Original Research Article

Study of contrast induced nephropathy intravenous iodinated contrast media in routine diagnostic imaging in Talangana population

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Abstract

Background: Radiological Procedures utilizing intra vascular iodinated contrast media injections are being widely applied for both diagnostic and therapeutic purposes. This has resulted in an increasing incidence of procedure related contrast induced nephropathy (CIN). **Method:** 150 adult patients of both sexes having renal diseases were studied they underwent IVU/CECT technique. The eGFR of the patients was calculated and various risk factors are also noted. The contrast was sub-divided on the basis of ionicity into ionic and non-ionic. Osmolarity into HoCM and LoCM. Structure in monomer and dimer. **Results:** 25 patients had CIN and 125 had NOCIN. After contrast administration, patients showing an increase in Sr. creatinine by 25% or an absolute increase of 0.5mg/dL from pre-procedural level were diagnosed as having CIN. **Results:** There was comparative a comparative study of risk factors including dehydration, abnormal routine blood examination, DM, pre-existing renal disease, HTN, CCF, intake of No-CIN patients and results were highly significant ($p<0.001$). The contrast characteristic studies were also compared between CIN and No-CIN patients and results were highly significant ($p<0.001$). **Conclusion:** CIN is an iatrogenic disorder resulting from the administration of CM although rare in general population. Hence the benefits of diagnostic information gained from contrast-enhanced imaging need to be balanced by the potential risk of contrast-induced AKI for the individual patient.
Keywords: CIN, NO-CIN, CM, AKI, IVP/CECT, eGFR.

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INTRODUCTION

After introduction of iodinated contrast agents in the last century their use was promptly linked to acute kidney injury (AKI).[1] The presumed causal relationship between contract medium (CM) exposure and AKI has since been axiomatic in clinical care. Indeed fear of contrast induced AKI is one of the most frequent reasons why CM is withheld from the patients and thus frequently compromises the diagnostic information gained from imaging. Despite the nearly universal concern about risk contrasts. Induced nephropathy (CIN) several recent large-scale studies have questioned the general concept of CIN and relationship between DM administration, AKI and worsened clinical outcome.[2] The widely accepted primary risk factors for CIN, is pre-existing renal insufficiency with reduced nephron capacity[3] several other parameters have been identified as Diabetes mellitus, dehydration and congestive cardiac failure increase the risk for AKI. The amount of CM per nephron, approximated by mgI/GFR, is the best metric for contrast dosage toxicity.[4] However influence of these risk factors on CIN especially after intravenous CM administration has been challenged by

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recent studies. Hence attempt was made to evaluate the risk factors and contrast characteristics between CIN and No CIN so that it can be the ideal information to Radiologist.

MATERIAL AND METHOD

150 adult patients of both sexes having renal disease regularly visiting to Radiology department of mediciti Institute of Medical Sciences Ghanpur Village Medchal, Malkageri (dist), Telangana-501401 were studied.

Inclusive Criteria: Suspected clinical diagnosis of renal diseases and ready for intravenous contrast were selected for study.

Exclusion Criteria: The patients who has allergic to contrast eGFR level less than 45 mL/min/1.73m2 was excluded from study.

Method: The patients under went Intravenous Urography (IVU) or contrast Enhanced computed Tomography (CECT). The contrast investigations were performed using low osmolal non-ionic contrast media after premedication with steroids as per guide lines.

Estimated Glomurular Filtration Rate (eGER) of the patients was calculated by using modification of Diet in renal disease (MDRD) equation.[5] eGFR (ml/min/1.732) = 186 X (serum creatinine) 1.154 X (Age)-0.203 X (0.742 if female).

