the definition employed. As has been observed in other studies, stricter definitions such as an absolute creatinine increase ≥0.5 mg/dL tend to yield lower estimates, whereas relative increases (>25%) and combined definitions produce higher prevalence rates. These thresholds align with established criteria for acute kidney injury such as the RIFLE classification.

In multivariate analysis, several variables emerged as independent predictors of CIN. The most significant was the volume of contrast administered, followed by lower baseline estimated glomerular filtration rate (eGFR), elevated baseline serum creatinine, baseline cystatin C, and increasing age. These findings mirror those of Okoye *et al.,* [6], Evola *et al.,* [44], Banda *et al.,* [54], and Kashif *et al.,* [51], who identified eGFR <60 mL/min/1.73 m², older age, and elevated baseline creatinine as consistent risk factors. Our study reinforces the well-documented inverse relationship between eGFR and CIN risk, as shown by Sany *et al.,* in diabetic cohorts and Kashif *et al.,* in patients undergoing cardiac catheterization.

At three months post-contrast, most patients with CIN had regained baseline renal function; however, a small proportion had persistent renal impairment and one remained dialysis-dependent. This recovery rate is lower than that reported by Shigidi *et al.,* [55]. in Sudan, where all affected patients recovered within days, likely due to universal prophylaxis with sodium bicarbonate and N-acetylcysteine and the discontinuation of nephrotoxic medications. In contrast, our study population predominantly outpatients in a resource-limited setting did not routinely receive such preventive measures.

The short-term renal outcomes in our cohort were not predicted by any of the baseline variables

assessed. This differs from findings by Wi *et al.,* [56], Kim *et al.,* [45], and Banda *et al.,* [54], who reported higher mortality, greater dialysis requirements, or poorer recovery among patients with advanced CKD, anaemia, or hypertension. The lack of predictive power in our cohort may be attributable to the relatively small sample size for outcome analysis and the predominance of less severely ill, ambulatory patients.

Interestingly, despite the high prevalence of CIN, no deaths were recorded during the follow-up period. This is in contrast to studies by Chong *et al.,* [53] and Banda *et al.,* [54], which reported increased mortality among patients developing CIN. The absence of mortality in our study may be explained by differences in patient selection, as most of our participants were stable outpatients, whereas many of the comparator studies involved hospitalized or critically ill patients.

Taken together, these findings suggest that CIN is a significant and under-recognized problem in our resource-limited setting. The high prevalence, coupled with the identification of modifiable predictors such as contrast volume, highlights the urgent need for preventive strategies tailored to our context. Interventions

CONCLUSION

This study reveals that contrast-induced nephropathy (CIN) is a common and under-recognized complication in our resource-limited setting, with prevalence rates markedly higher than many earlier global reports. The incidence varied with the definition employed, underscoring the need for standardized diagnostic criteria. Independent predictors identified—particularly contrast volume, impaired baseline renal function, and increasing age—align with established risk factors reported globally and emphasize the importance of careful patient selection and tailored preventive strategies.

Although most affected patients achieved renal recovery within three months, a subset developed persistent impairment, and one remained dialysis-dependent, highlighting the potential long-term consequences. Unlike studies from high-resource settings, the absence of routine prophylactic interventions in our cohort may have contributed to both the higher prevalence and delayed recovery.

The findings underscore the need to adopt context-appropriate preventive measures, including hydration protocols, minimization of contrast exposure, and closer monitoring of high-risk patients. Larger, prospective studies are warranted to further define the burden of CIN in sub-Saharan Africa and to develop cost-effective strategies to mitigate its impact.

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