Scientific Research Publishing

Open Journal of Nephrology, 2025, 15(3), 384-397
https://www.scirp.org/journal/ojneph
ISSN Online: 2164-2869
ISSN Print: 2164-2842

Evaluation of Contrast-Induced Nephropathy following Contrast-Enhanced Computed Tomography

Mahfuzur Rahman[1*], A. H. Hamid Ahmed[2], Syed Fazlul Islam[2], Md. Kabir Hossain[2], Ferdous Jahan[2], Md. Masudul Karim[2], Mahfuja Jahan[3], Sayed Rakib Hasan[4], Md. Abdullahil Baki[5], A. K. M Shahidur Rahman[2]

[1]Department of Nephrology, Sher-E-Bangla Medical College and Hospital, Barisal, Bangladesh
[2]Department of Nephrology, Bangladesh Medical University (BMU), Dhaka, Bangladesh
[3]Department of Radiology and Imaging, Bangladesh Medical University (BMU), Dhaka, Bangladesh
[4]Department of Medicine, Kushtia Medical College Hospital, Kushtia, Bangladesh
[5]Department of Nephrology, Shaheed Suhrawardy Medical College and Hospital, Dhaka, Bangladesh
Email: *drmahfuj34@gmail.com

How to cite this paper: Rahman, M., Ahmed, A.H.H., Islam, S.F., Hossain, M.K., Jahan, F., Karim, M.M., Jahan, M., Hasan, S.R., Baki, M.A. and Rahman A.K.M.S. (2025) Evaluation of Contrast-Induced Nephropathy following Contrast-Enhanced Computed Tomography. *Open Journal of Nephrology*, **15**, 384-397.
https://doi.org/10.4236/ojneph.2025.153036

Received: July 22, 2025
Accepted: August 26, 2025
Published: August 29, 2025

Abstract

Background: During computed tomography (CT), contrast is commonly used to identify structural and functional defects of various organs. Contrast-induced nephropathy (CIN) is a known complication of intravenous iodinated contrast, which appears as a common cause of hospital-acquired renal failure. **Objective:** To evaluate the contrast-induced nephropathy following contrast-enhanced computed tomography. **Method:** This prospective observational study was carried out at the Department of Nephrology, Bangladesh Medical University (BMU), Dhaka, Bangladesh, from November 2022 to September 2023, involving one hundred (100) patients who underwent contrast-enhanced computed tomography (CECT). A detailed case history was recorded, and a thorough clinical examination was carried out along with relevant investigations at baseline, after 48 - 72 hours, and on the 7th day following CECT was done. **Results:** The mean age of the study participants was (49.9 ± 14.4) years, ranging from 18 to 70 years, with a slightly higher number of male participants (n = 60). The most performed contrast studies were CECT abdomen (58%), followed by chest (32%) and others (10%). Out of 100 study participants, 10 (10%) developed CIN. Our study found that advanced age and type of contrast had a significant association with the development of CIN ($p < 0.05$). Baseline serum creatinine was found to be a significant predictor of CIN ($p = 0.013$; OR = 73.86). **Conclusion:** Contrast-induced nephropathy (CIN) is not uncommon. Advanced age and the type of contrast have a significant association with

CIN. Baseline renal status has a strong association with increased risk of CIN. Proper prophylactic measures could avoid or minimize progressive renal injury.

Keywords

Acute Kidney Injury (AKI), Contrast-Enhanced Computed Tomography (CECT), Contrast-Induced Nephropathy (CIN), Contrast Media (CM), Type of Contrast

1. Introduction

Acute kidney injury (AKI) secondary to iodinated contrast media (CM) is commonly referred to as contrast-induced nephropathy (CIN). CIN is a phenomenon of increased interest in recent years for a variety of reasons [1]. The term CIN has also become controversial; recently, the Acute Kidney Injury Network (AKIN) has chosen "Acute Kidney Injury (AKI)" as a synonym for CIN in cases where AKI occurs as a result of contrast administration [2]. Contrast-induced nephropathy (CIN) is defined as an increase in the serum creatinine level of ≥0.5 mg/dl or ≥25% from baseline within 48 - 72 hours following the administration of contrast medium [3]. An absolute increase in baseline serum creatinine is considered the most useful and practical definition of CIN. The serum creatinine typically starts to increase within 24 - 48 hours of contrast medium administration, with peak elevation occurring after 3 - 5 days and returning to baseline or near baseline within 7-10 days [4]. Acute kidney injury (AKI) caused by CIN is generally non-oliguric and reversible. The exact mechanism underlying contrast-induced nephropathy (CIN) is still elusive. The leading theories propose that it stems from two primary sources: first, hypoxic injury to the renal tubules due to renal vasoconstriction; and second, direct cytotoxic effects induced by the contrast media itself [5]. Alternatively, some experts suggest that acute kidney injury following intravascular contrast administration might be linked more to pre-existing risk factors and might merely coincide with the use of contrast media, particularly when administered intravenously [6]. A recent meta-analysis found no evidence for increased risk for AKI after contrast-enhanced CT among patients with estimated glomerular filtration rate (eGFR) ≥ 45 mL/min/1.73m^2 [7]. Based on emerging evidence in the literature, it was suggested that caution should be exercised in patients with hypertension and eGFR ≤ 30 mL/min/1.73m^2 [7]. The overall incidence of CIN in the general population remains uncertain and ranges from 0.6% to 2.3% in various studies [8]. Contrast-induced nephropathy (CIN) is a known complication following the intravascular administration of iodinated contrast media (CM) in the absence of other nephrotoxic events and is often associated with both short-term and long-term morbidity and mortality [8] [9]. The reassessment of the risk of CIN among individuals referred for contrast-enhanced computed tomography