Appropriate laboratory investigations were advised when history suggestive of one or more risk factors was present. The below mentioned risk factors was identified- Dehydration: Patients having recent history of prolonged diarrhoea or vomiting or having limited oral intake. Previous renal surgery: History of previous renal surgery like nephrectomy, Pyelolithotomy was identified as separate risk factor. Diabetes Mellitus: DM patients on anti diabetic treatment (on oral hypoglycaemic drugs or on insulin) are treated as one of the risk factor. Hypertension: Patients of HTN on anti HTN drugs > 140/90 mm /Hg are also having risk factors. Cardiac failure: Patients having past or present history of cardiac failure was also noted as risk factor. Nephrotoxic drug intake: Patients using nephrotoxic drugs like NSAID, beta blockers, amino glycosides or amphotericin B had also risk factors. Previous contrast use: Patients undergone previous contrast study were considered as separate risk factors. Abnormal routine Blood examinations were considered as separate risk factors which included anaemic, (less than 129/dl Haemoglobin), infections like leukocytosis (value greater than 11000/ml) or patients with elevated CRP.

After completion of investigation (IVU/CECT), volume and type of contrast media, total iodine content or any reaction if occurred was recorded. Contrast was sub-divided on the basis of iconicity into ionic and non-ionic; osmolarity into high osmolal contrast media (HOCM) and low osmolal contrast media (LOMC); structure into monomer and dimmer. The following contrasts were used in the study population:-

Sodium meglumine diatriozoate (ionic, HOCM and monomer), iohexol (non-ionic, monomer and LOCM); iopamidol (non-ionic, monomer and LOCM); and ioxaglate (Ionic LOCM and dimmer) Repeated serum creatinine estimation was done 48-72 hours after contrast (IVP or CECT) investigation. After the contrast administration, the patients showing an increase in serum creatinine by 25% or an absolute increase of 0.5 mg/dL from pre-procedural level was diagnosed as having contrast induced nephropathy. In patients who were diagnosed as CIN, serum creatinine was repeated weekly till it reached the pre-procedural values. All patients with CIN were followed 4-6 weeks and were watched for features of renal deterioration like oliguria symptoms related to pulmonary oedema, or any metabolic disturbances and were recorded separately. In the present study of CIN it was observed that, there was an increase of Sr. Creatinine value by 25% from the base line or an absolute increase of 0.5 mg/dL (44.2 mm ol/L) within three days to intra vascular contrast administration.

Statistical analysis: Demographic manifestations, clinical risk factors, contrast characteristics between CIN and NO CIN were compared with z test and results were noted. The statistical analysis was carried out in SPSS software. The ratio of male and female was 2:1.

This research paper was approved by Ethical committee of Mediciti Institute of Medical Sciences, Ghanpur Village, Medchal-501401, Telangana.

OBSERVATION AND RESULTS

Table-1: Demographic Manifestations between CIN and NO CIN group – In CIN males were 17 (11.3%) and in NO CIN group 95 (63.3%) males were observed. 8 (5.3%) females in CIN group and 30 (20%) in NO CIN groups were present.

The mean value of age in CIN was 43.10 (±15.4) and 40.11 (±14.23) in NO CIN group t test 0.89 and p>0.3 (Insignificant p value). Mean value of weight in CIN was 57.40 (±12.00) and 55.20 (±9.90) in NO CIN group t test was 0.80 p<0.4 (Insignificant p value).

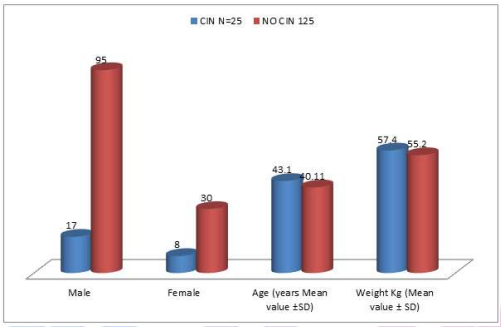
Table-2: Comparative study of clinical risk factors between CIN and NO CIN groups. Dehydration was 6 (±1.8) in CIN, 19 (±2.3) in NO CIN t test 31.3 and p<0.001. Abnormal routine blood examination was 8 (±1.3) in CIN group, 46 (±2.9) in NO CIN group, t test was 103.4 and p<0.001. Pre-existing renal disease was 5 (±1.8) in CIN group, 11 (±2.7) in NO CIN group t test was 13.8 and p<0.001. DM (diabetes Mellitus) were 5 (±1.15) in CIN group, 23 (±3.1) in NO CIN group t test 44.06 and p <0.001. Previous usage of contrast – 5 (±1.3) in CIN group, 3 (±0.7) in NO CIN group, t test 7.4 and p<0.001, HTN –

