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Incidence of contrast induced nephropathy following contrast enhanced computed tomography

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ABSTRACT

Background: Contrast induced nephropathy is a major adverse event following use of non-ionic iodinated contrast medium.

Aims and Objectives: To determine the incidence of contrast induced nephropathy following use of non-ionic contrast in contrast enhanced tomographic studies and to evaluate the risk factors that can predispose development of contrast induced nephropathy

Methodology: This observational study was conducted for a period of 18 months from January 2017 to June 2018 in a total of 310 patients who underwent contrast enhanced computed tomography examination with non-ionic contrast.

Results: The mean age of patients in our study was 52.6 years ± 16.4 years (mean ± SD) (range 23 to 90 years). CIN was observed in 12 patients (3.87%) all of whom had at least one risk factor. CIN resolved in all patients by seven days without any complications. The risk factors evaluated in our study were elderly (n = 67; 21.6%), hypertension (n = 30; 9.7%), diabetes mellitus (n = 26; 8.4%), NSAID use (n = 10; 3.2%) and renal insufficiency (n = 3; 1.3%). Risk factors were hypertension in five patients (1.61%), diabetes mellitus and elderly age group in four patients each (1.29%), renal insufficiency in two patients (0.65%). None of the patients with history of NSAID use developed CIN.

Conclusion: We observed a modest risk of CIN following CECT studies. The risk factors for developing CIN were diabetes mellitus, elderly age (>65 years), hypertension and renal insufficiency. We concluded that use of non-ionized iodinated contrast media is associated with low risk of CIN and that CECT studies do not cause significant increase in CIN.

Keywords: NIL

INTRODUCTION

Contrast media have increasingly been employed in most of the computed tomographic (CT) studies. Current CT imaging is largely dependent on contrast enhanced CT (CECT) studies. With the increase in use of contrast media there has been an increase in contrast media-related adverse events. Contrast induced nephropathy (CIN) is considered as a major adverse event following intravenous iodinated contrast use. The most commonly used definition of CIN is an absolute (≥ 0.5 mg/dL) or relative (≥25%) rise in serum creatinine from baseline within 48 to 72 hours. CIN has been considered as third commonest cause for hospital acquired renal failure with an incidence as high as 11% following impaired renal perfusion and nephrotoxic medications, thus highlighting its seriousness , .CIN has also been associated with increased morbidity and mortality , .

The overall incidence of CIN in the general population is not known and is known to range from 0.6% to 4.96% from various studies2, , . The data on CIN in patients who underwent intravenous contrast agents are largely based on intra-arterial cardiac interventions in which high volume and sometime high osmolar contrast iodinated contrast media are employed. This differs to the patient population who undergo CECT studies as high amount of contrast and high osmolar contrast media are not employed. These factors may play role in development of CIN5.

There is a lacuna between evidence-based guidelines and daily practice of radiologists for CIN prevention. It is therefore essential to estimate the prevalence of CIN in patients undergoing CECT studies and to identify the population who are at risk of CIN6. Comorbidities such as diabetes mellitus, advanced age, hypertension, use of non steroidal anti-inflammatory drugs (NSAIDs) and renal insufficiency are known to increase risk of CIN5.

There is paucity of data on risk of CIN in patients undergoing CECT studies in our population. Furthermore the risk factors for CIN in our population also need to be identified. Therefore this study has been undertaken to estimate the incidence of CIN in patients undergoing CECT and to identify risk factors that predispose CIN in rural population.

AIMS AND OBJECTIVES

The objectives of the study are:

1. To determine the incidence of contrast induced nephropathy following use of non ionic contrast used in contrast enhanced computed tomographic studies.
2. To identify the risk factors that can predispose development of contrast induced nephropathy.

MATERIALS AND METHODS

Source of data:

This observational study was conducted in individuals who underwent CECT studies at the Department of Radiodiagnosis at R. L. Jalappa Hospital attached to Sri Devaraj Urs Medical College. Individuals who met the inclusion and exclusion criteria were included in the study. The study was conducted over a period of 18 months (Jan 2017 to July 2018). All the patients underwent baseline renal function test prior to CECT study.

Inclusion Criteria:

Normal renal function (defined as serum creatinine ≤1.4 mg/dL), which is the standard of care at our hospital

Exclusion Criteria:

1. Age <18 years
2. Pregnancy
3. Allergy to contrast media

Method of collection of data:

The study was approved by the institutional review board. An informed consent was taken from all the patients for their willingness to participate in the study. Prior to entering the study individuals who were planned for CECT studies underwent renal function test (serum creatinine), which was considered as baseline recording. The various CECT studies performed were CECT abdomen, CECT thorax, CECT neck, CECT kidney ureter and bladder (KUB), CT pulmonary angiography, and CECT brain. Baseline demographic data was collected. History of CIN risk factors were also recorded, which included history of hypertension, renal insufficiency, age (age > 65 years was considered as high risk), chronic use of NSAIDs, and diabetes mellitus.

