



Original Research Article

A hospital-based study on risk factors and outcomes of Contrast Induced Acute Kidney Injury (CI-AKI)

Shivendra Singh¹, Shiv Shankar Sharma^{2,*}, Anupam Agrawal³, Prem Shankar Patel¹, Harish Saini¹, Partha Pratim Mandal¹, Khushboo Rani¹

¹Dept. of Nephrology, Institute of Medical Sciences BHU, Varanasi, Uttar Pradesh, India

²Dept. of Nephrology Subunit, Medicine, Mahatma Gandhi Memorial Medical College, Indore, Madhya Pradesh, India

³Vardhman Mahavir Medical College & Safdarjung Hospital, New Delhi, India



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ABSTRACT

Background: Contrast-induced acute kidney injury (CI-AKI) is one of the most common causes of hospital-acquired AKI. This study was aimed to analyse the incidence of CI-AKI and associated risk factors in hospitalized patients undergoing CT or Catheter related contrast based procedures.

Materials and Methods: This cross-sectional observational study was conducted between September 2016 and August 2018. Hospitalized patients of either sex, aged >18 years scheduled for contrast-enhanced computed tomography (CECT), peripheral angiography (PAG), percutaneous transluminal coronary angioplasty (PTCA), or coronary angiography (CAG), with eGFR >30 were evaluated for CI-AKI after excluding other causes of AKI.

Results: A total of 300 patients were enrolled, of which 266 patients completed the study (CECT/PAG, n=138; PTCA/CAG, n=128). The mean age of the patients was 45.85 ± 15.14 years and the majority of patients were males (n=164, 61.6%). The incidence of CI-AKI was 15.6% (n=41). A total of 28.8% of patients with diabetes and 31.1% patients aged >60 years developed CI-AKI. Overall, the increasing contrast volume significantly (p=0.002) increased the incidence of CI-AKI. The incidence of CI-AKI in patients mild renal dysfunction increased significantly (from 5.5% at <50ml to 44% at 150-200 ml) with the increasing volume of contrast, and was significantly higher (38.8%) in patients with moderate renal dysfunction.

Conclusion: Results showed that radio-contrast related procedure carries a significant risk of nephropathy and patients with diabetes, pre-existing renal dysfunction, and advanced age are at higher risk of CI-AKI.

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1. Introduction

The first case of contrast-induced acute kidney injury (CI-AKI) was reported several decades ago¹ and since then radio contrast media has been one of the commonest causes of hospital-acquired AKI.² Several different definition and classification criteria have been proposed for CI-AKI leading to marked variation in the reported incidence and outcomes of CI-AKI. Pre-existent renal insufficiency, diabetes mellitus, advanced age, reduced left ventricle

systolic function, advanced heart failure, and acute myocardial infarction are the major non-modifiable risk factors for CI-AKI; while there are few factors that are potentially modifiable factors like volume and type of contrast media, concomitant use of nephrotoxic medications, hypotension, dehydration, hypo-albuminemia, anaemia etc.³⁻⁵

In the current era when oral or intravenous hydration has become routine for patients undergoing the contrast-related procedure, the nephrotoxic potential of contrast agents is often questioned. The last decade has seen a new-found interest in CI-AKI as, amidst all this evidence of CI-AKI

* Corresponding author.

E-mail address: drshivssharma@gmail.com (S. S. Sharma).

being associated with morbidity and mortality, few studies suggested that CI-AKI actually is not a disease but probably a chance occurrence.^{6,7}

The present article reports results of a study that was aimed to analyse the incidence of CI-AKI, the impact of its various known risk factors, distribution of CI-AKI in catheter-based coronary intervention groups and peripheral computed tomographic angiography (PCTA) based groups, and evaluation of the outcomes of patients who develop a contrast-associated renal injury.