6 (±1.6) in CIN group, 15 (± 2.8) in NO CIN group, t test was 22.1 and p<0.001, Previous renal surgery – 3 (± 0.2) in CIN group, 5 (± 0.8) in NO CIN group, t test was 24.3 and p< 0.001. Cardiac failure – 3 (± 0.3) in CIN group, 2 (± 0.4) in NO CIN group, t test 14.3 and p<0.001, Intake of Nephrotoxic drugs – 4 (± 0.3) in CIN group, 15 (±1.9) in NO CIN group, t test 61.03 and p<0.001,

Table-3: Comparative study of contrast characteristic between CIN and NO CIN group. Ionicity – Ionic 5 (± 1.8) in CIN, 59 (± 3.9) in NO CIN, t test 1.7 and p<0.00, Non-Ionic – 20 (± 2.1) in CIN, 66 (± 3.2) in NO CIN, t test 84.1 and p<0.001, Osomlarity HOCM – 3 (± 1.5) in CIN, 44 (± 3.6) in NO CIN, t test 93.1 and p<0.01, LOCM – 22 (2.3) in CIN, 81 (± 4.8) in NO CIN, t test 93.7 and p<0.001, Structure Dimmer – 2 (± 0.5) in CIN, 13 (± 1.8) in NO CIN group, t test 58.04 and p<0.001, Monomer – 23 Monomer 1.3) in CIN, 112 (± 3.9) in NO CIN group, t test 36.6 and p<0.001, Total Iodine (gm) 22.35 (± 4.21) in CIN and 20.20 (± 5.33) in NO CIN, t test 2.22 and p<0.02.

Table 1: Demographic manifestations between CIN group and NO CIN group

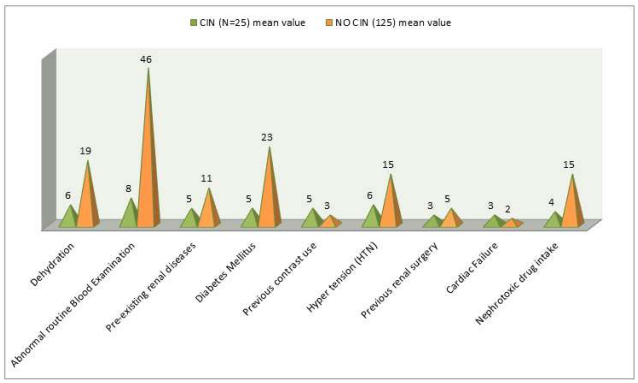
Sl No	Particulars	CIN N=25	NO CIN= 125	t test	p values
1	Male	17 (11.3%)	95 (63.3%)	-	-
2	Female	08 (5.3%)	30 (20%)	-	-
3	Age (years Mean value ±SD)	43.10 (±15.40)	40.11 (±14.23)	0.89	p>0.3
4	Weight Kg (Mean value ± SD)	57.40 (±12.00)	55.20 (±9.90)	0.86	p>0.4



Grpah 1: Demographic manifestations between CIN group and NO CIN group

Table 2: Comparative study of clinical risk factors between CIN and NO CIN groups