Assessment of CIN

Following the CECT study a repeat renal function test for serum creatinine was performed 48 to 72 hours after the CECT study. The patients in whom there was absolute (≥0.5 mg/dL) or relative (≥25%) rise in serum creatinine from baseline were considered as positive and these patients were followed up for a period of up to 11 days to assess short term outcome, which was return to baseline serum creatinine values (Figure 1). Individuals who were lost to follow-up were excluded in the final analysis

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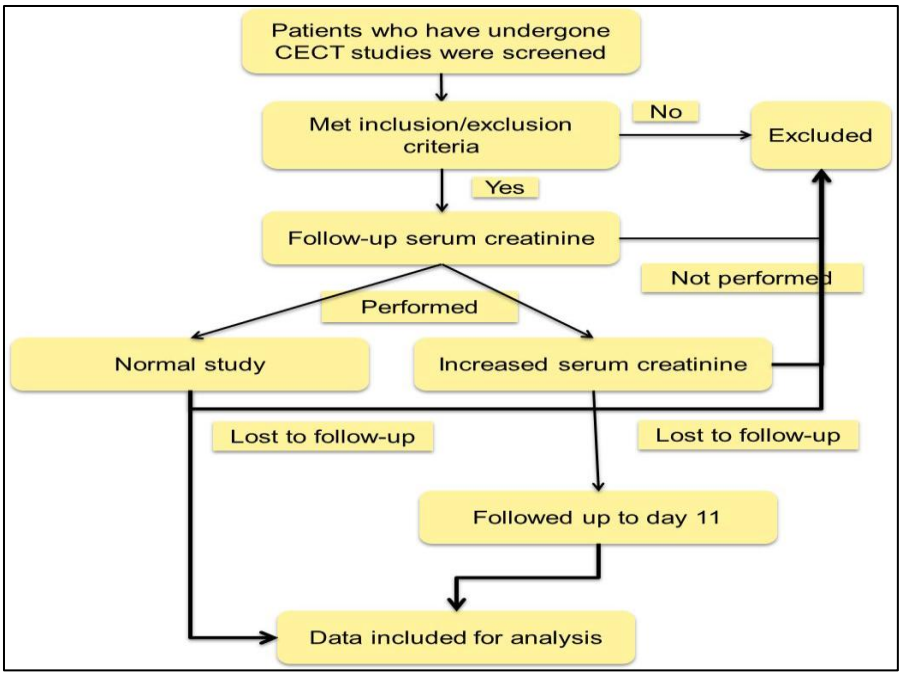

Figure 1: Study schematic

Statistical Analysis

Data was recorded into Microsoft® Excel® and was analyzed using OpenEpi® software. All the data were presented as mean ± SD. For radiation dose and mean mAs delivered, a paired t-test was performed to compare both the groups. Since each patient served as his/her own control, the results obtained in the standard-dose group was considered as standard and findings from low-dose group were compared with standard-dose group. Sensitivity and specificity for low-dose group was compared with results obtained from standard-dose group. A *P* value of <.05 was considered as statistically significant.