2. Materials and Methods

This was a cross-sectional observational study conducted at the Department of Nephrology, Sir Sunderlal Hospital, Institute of Medical Sciences, Banaras Hindu University, Varanasi, India (a super-speciality teaching institute) between September 2016 and August 2018. Patients of either sex, aged more than 18 years who were scheduled for contrast-enhanced computed tomography (CECT) or peripheral angiography (PAG) and percutaneous transluminal coronary angioplasty (PTCA) or coronary angiography (CAG), with eGFR more than 30 were eligible for the study. Patients who underwent dialysis in the last 3 months due to any reasons, patients with baseline eGFR <30 ml/min/1.73m² (by CKD-EPI equation), patients with renal allograft, dehydrated at the time of procedure, patients with possibility of AKI secondary to other pathologies (like intrinsic renal diseases, obstructive uropathy, patients in shock, acute pulmonary oedema, sepsis, burn, poly-trauma, acute myocardial infarction, acute respiratory distress syndrome, severe congestive cardiac failure [EF <40%] or other acute concurrent illness which could alter renal parameters) were excluded from the study.

The study protocol was approved by the institutional ethics committee. The study was conducted in accordance with the ethical principles that have their origin in the Declaration of Helsinki and its latest amendments 2013. Written informed consent was obtained from each study participant.

A detailed history was obtained and clinical examination was conducted with special reference to comorbid conditions, previous contrast exposure, other administered drugs, and hydration status. Serum creatinine was measured and eGFR was calculated by using CKD-EPI formula prior to radiocontrast exposure, after 48 hours and after one week. In case of patients having serum creatinine elevated in range of CI-AKI defining criteria even at seventh day, serum creatinine was repeated at fourteenth day again to assess the occurrence and outcome of CI-AKI which was defined as per 2012 European Society of Radiology criteria as an increase of serum creatinine by >25% or an increase in absolute value by >0.5 mg/dL from the pre-procedural values between second to seventh day after contrast exposure. For all procedures done in

the current study low osmolar non-ionic contrast Iohexol (Omnipaque® of GE lifesciences) was used. Details of the type of radiological/catheterization laboratory investigation and/or intervention done, the outcome of procedures and the contrast volume utilized were recorded.

Baseline characteristics were assessed with standard descriptive statistics. The data were analysed using Statistical Package for Social Sciences (SPSS version 19.0, IBM). Demographic characteristics were presented for continuous variables as means and standard deviations and categorical variables as frequencies and percentages. CI-AKI and non-CI-AKI patients were compared using the Chi-square test for categorical variables and Student's t-test for continuous variables. Multivariate predictors of CIN were identified by logistic regression using stepwise selection. A two-sided 95% confidence interval (CI) was constructed around the point estimate of the odds ratio (OR). Extended Mantel-Haenszel test for trend was used for analyzing the contributory effect of more than one factor present in the association. A p value of less than 0.05 was considered as statistically significant.

3. Results

A total of 300 patients (CECT/PAG, n=150; PTCA/CAG, n=150) were enrolled in the study of which 266 patients completed the study (CECT/PAG, n=138; PTCA/CAG, n=128). The incidence of CI-AKI was 15.6% with 41 patients developing CI-AKI. The mean age of the study population was 45.85 ± 15.14 years with the majority of patients being males (n=164, 61.6%). No statistically significant difference in CI-AKI incidence was observed between males and females (p=0.73) (Table 1). There was no significant difference (p>0.05) in the incidence of CI-AKI between patients undergoing peripheral CT, peripheral angiography or CAG/PTCA (Table 1). The outcome of CI-AKI in most of the patients was fair with 65% patients having a recovery of renal function at 1 week and 95.12% by the end of 2 weeks. Dialysis-requiring CI-AKI was seen in three (1.12%) patients.

A total of 118 patients had diabetes, of which CI-AKI was noted in 34 (28.8%) patients. More than one third (n=42) of patients with diabetes also had mild to moderate renal dysfunction. Of these, 18 (42.0%) patients developed CI-AKI which was statistically significant (p=0.0013). A total of 45 patients were older than 60 years, of these 14 (31.1%) developed CI-AKI (which was statistically significant (p=0.04) (Table 1).

Seventeen (28.81%) of 59 patients receiving two or more nephrotoxic drugs developed CI-AKI (P<0.05). The same significant incidence of CI-AKI was not seen in patients receiving the single nephrotoxic drug. Of the evaluated nephrotoxic drugs, renin-angiotensin-aldosterone system (RAAS) inhibitors were most commonly co-prescribed (n=103) and 27 of these patients (26.21%) developed CI-AKI

(Table 1). This was not significant on multivariate analysis (Table 3). Sixty-two patients were receiving diuretics and 25.8% of them developed CI-AKI ($p=0.051$), while 16 patients received aminoglycoside of which five (31.25%) patients developed CI-AKI(0.0959) (Table 1).