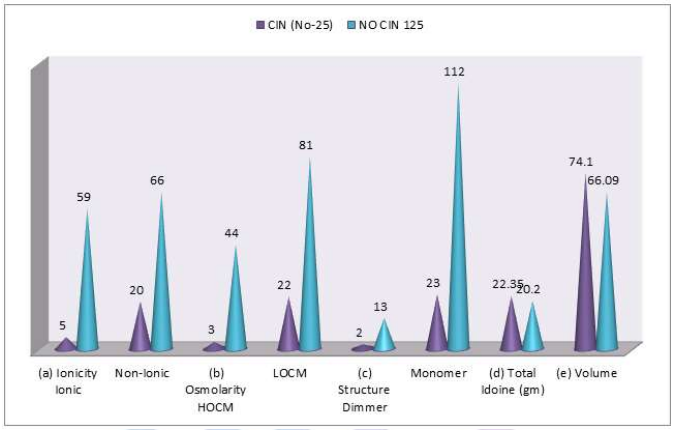
Sl No	Risk Factors	CIN (N=25) mean value	NO CIN (125) mean value	t test	p value
1	Dehydration	6(±1.8)	19(±2.3)	31.3	P<0.001
2	Abnormal routine Blood Examination	8(±1.3)	46(±2.9)	103.4	P<0.001
3	Pre-existing renal diseases	5 (±1.8)	11(±2.7)	13.8	P<0.001
4	Diabetes Mellitus	5(±15)	23(±3.1)	44.06	P<0.001
5	Previous contrast use	5(±1.3)	3(±0.7)	17.4	P<0.000
6	Hyper tension (HTN)	6 (±1.6)	15(±2.8)	22.1	P<0.001
7	Previous renal surgery	3(±0.2)	5(±0.8)	24.3	P<0.001
8	Cardiac Failure	3(±0.3)	2(±0.4)	14.3	P<0.001
9	Nephrotoxic drug intake	4(±0.3)	15(±1.9)	61.03	P<0.001



Graph 2: Comparative study of clinical risk factors between CIN and NO CIN groups

Table 3: Comparative Study of contrast characteristics between CIN and NO CIN

Contrast characteristics	CIN (No-25)	NO CIN 125	t test	p value
(a) Ionicity Ionic	5(±1.8)	59(±3.9)	107	P<0.001
Non-Ionic	20(±2.1)	66(±3.2)	84.1	P<0.001
(b) Osmolarity HOCM	3(±1.5)	44(±3.6)	93.1	P<0.001
LOCM	22(±2.3)	81(±4.8)	93.7	P<0.001
(c) Structure Dimmer	2(±0.5)	13(±1.8)	58.04	P<0.001
Monomer	23(±1.3)	112(±3.9)	136.6	P<0.001
(d) Total Idoine (gm)	22.35(±4.21)	20.20(±5.33)	2.22	P<0.03
(e) Volume	74.10(±16.17)	66.09(±15.93)	2.26	P<0.02