(CECT) is driven by the growing usefulness of CECT in diagnostic imaging. It is a well-established practice to avoid the use of high-osmolar contrast media (HOCM) because it is linked to nephrotoxicity, cardiovascular side effects, and anaphylactoid reactions. However, the choice between using iso-osmolar contrast media (IOCM) or low-osmolar contrast media (LOCM) remains a matter of uncertainty. There are conflicting findings in the literature regarding whether IOCM carries a lower risk of contrast-induced nephropathy (CIN) compared to LOCM [9] [10]. During recent years, contrast-related investigations have been common diagnostic tools. Despite several reported complications, contrast largely increases the diagnostic yield of radiological imaging, especially in the case of malignancy. CIN is not an uncommon cause of acute renal failure. In the majority of cases, CIN remains undiscovered, particularly when the investigation is conducted in the outpatient settings. The majority of CIN episodes are transient and typically resolve within one to three weeks, but in a small number of cases, permanent renal impairment may develop, as seen by an increase in serum creatinine level [11]. It is worth noting that CIN is associated with significant morbidity and mortality after computed tomography (CT) in both inpatient and outpatient settings. There is very little data regarding CIN after contrast-enhanced computed tomography (CECT), particularly in resource-limited countries. Therefore, the present study aimed to evaluate the contrast-induced nephropathy following contrast-enhanced computed tomography at a Tertiary Care Hospital in Bangladesh.

2. Methodology

This prospective observational study was carried out at the Department of Nephrology, Bangladesh Medical University (BMU), Dhaka, Bangladesh, from November 2022 to September 2023. A total of one hundred (100) patients who underwent contrast-enhanced computed tomography (CECT) at the Department of Radiology and Imaging, BMU, Dhaka, Bangladesh, were enrolled by a purposive sampling technique following selection criteria. Adult subjects (age >18 years) who underwent computed tomography (CT) were included. Subjects having contrast allergy or having had previous intravenous contrast scanning for any reason within 14 days before enrollment, patients with impaired renal function (serum creatinine ≥ 2 mg/dl) due to any cause, were excluded from the study. The selection criteria were scrutinized by medical history along with drug history, clinical examination, previous medical records, and recent laboratory investigations.

2.1. Sample Size Estimation

Sample size was calculated from the following formula:

$$n = \frac{\left\{u\sqrt{\left[\pi\left(1-\pi\right)\right]} + v\sqrt{\left[\pi_0\left(1-\pi_0\right)\right]}\right\}^2}{\left(\pi-\pi_0\right)^2}$$

where,

n = Sample size

u = 1.96 at a 95% confidence interval

v = 0.84 at an 80% power

π = 11% (11% patients had developed CIN [12])

π_0 = 4%

Putting the values in the above equation, the sample size is estimated as

$$n = 86(\text{estimated sample size})$$

Considering a 14% drop-out, the adjusted sample size was

$$n = \text{desired sample size} / (\%\ \text{retained participants after drop out})$$
$$= 86/0.86$$
$$= 100$$

2.2. Study Procedure

Prior to commencement of the study, ethical approval was obtained from the Institutional Review Board (IRB) of Bangladesh Medical University (BMU), Dhaka, Bangladesh. After getting IRB approval, a total of 100 patients who fulfilled the selection criteria were evaluated. All the study participants were informed about the aims, objectives, and detailed procedure of the study before enrollment. If they willingly agreed to participate in this study, blood samples were taken before the contrast-enhanced computed tomography (CECT), after 48 - 72 hours, and on the 7th day of CECT. Baseline investigations like complete blood count (CBC), random blood sugar (RBS), serum urea, serum creatinine, and serum electrolytes were done following standard procedure. The medical records of each study participant were collected to extract demographic information such as age, sex, medical history, blood pressure (BP), height, weight, body mass index (BMI), etc. An estimated glomerular filtration rate (eGFR) was calculated from serum creatinine values by using the modification of diet in renal disease (MDRD) formula [13].