In the present study, increasing contrast volume significantly ($p=0.002$) increased the incidence of CI-AKI. The incidence rate was highest in patients receiving 150-200 ml contrast (33.33%) and was least in the patients receiving less than 50ml contrast (6.3%). The incidence of CI-AKI in mild renal dysfunction patients was not statistically significant but the incidence increased significantly (from 5.5% at <50ml to 44% at 150-200 ml) with the increasing volume of contrast. The cumulative incidence of CI-AKI in patients with moderate renal dysfunction was significantly high (38.8%). The incidence increased further and highly significantly with the increasing volume of contrast used (27.2% with <50ml of dye and 64.2% with 150-200ml of dye) (Table 2).

The incidence of CI-AKI was 18.4% in hypertensive patients, 18.6% in mild to moderate congestive heart failure patients and 11% in anaemic patients (Table 1) and these were not found significant risk factors on either multivariate analysis (Table 3). The incidence of CI-AKI increased with the increasing cumulative number of risk factors. Incidence was minimum (2.2%) in patients with no risk factors and maximum in patients with 4 risk factors (40.0%). This finding was statistically significant ($p<0.001$).

On multivariate analysis diabetes followed by use of contrast volume over 150 ml, pre-existing moderate renal dysfunction (eGFR-30-60) and age over 60 years were found to be statistically significant risk factors (Table 3).

4. Discussion

The incidence of CI-AKI reported by varies widely depending on the definition and cut-off values used.^{8–10} In the present study the cumulative incidence of CI-AKI was 15.41%. In a previous study by Bhatt et al, the incidence CI-AKI of 10% was reported in outpatients with relatively low risk after excluding patients with GFR <45ml.¹¹ McDonald et al compared the incidence of AKI between patients, after CT scan with or without contrast and concluded that CI-AKI was inversely proportional to baseline eGFR and varied from 1.2% to 14%.¹² Contrary to these previous findings and our findings, Aycock RD et al¹³ in their meta-analysis and Hitchin et al¹² in their large study found similar frequencies of AKI in patients who did or did not receive contrast prior to CT scan. These researchers concluded that patients' comorbid illnesses and other coexisting factors contribute to the development of AKI rather than the use of contrast material.^{12,13} Many possible reasons have been proposed for these varying results. Nephroprotective practice patterns may be partially responsible for the observations in these studies as clinicians are likely to avoid contrast media

and employ aggressive preventive measures to patients with risk factors of CI-AKI.¹³ The incidence may also get affected due to different criteria used for diagnosing CI-AKI, and administration of intravascular contrast through varying routes (venous/arterial). One additional factor of discrepancy between the present study and above studies was that the meta-analysis included various intervention trials of prospective preventive¹³ and therapeutic strategies meant for CI-AKI, while the present study an observational study.

The incidence of CI-AKI in patients undergoing PAG and CECT group was 20% and 10.2%, respectively and the difference was not statistically significant. Similar to our findings, Luciano et al reported an incidence of 4% to 13% in CECT and PAG groups, respectively.¹⁴ Another meta-analysis of 42 studies reported the pooled incidence of CI-AKI as 4.2%.¹⁵ The incidence in our study was higher than this meta-analysis, possibly due to our study included hospitalized patients with more number of risk factors while the meta-analyses included studies of CI-AKI in all populations hospitalized and not hospitalized.

The CI-AKI and CAD share the common risk factors like old age, diabetes, renal dysfunction, congestive cardiac failure etc. Due to these factors, the occurrence of CI-AKI is more following coronary intervention than in average population undergoing CECT. Also, a larger volume of CM is required if CAG is converted to PTCA. The incidence of CI-AKI in CAG and PTCA (combined) in our study was 17.9% while the incidence in PTCA patients was 23.8% which was not statistically significant ($p<0.321$). Solomon et al reported that the incidence of CI-AKI in CAG patients was substantially higher than the peripheral contrast procedure groups.^{15,16} Weisbord et al¹⁷ noted the incidence of CI-AKI around 10%, while Mitchell et al¹⁸ in their prospective study of CI-AKI in outpatient settings found an incidence of 11%. The discrepancy in these reported incidences is wide and can be due to multiple factors like type of contrast, use of preventive measures and difference in the patient selection and exclusion criteria.