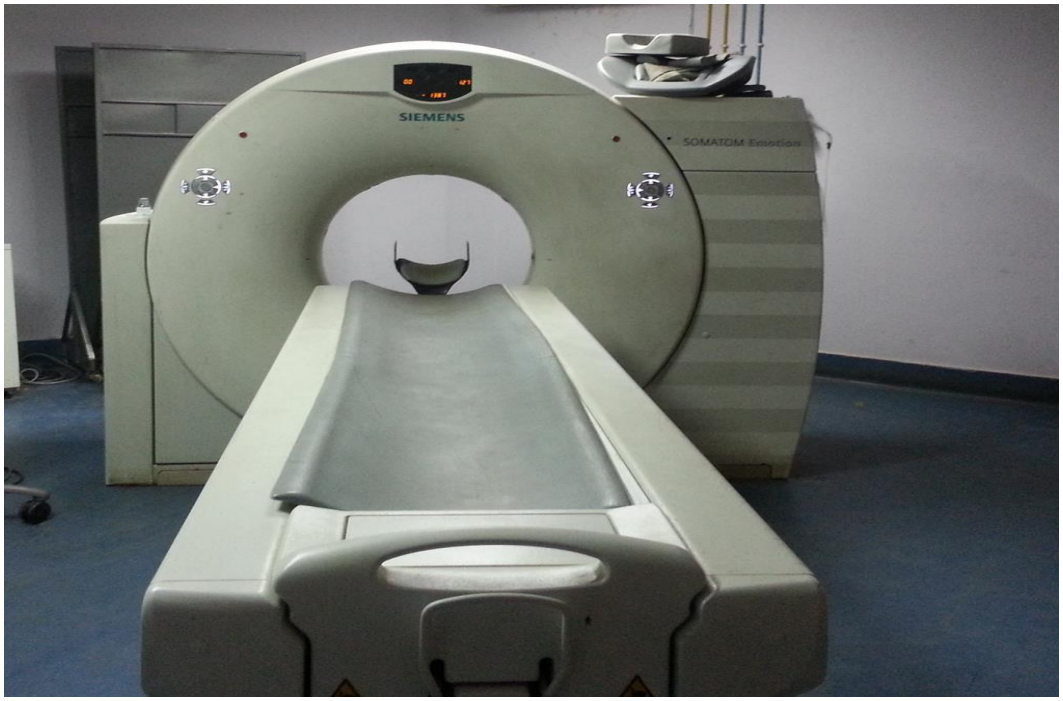

Figure 2: SIEMENS® SOMATOM EMOTION 16® CT scanner used in the study.

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RESULTS

In our study we screened a total of 1028 patients who underwent CECT studies for various indications. Among them total of 734 patients met the inclusion and exclusion criteria and were short listed for the study. Among these patients 401 patients were outpatients who could not be followed up as they did not turn up for follow-up investigations. Of the remaining 333 patients, 23 patients refused to provide consent for participation in the study. There were 310 patients who were included in the final analysis (Figure 3).

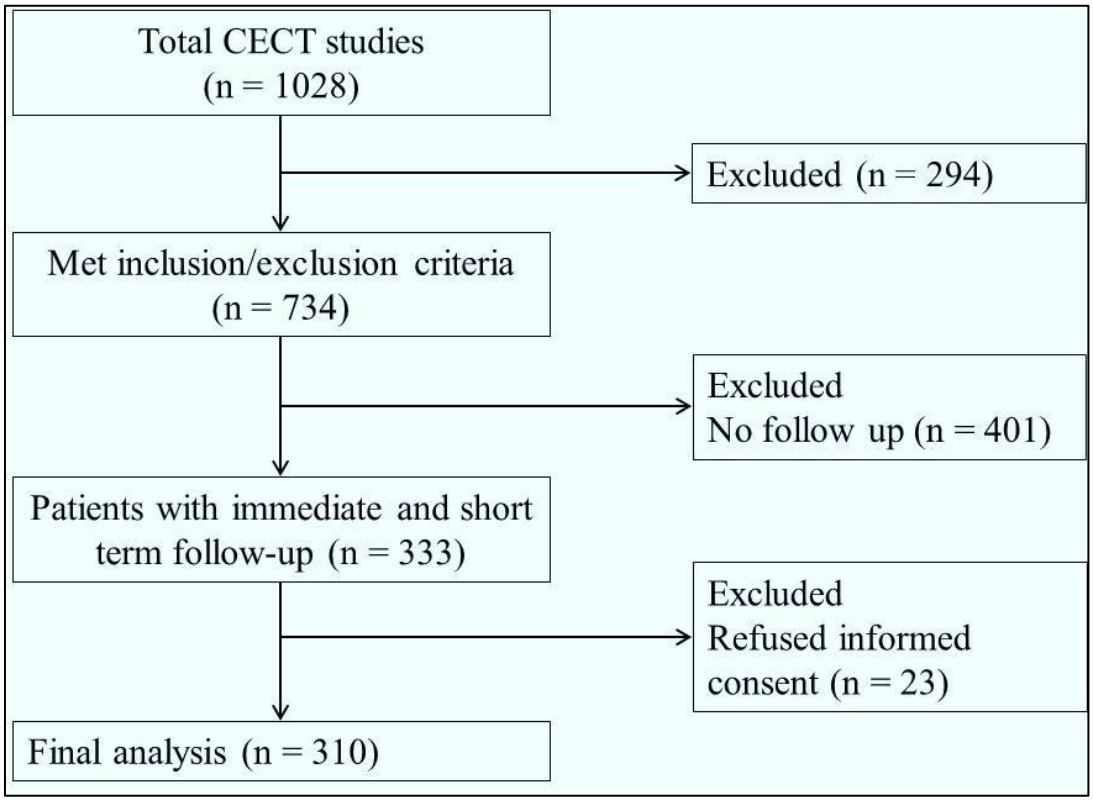

Figure 3**:** Flow chart showing screening of individuals for the study

Table 1: Gender-wise Distribution of Patients

Gender	Number of patients	%
Male	174	56.1
Female	136	43.9
Total	310	100

There were a total of 310 patients in our study. The mean age of patients in our study was 52.6 years ± 16.4 years (mean ± SD) (range 23 to 90 years). There was a slight male preponderance in our study (n = 174; 56.1%) (Table 1). The mean age of males was 51.09 ± 17.34 years (mean ± SD) and the mean age of females was 54.52 ± 14.9 years (mean ± SD), the difference of which was not statistically different (*P* = .06)

