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Incidence, risk factors, and outcome of contrast-induced nephropathy following contrast enhanced computed tomography: A pragmatic, observational study

Running title: CECT and incidence, risk factors, and outcome of CIN

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Abstract

Background: Despite significant advancement in molecular properties, intravascular injection of iodinated contrast agents (CAs) is associated with the risk of contrast induced nephropathy (CIN). Additionally, the incidence, risk factors, and outcome of CIN is variable across the literature. Thus, the study assessed the incidence, risk factors, and outcome of CIN following contrast enhanced computed tomography (CECT).

Materials and Methods: This prospective observational study involved adult patients with eGFR > 60 ml/min/m^2, and undergoing CECT examination. The study was performed in the Department of Nephrology of a tertiary care institute over a period of 12-months (November 2021 to October 2022). A pre-CECT serum creatinine (sCr) and post-CECT sCr levels, assessed after 48 hours and on the seventh day, were recorded.

Results: Of 409 patients included, 12 (2.93%) developed CIN. The mean age of patients with CIN was significantly greater than those without CIN (p=0.002). Significantly greater proportion of patients with CIN had comorbidities, including hypertension (p=0.002), DM (p=0.004), anemia (p=0.007), CCF (p<0.0001), and hypotension (p<0.0001) as well as history of NSAIDs use (p<0.0001). The patients with CIN received significantly greater mean CA volume (p<0.0001) as well as had significantly higher mean sCr levels, both pre- and post-CECT (both p<0.0001). Additionally, one (0.24%) patient developed severe renal failure, requiring hemodialysis.

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE 1, 2024

Conclusion: The incidence of CIN was 2.93%. The CIN was significantly associated with advancing age, comorbidities, use of NSAIDs, greater mean CA volume, and higher mean sCr levels. Additionally, severe renal failure was rare.

Key Words: *Acute renal injury, Computed tomography, Contrast agent, Diabetes mellitus, Nephropathy*

I. Introduction

Since its inception in October 1, 1971, computed tomography (CT) has transformed the diagnostic decision making.[1] Due to its numerous advantages, its use has increased exponentially, and numerous alterations were made to get best image quality. One such alteration was the use of iodinated intravenous (IV) contrast agents (CAs). However, use of these CAs may adversely impact the renal function (RF), especially in those with known renal dysfunction (RDF).[2]

Contrast-induced nephropathy (CIN) is the RDF that takes place following the use of iodinated CAs. It is the third most predominant cause of hospital-acquired acute renal injury (ARI), and is linked to longer hospitalization as well as greater morbidity and mortality. Based on the risk factors, procedure, and definition used, the incidence of CIN ranges from less than 1% to more than 50%.[3,4]

Following the report of the first case of CIN in 1954,[5] various controversies aroused, and it was recognized that CIN has rare occurrence in patients with normal RF. Rather, ARI is observed in patients with co-existent risk factors, especially those resulting in decreased renal perfusion. Various compounding risk factors include geriatric age group, diabetes mellitus (DM), cardiovascular disease, reduced effective intravascular volume (due to liver cirrhosis, congestive heart failure, or dehydration), and nephrotoxic agents.[6] Additionally, severity of present disease heightens the risk significantly.[2]

The incidence of CIN varies across the literature. Additionally, various studies have associated the use of CAs with ARI risk, although discrepancy remains. Some authors have demonstrated an association between use of CAs and the risk of ARI among patients with known RDF.[7,8] while others have reported no such association, irrespective of baseline RF.[9,10] Though latest consensus has downgraded the CA-associated ARI risk,[11] ongoing clinical concerns remain regarding the CA-associated ARI risk, particularly among patients with RDF. Thus, we assessed the incidence, risk factors, and outcome of CIN following contrast enhanced CT (CECT).

II. Materials and Methods

This prospective observational study involved patients aged 18 years or more, of either sex, with eGFR > 60 ml/min/m^2, and CECT examination for various pathologies. The study was performed in the Department of Nephrology of a tertiary care institute located in north-east India over a period of 12-months (November 2021 to October 2022). Patients with a history of allergic reaction to CAs, eGFR < 60 ml/min/m^2, previous exposure to iodinated contrast agents, who did not give informed consent for administration of CA, and pregnant or lactating women were excluded. The study commenced after obtaining approval of the Institutional Ethics Committee and written informed consent of the patients.

Both in- and out-patients were included in the study. To manage any contrast reactions, all the necessary equipments were kept at stand-by, if required. All the patients were evaluated as per a case report form, including the demographic details, clinical history, and diagnoses. A

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pre-CECT serum creatinine (sCr) levels were recorded. All the patients received low osmolar CA (LOCA), iohexol IV (Omnipaque, GE Healthcare, Princeton, NJ). Additionally, dose of the CA used was recorded. The post-CECT sCr levels were assessed after 48 hours and on the seventh day. Post-CECT sCr levels were then compared with the pre-procedure levels to evaluate if CIN had occurred.