Computed tomography was carried out by Siemens Somato Definition as CT Scanner from Siemens Healthiness USA. Iobitridol [Xenetix-350 (350 mg I/ml)] and iodixanol (Visipaque-320 mg I/ml) were used as contrast media (CM). In this study, 91 study participants received iobitridol, while only 9 participants received iodixanol. Regarding the volume of contrast media (CM), 61 of the study participants received a volume of CM 70 ml, and the remaining 39 participants received a volume of CM 50 ml.

We have used parenteral hydration with normal saline at a dose of 3 mL/kg/hour for one hour before the procedure and 1 mL/kg/hour for up to six hours following CECT, as part of our CIN prophylactic protocol in 17 participants having eGFR < 45 mL/minute/1.73m^2. This prophylactic protocol was not followed, and the remaining cases had eGFR > 45 ml/minute/1.73m^2. A diagnosis of CIN was made based on an absolute increase in serum creatinine levels by 0.5 mg/dl or by a relative increase in serum creatinine levels greater than 25% compared to baseline within 48 - 72 hours after contrast media (CM) administration [3].

2.3. Statistical Analysis

All data were recorded systematically in a preformed data collection sheet. Quantitative data were expressed as mean with standard deviation (SD), and qualitative data were expressed as frequency (n) with percentage (%). Statistical analyses were performed using Windows-based computer software, Statistical Packages for Social Sciences (SPSS) version 26. Associations between categorical variables were made using the Chi-square test, and continuous variables were analyzed using the Unpaired t-test. A multivariate logistic regression analysis was done to identify CIN predictors. A p-value of <0.05 was considered statistically significant.

3. Results and Observations

This prospective observational study was intended to assess the contrast-induced nephropathy (CIN) after contrast-enhanced computed tomography (CECT). The incidence of CIN was found to be 10% in this study. The study subjects were divided into 2 groups: "CIN group" and "No-CIN group".

The mean age of the participants was 49.9 ± 14.4 years. Of them, 60% were male, while 40% were female. The age distribution showed that the majority of them fall in the 60 - 70 years (36%) and 50 - 59 years (27%) age groups. The mean BMI was (20.7 ± 1.71) kg/m^2 (Table 1).

Table 1. Demographic characteristics of the study population (N = 100).

Variables	Number (%)	Mean ± SD
Mean age (years)		49.9 ± 14.4
Age groups (years)		
18 - 29	11 (11.0)	
30 - 39	16 (16.0)	
40 - 49	10 (10.0)	
50 - 59	27 (27.0)	
60 - 70	36 (36.0)	
Gender		
Male	60 (60.0)	
Female	40 (40.0)	
BMI (kg/m^2)		20.7 ± 1.74

In this study, 29% of the participants had hypertension (HTN), while 14% had diabetes mellitus (DM), 17% had cirrhosis of the liver, 10% had chronic kidney disease (CKD), and 3% had ischemic heart disease (IHD). Of them, 10% were on different types of nephrotoxic drugs such as NSAIDs. Regarding behavioral risk factors, 16% of the study participants were smokers (Table 2).

Table 2. Distribution of the study patients by comorbidities (N = 100).

Comorbidities*	Number (%)
Hypertension (HTN)	29 (29.0)
Diabetes mellitus (DM)	14 (14.0)
Cirrhosis of the liver	17 (17.0)
Chronic kidney disease (CKD)	10 (10.0)
Ischemic heart disease (IHD)	3 (3.0)
Smoking	16 (16.0)
Nephrotoxic medication	15 (15.0)

*Multiple response.

Among the study population, the majority (59%) had a CT scan of the whole abdomen. 32% of the participants had a chest CT scan, while 7% had a kidney ureter with bladder (KUB) CT scan, and 2% had a hepatobiliary system (HBS) CT scan (Figure 1).