Table 2: Various CECT Examination Performed

Type of study	No of patients	%
CECT abdomen	111	35.81
CECT neck	80	25.81
CECT thorax	56	18.06

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CECT KUB/ CT urography	31	10.00
CECT Brain	29	9.35
CE PA	3	0.97
Total	310	100

CECT = contrast enhanced computed tomography; KUB = kidney ureter bladder; PA = pulmonary angiography

The commonest contrast examination performed in our study was CECT abdomen in 111 patients (35.8%) followed by CECT neck (n = 80; 25.8%), CECT thorax (n = 56; 18.06%), CECT KUB/ CT urography (n = 31; 10%), CECT brain (n = 29; 9.35%) and lastly CE pulmonary angiogram (n = 3; 0.97%).

Table 3: Risk Factors for CIN

Risk factor*	**No of patients**	**%**
Elderly†	67	21.6
Hypertension	30	9.7
Diabetes	26	8.4
NSAID use	10	3.2
Renal insufficiency	4	1.3
Total	137	44.19355

CIN = contrast induced nephropathy; NSAID = non-steroidal anti-inflammatory drug;

†elderly age was defined as age >65 years

*There were 72 patients with one risk factor, 25 patients with two risk factors and five patients with three risk factors

The mean serum initial serum creatinine level was 1.135 ± 0.163 mg/dL (mean ± SD) (range 0.8 to 1.5 mg/dL). The risk factors evaluated in our study were elderly (n = 67; 21.6%), hypertension (n = 30; 9.7%), diabetes mellitus (n = 26; 8.4%), NSAID use (n = 10; 3.2%) and renal insufficiency (n = 3; 1.3%). Risk factors were seen in total of 102 patients (32.9%). Among them, 72 patients (23.2%) had one risk factor followed by two risk factors in 25 patients (16.13%) and lastly five patients (4.84%) had three risk factors with total of 137 risk factors (Table 3).

Table 4: Initial vs Post CECT Serum Creatinine Levels

	Serum creatinine level (mg/dL)				
	Initial		Post CECT		*P*
	Mean	SD	Mean	SD	
Elderly (n = 67)	1.149	0.479	1.267	0.526	*P* = .17
Hypertension (n = 30)	1.167	0.348	1.337	0.399	*P* = .08
Diabetes (n = 26)	1.135	0.318	1.306	0.365	*P* = .07

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NSAID use (n = 10)	1.16	0.208	1.18	0.210	*P* = .8
Renal insufficiency (n = 4)	1.15	0.130	1.45	0.166	*P* = .026
Overall	1.13	0.163	1.25	0.170	

CECT = contrast enhanced computed tomography; NSAID = nonsteroidal anti-inflammatory drug; SD = standard deviation.

P<.05 considered significant

The mean initial serum creatinine levels were 1.13 ± 0.163 mg/dL (mean ± SD) and mean post CECT serum creatinine levels were 1.25 ± 0.17 mg/dL (mean ± SD). When patients with risk factors were considered, the mean initial serum creatinine levels were 1.149 ± 0.479 mg/dL (mean ± SD) in elderly individuals and mean post CECT serum creatinine levels were 1.267 ± 0.526 mg/dL (mean ± SD). The increase in serum creatinine level was not statistically significant (*P* = .17). Similarly the mean initial serum creatinine level in patients with hypertension was 1.167 ± 0.348 mg/dL (mean ± SD) and mean post CECT serum creatinine level was 1.337 ± 0.399 mg/dL (mean ± SD), which was not statistically significant (*P* = .08). The initial mean serum creatinine level in diabetics was 1.136 ± 0.318 mg/dL (mean ± SD)and post CECT serum creatinine level was 1.306 ± 0.365 mg/dL (mean ± SD), which was not statistically significant (*P* = .07) (Table 4). When patients with NSAID use were considered, the mean initial serum creatinine level was 1.16 ± 0.208 mg/dL (mean ± SD) and mean post CECT serum creatinine level was 1.18 ± 0.21 mg/dL (mean ± SD) (*P* = .8). There was however, a statistically significant increase (*P* = .026) in the post CECT serum level in patients with renal insufficiency (initial mean serum creatinine level 1.15 ± 0.13 mg/dL (mean ± SD) and post CECT mean serum creatinine level 1.45 ± 0.166 mg/dL (mean ± SD) (Figure 4)). This difference could be attributed to the significant association in development of CIN in patients with renal insufficiency and that the sample size was limited, which could have biased our results.