The Kidney Disease: Improving Global Outcomes (KDIGO) guidelines were used to define CIN as an increase in sCr of ≥ 0.3 mg/dl within 48 hours following administration of CA.[12]

The primary outcome of the study was the incidence of CIN, while the secondary outcome was either severe renal failure or death. Severe renal failure was defined as a rise in sCr to ≥ 4.0 mg/dl or the need for dialysis.[12]

Statistical Analyses

The data was analysed with SPSS (IBM, Armonk, NY, USA) version 23.0 for Windows. The categorical and continuous variables are represented as frequency (percentage) and mean (standard deviation, SD), respectively. Between group comparison of categorical and continuous variables was performed with Chi-square and independent sample t-test, respectively. A two-tailed probability value of less than 0.05 was considered as statistically significant.

III. Results

Of 409 patients included, 64.55% were males. The mean age of the study population was 52.87 (11.81) years (range: 28 – 80 years). The predominant comorbidities were hypertension (27.38%), anemia (25.18%), DM (18.09%), congestive cardiac failure (CCF, 10.51%), and hypotension (4.16%). The patients mostly received non-steroidal anti-inflammatory drugs (NSAIDs, 29.58%), and diuretics (22.49%). The mean volume of CA used was 65.43 (11.65) ml. The mean pre-, and post-CECT sCr levels were 0.95 (0.21) mg/dL, and 1.08 (0.32) mg/dL, respectively. Following CECT, 12 (2.93%) patients developed CIN. Additionally, one (0.24%) patient developed severe renal failure, requiring hemodialysis.

Table 1 depicts the comparison of patients with and without CIN. The mean age of patients with CIN was significantly greater than those without CIN ($p = 0.002$). Significantly greater proportion of patients with CIN had comorbidities, including hypertension ($p = 0.002$), DM ($p = 0.004$), anemia ($p = 0.007$), CCF ($p < 0.0001$), and hypotension ($p < 0.0001$). Likewise, significantly greater proportion of patients with CIN had history of mediations, including NSAIDs ($p < 0.0001$). The patients with CIN received significantly greater mean CA volume ($p < 0.0001$) as well as had significantly higher mean sCr levels, both pre- and post-CECT at 48 hours (both $p < 0.0001$). The mean sCr levels did not differ significantly on post-CECT Day 7. Additionally, one (8.33%) patient with CIN had severe renal failure, had sCr of 1.6 mg/dl on Day 7, and required hemodialysis ($p < 0.0001$). However, the patients with and without CIN did not differ in terms of sex ($p = 0.648$), prior exposure to diuretics ($p = 0.833$) as well as anatomical site of CECT imaging i.e., abdomen ($p = 0.923$), chest ($p = 0.922$), and head and/or neck ($p = 0.764$).

IV. Discussion

In the present study, the incidence of CIN was 2.93%. The available literature suggests variable incidence of CIN among patients undergoing CECT (2.5 to 11%).[13-16] However, the findings of two meta-analyses suggest that incidence of CIN is probably overestimated. Moos et al.[17] and Kooiman et al.[4] reported CIN incidences of 4.96% and 6.4%, respectively.

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Contradictorily, in the latest systematic review and meta-analysis of 21 propensity score–matched cohort studies, Obed et al. demonstrated absence of any higher risk of AKI following CECT.[18] In another meta-analysis of six retrospective cohort studies, Lee et al. found that the use of CA in patients with chronic kidney disease (CKD) did not result in deterioration of RF.[19]

These findings suggest the multifactorial nature of CIN, and the incidence of CIN varies based on several factors, including the population characteristics, the study design, and the nature of the contrast procedure as well as the volume, osmolarity, and route of CA administration, apart from other possible reasons.[20,21] In the present study, all patients had normal RF and underwent identical contrast procedure, CECT. The patients with CIN had significantly higher mean SCr levels, both pre- and post-CECT, than those without CIN. These findings suggest that a higher pre-CECT SCr levels can increase the CIN risk. Based on the current KDIGO guidelines, patients with CKD are at a risk of developing CIN following imaging procedures with CAs.[12] Kistner et al. observed that CIN was present in 24% and 36% patients with moderate RDF at 48 hours and at 4-10 days following CECT, respectively.[2] Additionally, use of intra-arterial CA during catheter-based angiography is associated with greater incidence of CIN relative to IV use. However, following angiography, multiple factors are implicated in CIN, and CIN may be wrongly ascribed to the CAs.[12] While, dilution of CA following IV use may offer some protection against CIN. These factors might may have led to the lower incidence of CIN observed in the present study.

Currently, the available literature does not provide clear evidence regarding the CIN with iso-osmolar CAs (IOCAs) relative to LOCAs. Initial studies reported lower incidence of CIN with IOCAs than LOCAs; however, further studies did not confirm these findings.[22-24] A study found low-risk of CIN with IOCAs than LOCAs. Contradictorily, another study showed greater incidence of CIN with IOCA administration.[24] However, the risk of CIN is greater with use of high-osmolar CA.[12] In the present study, all the patients received LOCA. Additionally, the patients with CIN received significantly greater CA volume than those without CIN. Though higher dose of CA administered through intra-arterial route is linked to greater risk of CIN,[12] similar evidence is lacking for CA administered through IV route. Thus, double-blinded randomized trials are required to confirm the safety of IOCAs versus LOCAs as well as the role of dose for IV CAs.