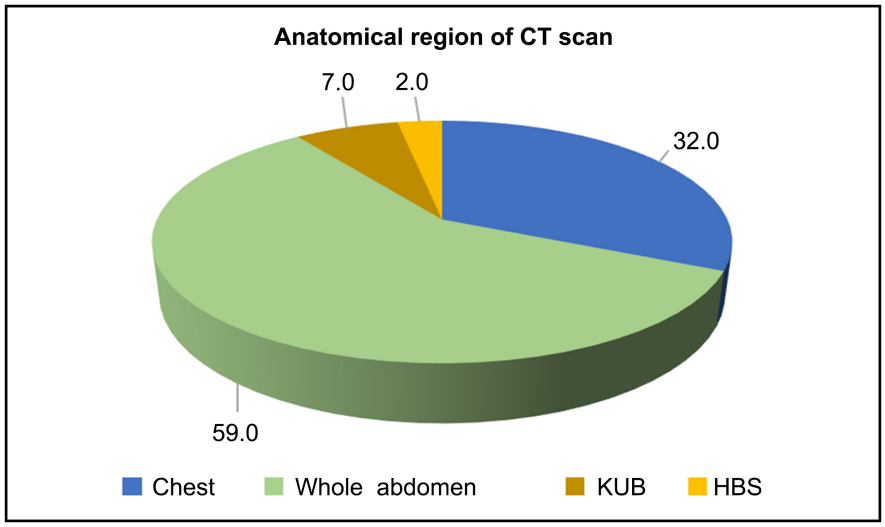

Figure 1. Distribution of the study population by anatomical region of the CT scan.

Out of 100 study participants, 10 (10%) participants had developed CIN, while 90 (90%) did not develop CIN. This data helps determine the prevalence of CIN in the study population and can be used to evaluate how contrast media affects kidney function (Figure 2).

Comparison of laboratory parameters between with (CIN) or without contrast-induced nephropathy (No-CIN) among the study subjects indicated that participants with CIN had significantly higher levels of serum urea, serum creatinine, and serum potassium compared to patients without CIN at baseline, after 48 - 72 hours, and on the 7th day of CECT (Table 3).

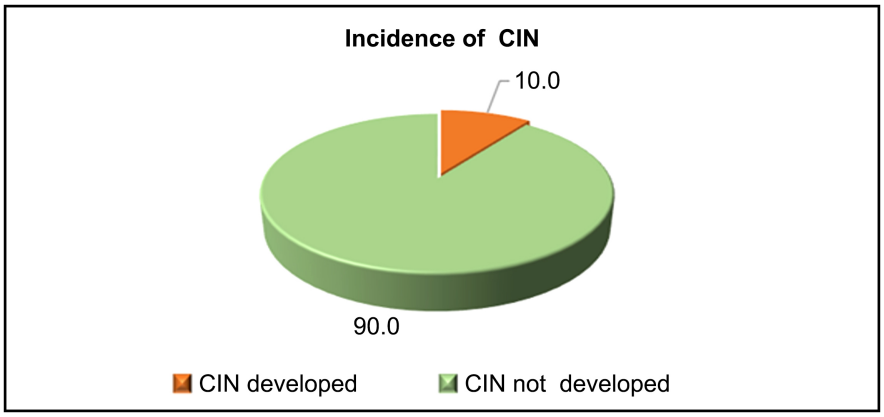

Figure 2. The incidence of CIN among the study participants (N = 100).

Table 3. Comparison of laboratory parameters with or without CIN (N= 100).

Laboratory parameters	No-CIN (N = 90) Mean ± SD	CIN (N = 10) Mean ± SD	p-value
Baseline			
S. urea (mg/dl)	18.86 ± 4.20	23.30 ± 3.86	0.002[s]
S. creatinine (mg/dl)	0.80 ± 0.21	1.40 ± 0.27	<0.001[s]
S. Na^+ (mmol/L)	133.98 ± 13.27	134.70 ± 5.08	0.865
S. K^+ (mmol/L)	3.95 ± 0.22	4.27 ± 0.53	<0.001[s]
After 48 - 72 hours			
S. urea (mg/dl)	18.73 ± 2.22	29.90 ± 4.43	<0.001[s]
S. creatinine (mg/dl)	0.83 ± 0.20	2.16 ± 0.47	<0.001[s]
S. Na^+ (mmol/L)	134.40 ± 12.79	135.10 ± 2.60	0.864
S. K^+ (mmol/L)	3.93 ± 0.22	4.71 ± 0.43	<0.001[s]
On 7^{th} day			
S. urea (mg/dl)	18.80 ± 2.25	27.80 ± 5.45	<0.001[s]
S. creatinine (mg/dl)	0.83 ± 0.21	1.99 ± 0.70	<0.001[s]
S. Na^+ (mmol/L)	135.72 ± 1.74	135.30 ± 2.91	0.501
S. K^+ (mmol/L)	3.93 ± 0.21	4.66 ± 0.40	<0.001[s]

p-value obtained by unpaired t-test, s = significant.

Table 4 presents the results of multivariate logistic regression to assess independent predictors of CIN. Baseline serum creatinine was found to be a statistically significant predictor (p = 0.013; OR = 73.86), indicating a strong association with increased risk of CIN. Although nephrotoxic drug use showed a near-signif-

icant trend (p = 0.059; OR = 0.064), it did not reach statistical significance. Other variables, including hypertension, diabetes mellitus, liver cirrhosis, and chronic kidney disease (CKD), were not significantly associated with CIN in this model (p > 0.05).

Table 4. Multivariate logistic regression analysis identifying predictors of contrast-induced nephropathy (CIN) among study participants (N = 100).