Patients with the pre-existing renal disease usually have a decreased vasodilatory response and delayed clearance of contrast media as compared to persons with normal renal function. The reduced number of nephrons increases the amount of dye that needs to be eliminated through individual nephrons. The CIN Consensus Working Panel labels pre-existing moderate renal dysfunction (eGFR <60 ml/min/1.73m²) as the most important risk factor to predict the risk of CIN in patients receiving iodinated contrast media.⁵ In our study, the incidence of CI-AKI in patients with mild renal dysfunction was 16.66% while the incidence in moderate renal dysfunction was 38.8%. Similar to our study, Dangas et al. reported in their study of CIN in patients undergoing PCI that the incidence of CIN was significantly lower in patients without CKD (13.1%) than in those with

Table 1: Incidence of CI-AKI

Parameter	Total (N=266)	CI-AKI (n=41)	P value
Sex			0.73
Males	164	24 (14.6)	
Females	102	17 (16.7)	
Age (years)			-
18-30	44	4 (10.0)	
31-40	59	6 (10.1)	
41-50	67	9 (13.4)	
51-60	51	8 (15.6)	
61-70	24	7 (29.0)	
>70	21	7 (33.3)	
Age >60 (years)	45	14 (31.1)	0.04
CECT	98	10 (10.2)	0.26
PAG	40	8 (20.0)	0.48
CAG/PTCA	128	23 (17.9)	0.51
Risk factor			0.003
Mild pre-existing CKD (eGFR- 61-90)	90	15 (16.66)	
Moderate pre-existing CKD (eGFR 31-60)	55	21 (38.18)	
Diabetes	118	34 (28.81)	
Hypertension	152	28 (18.4)	
Age >60 years	45	14 (31.1)	
Anaemia	141	16 (11.3)	
Nephrotoxic drugs	136	34 (25.0)	
Diabetes	118	34 (28.81)	0.014
Diabetes and CKD (eGFR 30-90)	42	18 (42.0)	0.0013
Hypertension	152	28 (18.4)	0.50
CHF (EF-40-60%)	43	8 (18.6)	0.57
Anaemia	141	16 (11.3)	0.32
Nephrotoxic drug			
All Nephrotoxic drugs	136	34 (25.0)	0.056
RAAS inhibitors	103	27 (26.21)	0.050
NSAIDs	42	7 (16.66)	0.85
Aminoglycosides	16	5 (31.25)	0.0959
Diuretics	62	16 (25.8)	0.051
Other nephrotoxic drugs	24	4 (16.66)	0.91
Patients receiving 2 or more nephrotoxic drugs	59	17 (28.81)	0.049
Number of risk factor			0.001
No risk factors	45	1 (2.2)	
1 Risk factor	77	7 (9.09)	
2 Risk factors	73	12 (16.4)	
3 Risk Factors	51	13 (25.4)	
4 Risk Factors	20	8 (40.0)	

CAG, coronary angiography; CECT, contrast-enhanced computed tomography; CHF, congestive heart failure; CI-AKI, contrast-induced acute kidney injury; CKD, chronic kidney disease; EF, ejection fraction; eGFR, estimated glomerular filtration rate; NSAIDs, nonsteroidal anti-inflammatory drugs; PAG, peripheral angiography; PTCA, percutaneous transluminal coronary angioplasty; RAAS, renin-angiotensin-aldosterone system.

CKD (19.2%).¹⁹ A retrospective analysis of the Mayo Clinic PCI registry revealed that all patients with baseline serum creatinine levels of ≥ 2.0 mg/dL had a significant risk of AKI irrespective of the presence of other risk factors.²⁰ Contrary to the reported high risk associated with radiocontrast in CKD, in a Mayo's clinic proceedings, McDonald et al reported no increased risk of CI-AKI in CKD patients if hydration was used but this retrospective analysis was done for patients undergoing CECT only and no interventional procedures were included while our study population was

admitted patient undergoing either interventional or non interventional contrast related procedure and most were with more than one risk factor for CI-AKI.²¹

Diabetics with pre-existing renal disease are reported to have a four-fold increase in the risk for CI-AKI then diabetics with normal renal function.²² In the present study, diabetes was found to be the most significant risk factor on multivariate analysis and 28.8% of the studied diabetics developed CI-AKI. The incidence of CI-AKI in hypertensive patients was 18.4%; however, in the previous