Graph 3: Comparative Study of contrast characteristics between CIN and NO CIN

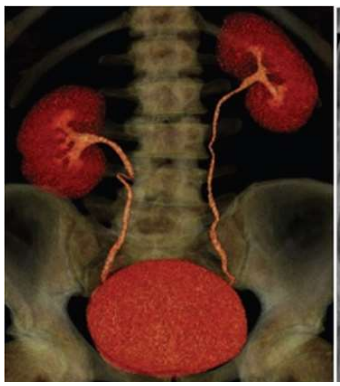


Figure 1

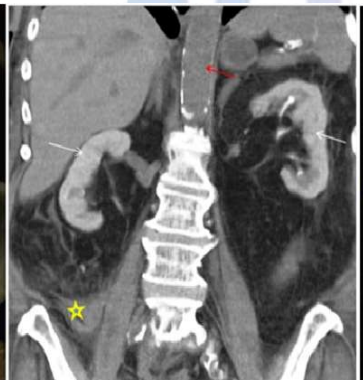


Figure 2

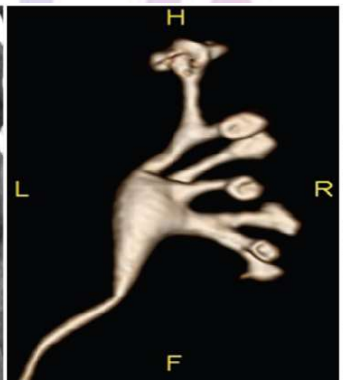


Figure 3

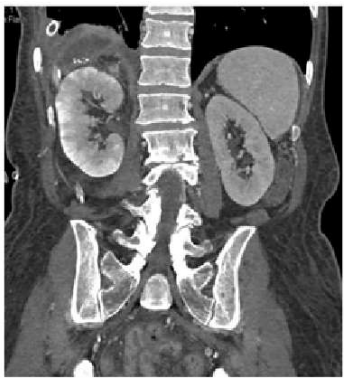


Figure 4

Figure 1: normal three-dimensional volumerendderedcolor-coded excretory phase image of a three-phase CT urogram supplemented with 10mg of intravenous furosemide demonstrates completely distended and opacified collecting systems, ureters, and bladder; **Figure 2:** Contrast-Induced Nephropathy; **Figure 3:** Normal MR urogram in adult man. Volume-rendered image of excretory phase, coronal, three-dimensional, fat-suppressed, gradient-echo sequence(repetition time, minimum; echo time, minimum flip angle, 12^0; partition thickness, 3mm) at 1.5T with 250mL intravenous normal saline, 5mg intravenous furosemide, and 0.1mmol/kg gadolinium chelate administered 5 minutes prior to acquisition demonstrates detail obtainable with excretory MR urography in the normal intrarenal collection system using diuretic and hydration. F^L fat, H^L head, L^L left, R^L right; **Figure 4:** Retained contrast in the tubukes right kidney usually means failure but may also be seen in infection.

DISCUSSION

Present study of CIN with intravenous Iodinated CM in routine diagnostic imaging in Telangana Population. There was comparison of demographic manifestation between CIN and NO-CIN patients. In CIN 17 (11.3%) male and in NO-CIN 95 (63.3%) male, In CIN 8 (5.3%) female and in NO-CIN 30 (20%) female were studied. Mean value of age in CIN was 43.10 (± 15.4) and in NO-CIN 40.11 (± 14.2), t test was 0.89 and 9 value was p<0.3 (Insignificant), similarly weight of patients in CIN was 57.40 (± 12.00) and in NO-CIN was 55.20 (± 9.90), t test was 0.86 and p<0.01 (Insignificant p value) (Table-1). The risk factors

included dehydration, abnormal routine blood examination, pre existing renal diseases, DM previous contrast use, HTN (Hypertension) previous renal surgery, Cardiac failure intake of nephtoxic drugs were compared between CIN and NO CIN patients and p value was highly significant ($p<0.001$) (Table-2). The contrast characteristics included Ionicity, Osmolarity, Structure dimmer, monomer, total Iodine (gm) volume were compared between CIN and NO-CIN and results were highly significant ($p<0.001$) (Table-3) (Figure- 1, 2, 3 and 4). These findings are more or less in agreement with previous studies.[6,7,8] Variations in the incidence of CIN present in the literature, ranging from 1.3 to 14.5%. The wide range might be due to different criteria used from diagnosing CIN, wide variation in the study sample and administration of intravascular contrast through varying routes. There is no consensus regarding clinical significance of a mild but statistically significant increase in serum creatinine following contrast administration. It is also reported that change of 0.3 mg/dL of serum creatinine has no clinical significance.[9] On the other hand, it was found that even a small increase in serum creatinine increases mortality by causing significant decrease in GFR and thus the renal function.[10] It is believed that, there is no biological significance of CIN where as few are still un-clear regarding its clinical significance. It is reported that, CIN was responsible only in 11% of kidney impairment required hospitalization when intra-arterial CM was given.[11] Some authors observed that, incidence of renal failure were 2% with Intravenous contrast.[12] In the present study there were 25 (16.6%) of patients with CIN, there was reduction of renal function was more significant with the risk renal disease heart failure as mentioned in previous studies. Dehydration increases the risk of CIN due to decreased intravascular volume resulting in decreased renal blood flow and ischemia result into renal failure. Pre-existing renal disease is an independent risk factor of nephrotoxicity and development of CIN. It is the single greatest risk factor with the severity of CIN increasing in proportion to the base line renal insufficiency. The higher is the base line serum creatinine value will have greater risk, more over there was significant increase in the risk of CIN in the patients of cardiac failure, because reduction in effective intravascular volume associated with reduced cardiac output decreases the renal perfusion and there is an increased risk of CIN.[13] Non-insulin dependent patients with diabetes who are on Biguinde therapy (metformin, glyburide, glucophage and metaglip) are at particular risk of CIN because of high viscosity of blood present in diabetes patients will enhance the CIN.