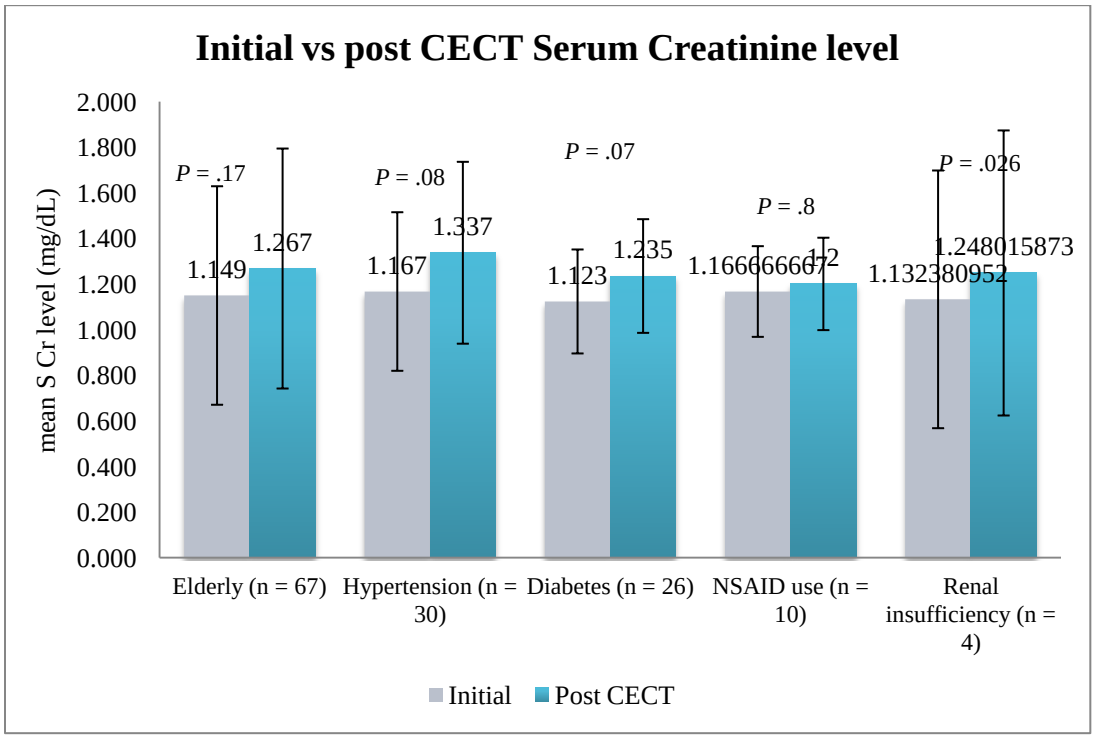

Figure 4: Initial vs post CECT mean serum creatinine levels

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Table 5: Risk Factors in Patients with CIN

Risk Factors in CIN*	No of patients	%
Hypertension	5	1.61
Diabetes	4	1.29
Elderly†	4	1.29
Renal insufficiency	2	0.65
NSAID use	0	0.00
Total	15	4.84

CIN = contrast induced nephropathy; NSAID = non-steroidal anti-inflammatory drug; †elderly age was defined as age >65 years

*10 patients had one risk factor and one patient each had two and three risk factors.

In our study CIN was observed in 12 patients (3.87%) all of whom had at least one risk factor. On follow up, CIN resolved in all patients by seven days without any complications. The risk factors were hypertension in five patients (1.61%), diabetes mellitus and elderly age group in four patients each (1.29%), renal insufficiency in two patients (0.65%). None of the patients with history of NSAID use developed CIN in our study. There were 10 patients with one risk factor (3.2%) and one patient had two risk factors (diabetes mellitus and hypertension) and one person had three factors (diabetes mellitus, elderly and hypertension) (0.3% each). Patients who developed CIN were treated with hydration and N-acetyl cysteine.