Variables	p-value	OR	95% CI	
			Lower	Upper
Baseline creatinine	0.013	73.86	0.252	21,683
HTN	0.277	0.265	0.024	2.906
DM	0.623	2.024	0.122	33.627
Liver cirrhosis	0.236	0.168	0.009	3.206
CKD	0.441	0.203	0.004	11.765
Nephrotoxic drugs	0.059	0.064	0.004	1.110

Regarding the association of anatomical region, contrast types, and volume of contrast with contrast-induced nephropathy (CIN) among the study subjects, the type of contrast had a statistically significant association with the development of CIN (p < 0.001) (Table 5).

Table 5. Association of contrast variables with CIN (N = 100).

Variables	No-CIN (N = 90) N (%)	CIN (N = 10) N (%)	p-value
Anatomical region			
Chest	29 (32.2)	3 (30.0)	0.743
Whole abdomen	52 (57.8)	7 (70.0)	
KUB	7 (7.8)	0 (0.0)	
HBS	2 (2.2)	0 (0.0)	
Type of contrast			
Iodixanol	3 (3.3)	6 (60.0)	<0.001[s]
Iobitridol	87 (96.7)	4 (40.0)	
Volume of contrast			
50 ml	36 (40.0)	3 (30.0)	0.539
70 ml	54 (60.0)	7 (70.0)	

p-value obtained by Chi-square test for qualitative data, s = significant.

On the 7th day of CIN follow-up, patients whose renal function returned to baseline had a mean serum creatinine of (1.00 ± 0.15) mg/dl, while those improving showed serum creatinine levels of (1.30 ± 0.11) mg/dl. Patients with static renal function had serum creatinine levels of (1.40 ± 0.15) mg/dl, and those deteriorating reached mean serum creatinine levels of (2.10 ± 0.2) mg/dl (Table 6).

Table 6. Renal function status and serum creatinine level on the 7th day following CECT in CIN patients (N = 10).

Renal function status	Frequency (%)	Serum creatinine (Mean ± SD)
Returned to baseline	3 (30.0%)	(1.00 ± 0.15) mg/dl
Improving	3 (30.0%)	(1.30 ± 0.11) mg/dl
Static	2 (20.0%)	(1.40 ± 0.15) mg/dl
Deteriorating	2 (20.0%)	(2.10 ± 0.2) mg/dl

4. Discussion

In hospitalized patients, contrast-induced nephropathy (CIN) is the third most common preventable cause of acute kidney injury (AKI) [14]. The evaluation of contrast-induced nephropathy (CIN) following computed tomography (CT) is essential for understanding the risk factors associated with this condition and for implementing preventive measures. The purpose of the present study was to assess the CIN following contrast-enhanced computed tomography (CECN). A total of 100 patients who underwent contrast-enhanced computed tomography (CECT) were selected.

In this study, hypertension (HTN), diabetes mellitus (DM), cirrhosis of the liver, chronic kidney disease (CKD), and ischemic heart disease (IHD) were the comorbidities, while nephrotoxic drugs and smoking were risk factors among the study participants. In multivariate regression analysis, baseline serum creatinine was found to be a significant predictor for CIN ($p = 0.013$; OR = 73.86). Although other variables, including hypertension, diabetes mellitus, liver cirrhosis, chronic kidney disease (CKD), and nephrotoxic drugs, were not significantly associated with CIN ($p > 0.05$). It was documented that the existence of cardiovascular disease (*i.e.*, myocardial infarction, angina pectoris, and chronic heart failure) and pre-existing renal dysfunction were associated factors for CIN [15] [16]. Khodabandeh *et al.*, in a systematic review, observed that acute kidney injury (AKI) is not caused by intravenous contrast media at a standard dose for CT scans unless a specific risk factor is present [17].

In this study, the most commonly performed imaging studies were CT abdomen (58%), followed by CT chest (32%) and others (10%). These findings were consistent with one previous study where a similar anatomical distribution of contrast-enhanced CT was performed [12]. The incidence of contrast-induced nephropathy (CIN) was 10% in this current study. This information provides an understanding

of the occurrence of CIN within the study population and can be used to assess the impact of contrast administration on kidney function. In accordance with this current study, Ong *et al.* reported that the incidence of CIN ranged from 5.69% to 36.9% [18]. The reported incidence of CIN percentage varies across the studies, with some studies reporting a high incidence of CIN [19] [20] and others reporting a low incidence [21] [22]. These findings suggest that the incidence of CIN may vary depending on the study population, the type and amount of contrast agent used, and other factors.