The advancing age is a risk factor for CIN. Likewise, in the present study, the mean age of patients with CIN was significantly higher than those without CIN. Following assessment of CIN in elderly patients, a meta-analysis reported an incidence of 13.6%,[25] and the risk of developing CIN was two-folds greater among elderly than the younger patients.[17,25] This could be attributed to the higher prevalence of comorbidities, and concurrent nephrotoxic medications among this age group.

In addition to age, the present study found that hypertension, DM, anemia, CCF, and hypotension were significantly greater among the patients with CIN than those without CIN. In their meta-analysis, Kooiman et al.,[4] and Moos et al.[17] reported that DM was associate with three- and two-fold increased risk of CIN, respectively. Also, the incidence of CIN was significantly greater in those with DM (9.3%) than those without DM (3.7%). This was attributed to disturbed auto-regulation, and wider variability in SCr observed in patients with DM.[4] Some of the meta-analyses have reported no significant association of CIN with

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE 1, 2024

hypertension,[4,17] anemia, and CCF.[17] However, in another meta-analysis, Obed et al. demonstrated that presence of hypertension was significantly associated with increased risk of CIN, while DM and CCF were not associated with CIN.[18] In their meta-analysis, Liang et al. confirmed that anemia is associated with increased risk of CIN.[26] In a prospective study, Lei et al. found that CCF, large CA volume, hypotension, hypertension, CKD, acute myocardial infarction, and age more than 75 years were independent risk factors of CIN.[27] Though there was conflicting findings, presence of DM and CKD are the confirmed risk factors of CIN, while other risk factors need to be evaluated further in well-designed studies.

Use of various medications is associated with high incidence of CIN. In the present study, prior use of NSAIDs, not diuretics, was significantly associated with CIN. Likewise, Moos et al. reported that use of NSAIDs was associated with 2.3-fold higher risk of CIN.[17] In a multicenter study, Su et al. found that prior used of diuretics and NSAIDs was not linked to CIN.[28] Most clinical guidelines recommend holding angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, NSAIDs, and diuretics prior to the CAs use. However, except for NSAIDs, holding other medications is not backed evidence, and thus require further research.[29]

In CIN, RDF is generally reversible. The RF deteriorate within 48-72 hours of CA use, and resumes to baseline level within 1-2 weeks.[3] Around 1% patients continue to have deteriorated RF for 2 months; however, the incidence of renal replacement therapy is reported to be as low as 0.06%.[12] Likewise, in the present study, one (0.24%) patient developed severe renal failure and required hemodialysis.

The present study had certain limitations. First, lack of control group did not allow us assess the definite role of risk factors. Second, due to study design, CIN was estimated at 48 hours and on seventh day following CECT, thus long-term contrast-related complications were not evaluated. Finally, exclusion of patients with CKD might have led to reduced incidence of CIN.

V. Conclusion

To conclude, the incidence of CIN in the study population was 2.93%. The CIN was significantly associated with advancing age, comorbidities (hypertension, DM, anemia, CCF, and hypotension), use of NSAIDs, previous exposure to CA, greater mean CA volume, and higher mean sCr levels, both pre- and post-CECT. Additionally, severe renal failure, requiring hemodialysis, is rarely observed.

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Competing interest:

There is no competing interest.

Authors contribution:

All authors in our study contributed to the data collection of the patients.

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ISSN: 0975-3583, 0976-2833 VOL15, ISSUE 1, 2024

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Table 1. Comparison of various clinical characteristics

Characteristics	With CIN (n=12)	Without CIN (n=397)	p
Age, years, mean (SD)	63.42 (13.69)	52.56 (11.62)	0.002
Male sex, n (%)	7 (58.33%)	257 (64.74%)	0.648
Anatomic site			
Abdomen	7 (58.33%)	226 (56.93%)	0.923
Chest	4 (33.33%)	127 (31.99%)	0.922
Head and/or neck	1 (8.33%)	44 (11.08%)	0.764
Comorbidities, n (%)			
Hypertension	8 (66.67%)	104 (26.19%)	0.002
Diabetes mellitus	6 (50%)	68 (17.13%)	0.004
Anemia	7 (58.33%)	96 (24.18%)	0.007
CCF	5 (41.67%)	38 (9.57%)	< 0.0001
Hypotension	4 (33.33%)	13 (3.27%)	< 0.0001
Medication, n (%)			
Diuretics	3 (25%)	89 (22.42%)	0.833
NSAIDs	9 (75%)	112 (28.21%)	< 0.0001
Contrast volume, ml, mean (SD)	80.42 (13.89)	64.97 (11.29)	< 0.0001
Previous contrast exposure	3 (25%)	7 (1.76%)	< 0.0001
Creatinine, mg/dL, mean (SD)			
Pre-CECT	1.14 (0.11)	0.94 (0.21)	< 0.0001
Post-CECT			
48 hours	2.06 (0.67)	1.06 (0.25)	< 0.0001
Day 7	0.9 (0.1)	0.8 (0.1)	0.548

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Dialysis, n (%)	1 (8.33%)	0 (0%)	< 0.0001