Table 6: Proportion of Patients with Risk Factors Developing CIN

Risk Factor	CIN present	CIN absent	%	P
Renal insufficiency	2	2	50	<0.001
Hypertension	5	25	16.67	<0.001
Diabetes	4	22	15.38	<0.001
Elderly*	4	63	5.97	0.001
NSAID use	0	10	0	NA

CIN = contrast induced nephropathy; NA = not applicable; NSAID = non-steroidal anti-inflammatory drug;

P = probability; Mid-P exact test

*Elderly age was defined as age >65 years

We further analyzed the proportion of patients with risk factors who developed CIN. We observed that patients with renal insufficiency had highest risk of developing CIN (50% risk; *P*<.001) followed by hypertension (five out of 30 patients; 16.67% risk; *P*<.001), diabetes mellitus (four out of 26 patients; 15.38%; *P*<.001) and lastly elderly age group (four out of 63 patients; 5.97%; *P* = .001). There were no patients with NSAID use who developed CIN. There was a significantly increased risk of developing CIN in patients with renal insufficiency, hypertension, diabetes mellitus and age >65 years. However, when compared with overall general population there was no statistically significant risk of developing CIN (Table 6).

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DISCUSSION

Our study was designed for rural population who underwent CECT studies. There was a slight male preponderance in our study with males constituting 56.1%. The mean age of patients was 52.6 ± 16.4 years (mean ± SD) (range 23 to 90 years).

Our study population is similar to study by Bhatt et al. In their study in 250 patients, they found a male preponderance as compared with females (58.8% vs 41.2% respectively) . Other studies have also shown a male preponderance5 with very few studies showing a female predominance , . The studies which showed more female population in their baseline data had specific patient population such as patients with underlying carcinoma8 or azotaemia9.

Lee et al in their study of 140838 CT examinations also reported a similar age group as our study with the mean age being 57.9 ± 15.5 years**Error! Bookmark not defined.**. However, Bhatt et al reported a lower age group of patients in their study with mean age of 41.41 ± 16.63 years (range 18 to 86 years)**Error! Bookmark not defined.**. The difference in the mean age could perhaps be explained by the rural set up in our patients and urban set up in their study.

In our study the commonest contrast studies performed were CECT abdomen (35.81%) followed by CECT neck (25.81%) and CECT thorax (18.06%). The rest of studies combined constituted about 20% total studies performed (Table 2). This pattern probably reflects the common indications seen in the rural set up. CECT abdomen is commonly performed for variety of indications, which include bowel obstruction, trauma, gastrointestinal (GI) and hepatobiliary (HPB) malignancies. CECT neck is commonly performed for head and neck cancers, which are very common in this region. Similarly, CECT thorax is commonly performed as lung infections and carcinoma lung. Recently CT urography is performed rather than intravenous urography and therefore it is seen at increasing frequency.

We observed a low risk of CIN in our study (n = 12; 3.87%). All the patients with CIN had at least one risk factor. Data from various studies has showed varying incidence of CIN, based on the criteria used1,5,6, , . Our study was designed based on the widely used definition for CIN, which was defined as an absolute (≥ 0.5 mg/dL) or relative (≥ 25%) rise in serum creatinine from baseline at 48 to 72 hours5,7, . We decided to use this criterion to define CIN as this was considered more specific, widely accepted by radiologists, easy to perform and calculate, and has less likelihood to yield false positive result and continues to remain as a commonly used definition and therefore can be easily reproducible7,11, , , .

There is a wide variability of incidence in literature ranging from 2.2% to >11%4,5,6, . Lee et al reported 2.2% incidence of CIN in their large population based study in Korea6. Similarly, Moos et al in their large meta-analysis reported a variable incidence of CIN based on the criteria used. The authors reported that when criteria of relative increase in serum creative of ≥ 25% was used the incidence of CIN was 4.72%; when the criteria used was absolute increase of serum creatinine by ≥ 0.5 mg/dL, the incidence of CIN was 2.77%; when the criteria used was relative increase of serum creatinine by ≥ 25% or absolute increase of serum creatinine by ≥ 0.5 mg/dL the incidence of CIN was 4.96%5. A meta-analysis by Bhatt et al in tertiary hospital in North India, reported 10% risk of CIN in patients undergoing CECT studies. Furthermore, they also observed that risk of CIN was 10.7% in patients who received non-ionic contrast versus 7.9% in patients who received ionic contrast7. A higher risk of CIN was reported by Mitchell et al11 and Rashid et al18. Our findings are similar to findings reported by Moos et al meta-analysis.

The risk factors evaluated in our study were elderly (n = 67; 21.6%), hypertension (n = 30; 9.7%), diabetes mellitus (n = 26; 8.4%), NSAID use (n = 10; 3.2%) and renal insufficiency (n = 3; 1.3%). Risk factors were seen in total of 102 patients (32.9%). Among them, 72 patients (23.2%) had one risk factor followed by two risk factors in 25 patients (16.13%) and lastly five patients (4.84%) had three risk factors with total of 137 risk factors.