It was observed that participants who developed CIN had significantly higher levels of serum urea, creatinine, and potassium at baseline than those who did not (No-CIN). Although the underlying mechanism is not yet fully understood, iodinated contrast media frequently increase the strain on already impaired kidneys, lower renal blood flow and glomerular filtration rate (GFR), and permit the accumulation of contrast molecules within the kidney, exacerbating kidney injury [23]. Additional hypothesized mechanisms for CIN include activation of endothelin and other vasoconstrictor mediators, increased production of reactive oxygen species that cause lipid peroxidation and tubular damage, impairment of tubular endothelium mitochondrial function, and stimulation of cytokines and local inflammatory responses [24].

The present study revealed that the age distribution of the study population is important, with a significant portion falling into the 50 - 59 and 60 - 70 age groups, which were 27% and 36% respectively. The mean age of the participants was approximately 50 years, with a higher proportion of males (60%). This information is important because age was found to be associated with the development of CIN. Participants in the 60 - 70 age group had a higher incidence of CIN (70%), suggesting that older individuals may be more vulnerable to contrast-induced kidney injury. Clinicians should exercise caution when administering contrast media to older participants and closely monitor their renal function. This finding was in agreement with a related study that reported the mean age of the CIN group was 54 (±14) years compared to the non-CIN group 46 (±15) years [12]. Our study population shares similarities with a study conducted by Bhatt *et al.* (2016) in which they also found a higher percentage of males (58.8% males compared to 41.2% females) [2]. Similar patterns of a higher male presence have been observed in other studies, although there are some studies that have reported a higher percentage of females also [12]. In a couple of previous studies, demographic factors were not identified as influential factors affecting the occurrence of CIN [2] [25].

Among the study population, the type of contrast had a significant association with the development of CIN ($p < 0.001$). Specifically, participants who received iodixanol as the type of contrast had a higher incidence of CIN (60%) compared to patients who received iobitridol (40%). The anatomical region of the computed tomography scan and volume of contrast did not show any statistically significant association with the development of CIN ($p > 0.05$). These findings suggest that the type of contrast could be a potential risk factor for the development of CIN

following CECT, and precautions should be taken while selecting a contrast media for participants. In our study, 91 participants received Iobitridol (LOCM) as contrast media, and the remaining 9 participants received iodixanol (IOCM). The result showed that in participants who received Iodixanol (IOCM), CIN developed in 6 out of 9 participants. On the other hand, participants with normal baseline renal function who received Iobitridol (LOCM) developed CIN in 4 out of 91 participants. Cheruvu *et al.* reported that participants who received Iodixanol (IOCM) had significantly experienced CIN ($p = 0.015$) [26]. Another recent study showed that the CIN rate for iohexol (LOCM) was 2% compared to 9% for iodixanol (IOCM) [27]. It was documented that participants who received iodixanol (IOCM) were almost 5 times more likely to experience CIN [27] [28].

In this study, at the 7th day out of 10 patients of CIN; 3 patients' renal function (Serum creatinine) returned to baseline, 3 patients' renal function (Serum creatinine) were improving, 2 patients' renal function (Serum creatinine) remained static, and 2 patients' renal function (Serum creatinine) were deteriorating. Typically, CIN is a self-resolving process, with renal function returning to normal within 7 - 14 days after administration of contrast. Less than one-third of patients with CIN experience residual renal impairment to some degree, while less than 1% of patients with CIN require renal replacement therapy, with a slightly higher rate in those with chronic renal impairment (3.1%) [29].

5. Conclusion

This study demonstrated that the incidence of contrast-induced nephropathy (CIN) in subjects who underwent CECT is 10%. Advanced age and the type of contrast have a significant association with the development of CIN. Baseline serum creatinine is a significant predictor of CIN. Therefore, participants' age, renal status, and the type of contrast should be taken into consideration when performing CECT.

Limitations of the Study

The current study had certain limitations. It was a single-center study that included a small number of participants. Moreover, follow-up periods were relatively short.

Recommendations

A long-term multicenter study with close follow-up is warranted to validate the findings of this study. Preventive measures should be taken before, during, and after the CECT procedure. Follow-up should be continued at least 4 weeks following CECT.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

[1] Meinel, F.G., De Cecco, C.N., Schoepf, U.J. and Katzberg, R. (2014) Contrast-Induced

Acute Kidney Injury: Definition, Epidemiology, and Outcome. *BioMed Research International*, **2014**, Article ID: 859328. https://doi.org/10.1155/2014/859328

[2] Bhatt, S., Rajpal, N., Rathi, V. and Avasthi, R. (2016) Contrast Induced Nephropathy with Intravenous Iodinated Contrast Media in Routine Diagnostic Imaging: An Initial Experience in a Tertiary Care Hospital. *Radiology Research and Practice*, **2016**, Article ID: 8792984. https://doi.org/10.1155/2016/8792984

[3] Luk, L., Steinman, J. and Newhouse, J.H. (2017) Intravenous Contrast-Induced Nephropathy—The Rise and Fall of a Threatening Idea. *Advances in Chronic Kidney Disease*, **24**, 169-175. https://doi.org/10.1053/j.ackd.2017.03.001