SUMMARY AND CONCLUSION

The incidence of CIN is high in an emergency department population undergoing IVP/CECT imaging risk for severe renal failure and death. The data the use of an alternate definition of CIN, an absolute increase in the serum creatinine ≥ 0.3 mg/dL may be equally sensitive and more specific for the outcome of severe renal failure. Traditional risk factors may not adequately identify patients who are at risk for CIN. Further research is needed to determine. The potential factors for delayed complication in patients who have CIN and the factors which elevate the serum creatinine levels are still un-clear.

This research paper was approved by Ethical committee of Mediciti Institute of Medical Sciences, Ghanpur Village, Medchal-501401, Telangana.

REFERENCES

1. New house JH, Roy Choudhary A – Quantitating contrast medium induced Nephropathy. Radiology 2013, 267; 4-8.
2. MC Donald RJ, MC Donald JS – Intravenous contrast material exposure is not an independent risk factor dialysis or mortality, Radiology 2014, 273; 714-725.
3. MC Donald JS, MC Donald RJ – Frequency of acute kidney injury following intravenous contrast medium administration a systemic review. Radiology 2013, 2676; 119-128.
4. Solomon R – Contrast induced acute kidney injury. Is there of risk after intravenous contrast? Clin. J. Am. Soc. Nephrol. 2008, 3; 1242-1243.
5. Tramer MR, Von Elm E – Pharmacological prevention of serious anaphylactic reactions due to iodinated contrast media: a systemic review; BMJ. 2006, 333; 675-78
6. Konen E, Konen O – Are referring clinicians aware of patients at risk from intravenous of Iodinated contrast Media? Clin. Radiol. 2002, 57; 132-135.
7. Nash K, Hafeez A – Hospital required renal insufficiency Am. J. Kidney Dis. 2002, 39'; 930-36.
8. Guitterez NV, Diaz A – Determinants of serum creatinine trajectory in acute contrast nephropathy J. Inter Cardiol. 1999, 33; 403-411.
9. R L Mehta, I A Kellum – Acute kidney injury Network; report of an initiative to improve out comes in acute kidney injury critical care 2007, 31; 401-03
10. Thomson H S and Morcus SK – Contrast media and the kidney; European society of Uro-genital Radiology guidelines Br. J. of Radiology 2003, 76; (908); 513-518.
11. Berns AS – Nephrotoxicity of contrast media kidney international 1989, 36 (4); 730-40.
12. Mehran R, Nikosy – Contrast induced nephropathy; Definition, epidemiology and patients at risk. J. of kidney international 2006, 69; 511-515.
13. Hall KA, Wong RW – Contrast induced nephrotoxicity; the effects of vasodilator therapy J. Surg. Res. 1992, 53; 317-320.

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