Table 2: incidence of CI-AKI according toradiocontrast media volume and renal dysfunction

Parameter	Total (N=266)	CI-AKI (n=41)	P value
Contrast media volume			0.002
<50 ml	47	3 (6.3)	
50-100 ml	91	11 (12.0)	
100-150 ml	101	18 (17.8)	
150-200 ml	27	9 (33.3)	
Mild CKD (eGFR 60 to 90)			0.006
<50 ml (n=47)	18	1 (5.5)	
50-100 ml (n=91)	32	3 (9.37)	
100-150 ml (n=101)	31	7 (22.5)	
150-200 ml (n=27)	9	4 (44)	
Moderate CKD (eGFR 30 to 60)			0.046
<50 ml (n=47)	11	3 (27.2)	
50-100 ml (n=91)	15	4 (26.6)	
100-150 ml (n=101)	15	5 (33.3)	
150-200 ml (n=27)	14	9 (64.2)	

CI-AKI, contrast-induced acute kidney injury; CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate.

Table 3: Multivariate logistic regression analysis of the risk factors

Variable	Odds ratio	95% CI	P value
Mild pre-existing renal dysfunction (eGFR 61-90mL/min/1.73 m ²)	0.589	0.198-1.458	0.239
Moderate CKD (eGFR <60 mL/min/1.73 m ²)	3.9	1.3 -11.0	0.025
Diabetes	10.2	3.4 -31.0	0.010
Contrast volume more than 150ml	5.12	2.7-14.12	0.012
Hypertension	1.4	0.4-5.5	0.5
Mild-moderate CHF (EF 40-60)	0.235	0.021-2.114	0.996
Age >60	6.3	1.8 -22.5	0.021
Anaemia	1.259	0.825-2.235	0.089
Nephrotoxic drugs	1.444	0.235-2.457	0.066
Female gender	0.9	0.3 -2.5	0.8

CHF, congestive heart failure; CKD, chronic kidney disease; EF, ejection fraction; eGFR, estimated glomerular filtration rate.

report by Conen et al reported the incidence of CI-AKI of 12% in hypertensive patients which was statistically significant.²³ It has been proposed that hypertension leads to alterations in the intra-renal expression of vasoactive mediators, such as the RAAS or nitric oxide expression making patients susceptible to contrast-related injury. In the present study, 11.3% of patients with anaemia had CI-AKI. Anaemia may cause AKI due to its hypoxic effects on kidneys and its contribution as a risk factor for CI-AKI has been a matter of interest. However, the studies have contradictory findings.²⁴⁻²⁶

In the present study, 18% of patients with mild to moderate CHF had CI-AKI. Studies have shown that a reduced LVEF and advanced CHF are independent risk factors for CI-AKI.^{27,28} Dangas et al¹⁹ showed that an LVEF<40% is an independent predictor of CIN, while contrary to this Toprak et al²⁹ reported that if the LVEF>30% has no significant effect on the development of CIN. This difference from the present study could be due to the exclusion of patients with acute myocardial infarction and severe CHF. The incidence of CI-AKI was 31% in

elderly patients in our study which was significantly higher than the average of 15.41%. Many CI-AKI risk calculation protocols include age as a factor for predicting CI-AKI and studies have confirmed older age an independent risk factor.³⁰⁻³²

More than half of the patients were receiving at least one nephrotoxic drug and around 28.8% of patients receiving two or more nephrotoxic drugs developed CI-AKI. Ho et al studied the impact of poly-pharmacy on contrast nephropathy and reported that around 45% patients from their cohort were receiving nephrotoxic drugs from different subclasses and this poly-pharmacy was associated with increased incidence of CI-AKI.³³ The most commonly used nephrotoxic drugs in our study were RAAS inhibitors followed by diuretics. RAAS-inhibitors have vasodilatory effect on efferent arterioles while contrast media causes vasoconstriction of the afferent arteriole decreasing the glomerular filtration.³⁴ In the present study, amongst the individually studied nephrotoxic drugs, the highest incidence of CI-AKI was seen with the use of aminoglycoside antibiotics. CI-AKI is primarily a tubular

disorder and aminoglycosides are amongst the most well-known tubule-toxic drugs so complementary nephrotoxicity was expected. Aminoglycoside toxicity may also increase in elderly patients and patients with diabetes increases the risk of CI-AKI. Lopez et al reported that up to 25% of all patients who receive aminoglycoside therapy may develop nephrotoxicity.³⁵