[4] Katsiki, N., Tsioufis, C., Hahalis, G. and Athyros, V.G. (2017) Contrast-Induced Acute Kidney Injury: Beware of the Risk after Coronary Angiography. *Expert Review of Cardiovascular Therapy*, **16**, 73. https://doi.org/10.1080/14779072.2018.1421070

[5] Heyman, S.N. (2005) Regional Alterations in Renal Haemodynamics and Oxygenation: A Role in Contrast Medium-Induced Nephropathy. *Nephrology Dialysis Transplantation*, **20**, i6-i11. https://doi.org/10.1093/ndt/gfh1069

[6] McDonald, R.J., McDonald, J.S., Bida, J.P., Carter, R.E., Fleming, C.J., Misra, S., *et al.* (2013) Intravenous Contrast Material-Induced Nephropathy: Causal or Coincident Phenomenon? *Radiology*, **267**, 106-118. https://doi.org/10.1148/radiol.12121823

[7] Obed, M., Gabriel, M.M., Dumann, E., Vollmer Barbosa, C., Weißenborn, K. and Schmidt, B.M.W. (2022) Risk of Acute Kidney Injury after Contrast-Enhanced Computerized Tomography: A Systematic Review and Meta-Analysis of 21 Propensity Score-Matched Cohort Studies. *European Radiology*, **32**, 8432-8442. https://doi.org/10.1007/s00330-022-08916-y

[8] Mehran, R. and Nikolsky, E. (2006) Contrast-Induced Nephropathy: Definition, Epidemiology, and Patients at Risk. *Kidney International*, **69**, S11-S15. https://doi.org/10.1038/sj.ki.5000368

[9] Au, T.H., Bruckner, A., Mohiuddin, S.M. and Hilleman, D.E. (2014) The Prevention of Contrast-Induced Nephropathy. *Annals of Pharmacotherapy*, **48**, 1332-1342. https://doi.org/10.1177/1060028014541996

[10] Eng, J., Wilson, R.F., Subramaniam, R.M., Zhang, A., Suarez-Cuervo, C., Turban, S., *et al.* (2016) Comparative Effect of Contrast Media Type on the Incidence of Contrast-Induced Nephropathy: A Systematic Review and Meta-Analysis. *Annals of Internal Medicine*, **164**, 417-424. https://doi.org/10.7326/m15-1402

[11] Gruberg, L., Mehran, R., Dangas, G., Mintz, G.S., Waksman, R., Kent, K.M., *et al.* (2001) Acute Renal Failure Requiring Dialysis after Percutaneous Coronary Interventions. *Catheterization and Cardiovascular Interventions*, **52**, 409-416. https://doi.org/10.1002/ccd.1093

[12] Mitchell, A.M., Jones, A.E., Tumlin, J.A. and Kline, J.A. (2010) Incidence of Contrast-Induced Nephropathy after Contrast-Enhanced Computed Tomography in the Outpatient Setting. *Clinical Journal of the American Society of Nephrology*, **5**, 4-9. https://doi.org/10.2215/cjn.05200709

[13] Mula-Abed, W.S., Al Rasadi, K. and Al Riyami, D. (2012) Estimated Glomerular Filtration Rate (EGFR): A Serum Creatinine-Based Test for the Detection of Chronic Kidney Disease and Its Impact on Clinical Practice. *Oman Medical Journal*, **27**, 108-113. https://doi.org/10.5001/omj.2012.23

[14] Selistre, L.d.S., Souza, V.C.d., Dubourg, L., Wagner, M.B., Hoefel Filho, J.R. and Saitovitch, D. (2015) Contrast-Induced Nephropathy after Computed Tomography. *Jornal Brasileiro de Nefrologia*, **37**, 27-31. https://doi.org/10.5935/0101-2800.20150005

[15] Moriya, H., Mochida, Y., Ishioka, K., Oka, M., Maesato, K., Yamano, M., Suzuki, H., Ohtake, T., Hidaka, S. and Kobayashi, S. (2022) Impact of Contrast-Induced Nephropathy on Long-Term Renal Function after Coronary Angiography and Contrast-Enhanced Computed Tomography. *Cardiology and Cardiovascular Medicine*, **6**, 473-479.