Data from literature has shown a similar incidence of risk factors in patients undergoing CECT studying. Lee et al in their large population involving 140838 examinations in 101487 patients reported advanced age (>70 years) in 25.1% of cases, diabetes mellitus in 11.9% of patients, and hypertension in 13.7%. However, Lee et al reported a large number of users

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with NSAID usage (25.2%)6, unlike our study, where it was only seen in 3.2% of patients. Some studies have reported a higher percentage of risk factors in patients undergoing CECT studies. A large meta-analysis by Moos et al reported diabetes in about 20.2% of the pooled data. Furthermore, they observed hypertension in 55.2% of the pooled data5. Similar observations were also made by Kooiman et al, who in their meta-analysis showed presence of diabetes in 28% of pooled data, hypertension in 19%, and chronic renal disease in 35% . A similar incidence of risk factors was also reported by Kim et al, who in their study reported hypertension in >40% of patients and diabetes in >25% of patients . The reason for wide variability of risk factors could be due to the characteristics in the native patient population and the type of studies going on. In our study, significant proportion of patients present with road traffic accidents or for evaluation of malignancy and it is possible that these patients may not have significant risk factors. It also probably the reason of high percentage of elderly in our population compared to other risk factors.

he mean initial serum creatinine levels were 1.13 ± 0.163 mg/dL (mean ± SD) and mean post CECT serum creatinine levels were 1.25 ± 0.17 mg/dL (mean ± SD). When patients with risk factors were considered, the mean initial serum creatinine levels were 1.149 ± 0.479 mg/dL (mean ± SD) in elderly individuals and mean post CECT serum creatinine levels were 1.267 ± 0.526 mg/dL (mean ± SD). The increase in serum creatinine level was not statistically significant (P = .17). Similarly the mean initial serum creatinine level in patients with hypertension was 1.167 ± 0.348 mg/dL (mean ± SD) and mean post CECT serum creatinine level was 1.337 ± 0.399 mg/dL (mean ± SD), which was not statistically significant (P = .08). The initial mean serum creatinine level in diabetics was 1.136 ± 0.318 mg/dL (mean ± SD) and post CECT serum creatinine level was 1.306 ± 0.365 mg/dL (mean ± SD), which was not statistically significant (P = .07). When patients with NSAID use were considered, the mean initial serum creatinine level was 1.16 ± 0.208 mg/dL (mean ± SD) and mean post CECT serum creatinine level was 1.18 ± 0.21 mg/dL (mean ± SD) (P = .8). There was however, a statistically significant increase (P = .026) in the post CECT serum level in patients with renal insufficiency (initial mean serum creatinine level 1.15 ± 0.13 mg/dL (mean ± SD) and post CECT mean serum creatinine level 1.45 ± 0.166 mg/dL (mean ± SD)). This difference could be attributed to the significant association in development of CIN in patients with renal insufficiency and the limited sample size, which could have biased our results. We further analyzed the proportion of patients with risk factors who developed CIN. We observed that patients with renal insufficiency had highest risk of developing CIN (50% risk; P<.001) followed by hypertension (five out of 30 patients; 16.67% risk; P<.001), diabetes mellitus (four out of 26 patients; 15.38%; P<.001) and lastly elderly age group (four out of 63 patients; 5.97%; P = .001). There were no patients with NSAID use who developed CIN. There was a significantly increased risk of developing CIN in patients with renal insufficiency, hypertension, diabetes mellitus and age >65 years. However, when compared with overall general population there was no statistically significant risk of developing CIN.

TOur findings are in agreement with data reported in literature. Moos et al have shown that diabetes significantly increases the risk for developing CIN with an odds ratio (OR) of 1.87 (95% CI: 1.55 to 2.26) (P<.001). They also observed that elderly individuals (age > 65 years) have a significantly higher risk of developing CIN with an OR of 1.95 (95% CI: 1.02 to 3.70) (P = .04). Renal insufficiency was also associated with significantly increased risk of CIN with highest OR of 4.1 (95%: 2.26 to 7.42) (P<.05). A significantly increased risk of CIN was also observed with chronic NSAID use (P = .04). The authors however did not find a significantly increased risk of CIN in patients with hypertension OR 1.33 (95% CI: 0.91 to 1.95; P = .13)5. Kooiman et al also reported an increased risk of CIN in patients with diabetes mellitus (9.3% vs 3.7% in patients with and without diabetes respectively; P<.001), chronic kidney disease (8.8% vs 5.2 in patients with and without chronic kidney disease respectively; P<.001)19. Lee et al also observed an increased risk of developing CIN in patients with history of diabetes mellitus, hypertension, advanced age (≥ 70 years), used of NSAIDS and in patients with reduced renal function (renal insufficiency) with RR of 1.5 (95% CI: 1.35 to 1.66), 1.37 (95% CI: 1.24 to 1.51), 1.36 (95% CI:1.25 to 1.47), 1.07 (95% CI: 0.98 to 1.16) and 12.99 (95% CI: 11.92 to 14.17) respectively. They did not find significantly increased with NSAID