In the present study, concurrent NSAIDs use was not associated with significantly increased incidence of CI-AKI. Hörl et al. in a systematic review on renal impact of NSAIDs opined that due to the prostaglandin inhibitory action NSAIDs are associated with vasoconstriction of the afferent arteriole leading to possibility of renal insult and similar action is attributed to contrast agents.³⁶ It is, therefore, recommended to discontinue selective or non-selective NSAID therapy 48 hours before administration of radiocontrast agents in those patients. The outcome of most patients in our study was benign with no mortality and 97% of patients showing recovering by the end of two weeks observation period. Few retrospective studies have demonstrated an association between CI-AKI and increased short-term mortality.^{37,38}

There are many different risk scoring systems proposed for predicting CI-AKI, but due to the lack of prospective good quality evidence, most international guidelines recommend against using risk scoring systems. Despite these controversies, there are many scoring systems that have been in use since decades of which the most prominent ones are those developed by Bartholomew and colleagues and Mehran risk score.^{39–41} Bartholomew used a database of over 20,000 patients to develop a risk scoring system of eight variables (creatinine clearance <60 ml/min, use of IABP, urgent coronary procedure, diabetes, CHF, hypertension, peripheral vascular disease, and contrast volume).³⁹ Similar to this, Mehran risk score for CI-AKI too uses eight variables (hypotension, use of IABP, CHF, age >75 years, anemia, diabetes mellitus, contrast volume and baseline serum creatinine >1.5 mg/dL).^{40,41} In the present study, we excluded most risk factors of these calculators still we found a higher incidence of CI-AKI in patients with more number of risk factors. On using extended Mantel-Haenzel test the incidence of CI-AKI was least in patients with no risk factors and highest in patients with 4 or more risk factors.

Contrast media volume is one of the most important modifiable risk factors of CI-AKI and is a key contributor of CI-AKI in patients with normal renal function. In the present study, the minimum incidence of CI-AKI has seen with contrast volume <50 ml and the incidence of CI-AKI increased with increasing volume of contrast. Several studies support that even patients with no risk factors may develop CI-AKI when a large volume of contrast was used.^{42–45} In our study on extended Mantel-Haenzel test, we found a very significant correlation of increasing contrast volume with CI-AKI in patients with pre-existing renal

dysfunction. We found that even patients with significant renal dysfunction had a low incidence of CI-AKI when contrast volume used was less. Similar to our observation, Kane et al used an ultra-low volume of contrast for PCI and showed that even in patients with severe renal dysfunction low contrast use can prevent CI-AKI.⁴⁶ Our findings were similar to observations by Mager et al and Ogata et al.^{45,47}

Authors acknowledge several limitations of the study. This being a single-centre prospective observational study, the evidence may not be as compelling as that obtained in a randomized controlled trial or a multi-centric study. The primary objective of the study was to analyze occurrence and outcomes of CI-AKI in hospitalized patients and to avoid confounding factors, illnesses directly associated with AKI were excluded, which may not be the case in real clinical settings. The study enrolled only admitted patients who in general were expected to be sicker than the outpatient population. The open-label nature of the study could have led to a positive reporting bias with a higher incidence of CI-AKI. To minimize the bias associated with multiple risk factors, we used propensity score for matching and multivariate analysis; however, there could be few factors that were not included and could have affected results.

5. Conclusion

Results showed that, in hospitalized patients, radio-contrast related procedure carries a significant risk of nephropathy and patients with diabetes, pre-existing renal dysfunction, and advanced age are at higher risk of CI-AKI. A higher volume of contrast may further increase the risk for the patients. As the number of risk factors increased, the incidence of nephropathy also increased. Strategies to prevent CI-AKI should target identification and possible correction of risk factors while trying to utilise the minimum volume of contrast possible.

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8. Conflict of Interest

The authors declare they have no conflict of interest.

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Author biography

Shiv Shankar Sharma, Professor

Anupam Agrawal, Senior Medical Officer Nephrology

Prem Shankar Patel, Senior Registrar Nephrology

Harish Saini, Senior Registrar Nephrology

Partha Pratim Mandal, Senior Registrar Nephrology

Khushboo Rani, Senior Registrar Nephrology

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