[16] Alhozali, H., Toonsi, F.A., Alsubhi, J.M., Eissa, G.A., Almaghrabi, S.J., Alharbi, S.H., *et al.* (2024) Assessing the Risk of Contrast Induced-Acute Kidney Injury (CI-AKI) after Enhanced CT Scan: Single-Centre Experience. *Journal of Contemporary Medical Sciences*, **10**, 277-287. https://doi.org/10.22317/jcms.v10i4.1510

[17] Khodabandeh, H., Izadpanah Ghahremani, A., Zolfi gol, A., Jafari, N., Soleimantabar, H., Shahabi Rabori, V., *et al.* (2023) Contrast-Associated Acute Kidney Injury Following Intravenous Contrast Media Computed Tomography; New Concept and Future Directions: A Systematic Review Study on Emergencies Patients. *Journal of Renal Injury Prevention*, **13**, e32234. https://doi.org/10.34172/jrip.2023.32234

[18] Ong, M., Koh, J.J., Kothan, S. and Lai, C. (2022) The Incidence and Associated Risk Factors of Contrast-Induced Nephropathy after Contrast-Enhanced Computed Tomography in the Emergency Setting: A Systematic Review. *Life*, **12**, Article 826. https://doi.org/10.3390/life12060826

[19] Kene, M., Arasu, V., Mahapatra, A., Huang, J. and Reed, M. (2021) Acute Kidney Injury after CT in Emergency Patients with Chronic Kidney Disease: A Propensity Score-Matched Analysis. *Western Journal of Emergency Medicine*, **22**, 614-622. https://doi.org/10.5811/westjem.2021.1.50246

[20] Akman, C. and Bakirdogen, S. (2020) The Impact of Serum Creatinine, Albumin, Age, and Gender on the Development of Contrast-Induced Nephropathy in Patients Exposed to Contrast Agent Upon Admission to the Emergency Department. *Cureus*, **12**, e11051. https://doi.org/10.7759/cureus.11051

[21] McGillicuddy, E.A., Schuster, K.M., Kaplan, L.J., Maung, A.A., Lui, F.Y., Maerz, L.L., *et al.* (2010) Contrast-Induced Nephropathy in Elderly Trauma Patients. *Journal of Trauma: Injury, Infection & Critical Care*, **68**, 294-297. https://doi.org/10.1097/ta.0b013e3181cf7e40

[22] Hopyan, J.J., Gladstone, D.J., Mallia, G., Schiff, J., Fox, A.J., Symons, S.P., *et al.* (2008) Renal Safety of CT Angiography and Perfusion Imaging in the Emergency Evaluation of Acute Stroke. *American Journal of Neuroradiology*, **29**, 1826-1830. https://doi.org/10.3174/ajnr.a1257

[23] Yadollahi Fars, Y., Moghadasi, R. and Shayanpour, S. (2023) Contrast-Induced Nephropathy; An Update on Pathophysiology. *Journal of Renal Endocrinology*, **9**, e25099. https://doi.org/10.34172/jre.2023.25099

[24] Li, Q. and Pan, S. (2022) Contrast-Associated Acute Kidney Injury: Advances and Challenges. *International Journal of General Medicine*, **15**, 1537-1546. https://doi.org/10.2147/ijgm.s341072

[25] Evola, S., Lunetta, M., Macaione, F., Fonte, G., Milana, G., Corrado, E., *et al.* (2012) Risk Factors for Contrast Induced Nephropathy: A Study among Italian Patients. *Indian Heart Journal*, **64**, 484-491. https://doi.org/10.1016/j.ihj.2012.07.007

[26] Cheruvu, B., Henning, K., Mulligan, J., Klippenstein, D., Lawrence, D., Gurtoo, L., *et al.* (2007) Iodixanol: Risk of Subsequent Contrast Nephropathy in Cancer Patients with Underlying Renal Insufficiency Undergoing Diagnostic Computed Tomography Examinations. *Journal of Computer Assisted Tomography*, **31**, 493-498. https://doi.org/10.1097/rct.0b013e31802e29d9

[27] Cernigliaro, J.G., Haley, W.E., Adolphson, D.P., Jepperson, M.A., Crook, J.E., Thomas,

C.S., *et al.* (2016) Contrast-Induced Nephropathy in Outpatients with Preexisting Renal Impairment: A Comparison between Intravenous Iohexol and Iodixanol. *Clinical Imaging*, **40**, 902-906. https://doi.org/10.1016/j.clinimag.2016.04.008

[28] Lenhard, D.C., Pietsch, H., Sieber, M.A., Ernst, R., Lengsfeld, P., Ellinghaus, P., *et al.* (2012) The Osmolality of Nonionic, Iodinated Contrast Agents as an Important Factor for Renal Safety. *Investigative Radiology*, **47**, 503-510. https://doi.org/10.1097/rli.0b013e318258502b

[29] Maioli, M., Toso, A., Leoncini, M., Gallopin, M., Musilli, N. and Bellandi, F. (2012) Persistent Renal Damage after Contrast-Induced Acute Kidney Injury: Incidence, Evolution, Risk Factors, and Prognosis. *Circulation*, **125**, 3099-3107. https://doi.org/10.1161/circulationaha.111.085290