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use on multivariate analysis6, which is consistent with our finding. In our study, hypertension was associated with increased risk of CIN, which was not observed in some studies. The reason for variability of risk factors is not entirely understood. Although we observed hypertension as risk factor for CIN, it was not reported by Moos et al5. Similarly, Moos et al reported risk of CIN with use of NSAIDs, which was not observed in our study or in meta-analysis by Moos et al5,19. The most consistent risk factors include diabetes mellitus, hypertension and advanced age in most of studies5,6,19

The exact mechanism for CIN is not completely understood and it is believed to be caused due to myriad factors that ultimately play a role in deterioration of renal function. CIN is believed to result from direct damage to renal tubules and hypoxia induced damage to medullary portion of kidney19, , , . There are four primary mechanisms, which are considered to contribute to development of CIN. Initially, there is endothelium independent transient vasodilation following contrast injection, which results in release of endothelin, adenosine and various other renal vasoconstrictors. This results in renal vasoconstriction reducing renal blood supply. It is believed that contrast medium induced vasoconstriction results in renal hypoxia (reduced blood flow). This further results in spiraling reaction causing further loss of renal autoregulation, which in turn leads to reduced blood flow to outer renal medulla. This in turn results in generation of reactive oxygen species due to reduced renal blood flow and contrast media toxicity, which exceeds the normal anti-oxidant reserve of kidneys causing further renal hypoxia. It is also believed that contrast media in itself can cause direct cytotoxic effect on renal tubules resulting in renal hypoxia19,22,23, , , . It is for this reason that there is an increased risk of CIN in patients with renal insufficiency. It is also believed that diabetes mellitus results in disturbed renal autoregulation, predisposing diabetics for risk of developing CIN19. Diabetics are hypothesized to have wide variations in serum creatinine levels following contrast administration and this might be the result of higher incidence of CIN. It is possible the findings may reflect this variation in serum creatinine rather than CIN or renal damage. There is also an argument that the increased risk of CIN could possibly due to use of high osmolar contrast agents, primarily used in cardiac catheterizations and may not be true with the currently used low- or iso-osmolar contrast media, used for CECT studies . Some authors have argued that the current observational and retrospective studies evaluating risk of CIN and renal injury may not be sufficient understand the true risk posed by CIN on mortality and morbidity. Most of the studies in the literature don't have a control group and therefore, data from comparative studies using a control group may be needed. One can also argue that in general practice patients are generally followed up after CECT and this may affect the true incidence of CIN. Furthermore, patients who are hospitalized usually have one or more risk factors for CIN and this may result in skewed data on risk of CIN. These patients are also at increased risk of mortality and morbidity due to underlying disease conditions and makes causality of CIN difficult if not impossible. Further data is needed to evaluate the true risk of CIN with iodinated contrast media27, . Until we determine the exact relation between use of contrast media and development of CIN, the current data strongly supports the observation that iodinated contrast media increases the risk of CIN and that the risk factors for CIN are diabetes mellitus, hypertension, advancing age (>65 years), and renal insufficiency19,28.

Our study has certain limitations. We did not a control group in our study and our study was observational in nature. Secondly our sample was relatively smaller considering some of the large databases reported in literature. We also had fewer patients with renal insufficiency who underwent CECT study as these patients are usually not taken up for contrast studies. A greater number of patients with renal insufficiency would have been ideal. Also history of NSAID use in our population tends to be inaccurate as patients prescribed long-tern NSAIDs seldom take them. We did not evaluate the long-term impact of CECT study. Also many patients who undergo CECT studies come on OPD basis and follow-up may not be possible in those patients, thus skewing our data towards inpatients

CONCLUSION

We observed a modest risk of CIN following CECT studies. The risk factors for developing CIN were diabetes mellitus, elderly age (>65 years),

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hypertension and renal insufficiency. Serum creatinine levels reached baseline in all the patients who developed CIN within a week. We concluded that use of non-ionized iodinated contrast media is associated with low risk of CIN and that CECT studies do not cause significant increase in CIN. Further studies with control group may be needed to quantify exact risk of iodinated contrast media for developing CIN.

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