

Original Article

Assessment of Effect of Radio Contrast Enhanced Computed Tomography on the Renal System in Hypertensive Patients

Meeta Burande¹, Ravindra Patil², Archana Dhavalshankh³, Sunita Patil⁴

^{1,2,3,4}Department of Pharmacology, Dr. D. Y. Patil Medical College, Hospital & Research Centre, Kolhapur, Maharashtra, India

ABSTRACT:

Background: Contrast media (CM) are increasingly used in diagnostic imaging procedures. This results in the rising incidence of iatrogenic renal function impairment caused by the exposure to CM, a condition known as contrast induced nephropathy (CIN). We planned the present study to assess the effect of contrast agents on renal functions based on serum creatinine and creatinine clearance in patients with hypertension who are well controlled on medications. **Materials & methods:** We planned the present study to assess the effect of contrast agents on renal functions based on serum creatinine and creatinine clearance in patients with hypertension who are well controlled on medications. We included a total of 60 patients and divided them broadly into two groups; Group 1 and group 2. Group 1: All patients without a pre-existing renal disease non diabetic non hypertensive. Group 2: Known Hypertensive patients on treatment but not known diabetic. Incidence rate of CIN was calculated for each group. All the results were analyzed by SPSS software. **Results:** The total incidence of CIN in the group 1 was 13.3% and 26.7 in group 2. We observed non-significant results while comparing the serum creatinine values in between group 1 and group 2. **Conclusion:** Hypertension is associated with significantly increased risk of contrast nephropathy.

Key words: Computed tomography, Radio contrast, Renal system.

Corresponding Author: Dr. Ravindra Patil, Department of Pharmacology, Dr. D. Y. Patil Medical College, Hospital & Research Centre, Kolhapur, Maharashtra, India

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INTRODUCTION

Contrast media (CM) are increasingly used in diagnostic imaging procedures. This result in the rising incidence of iatrogenic renal function impairment caused by the exposure to CM, a condition known as contrast induced nephropathy (CIN).^{1,2} Patients with an acute deterioration in renal function after contrast administration are at risk for chronic kidney disease as well as renal failure requiring dialysis. On the other hand hospitalized patients with acute renal function deterioration are at an increased risk of death. Once other causes of acute kidney injury have been excluded, this deterioration in renal functions usually attributed to contrast material exposure and is termed "contrast-induced nephropathy".^{3,4} Radiographic CM is responsible for 11% of cases of hospital-acquired renal insufficiency, the third most common cause of renal failure.¹ Fortunately, most cases of contrast-induced nephropathy are self-limited, and renal function returns to normal within 2 weeks of contrast administration.^{5, 6} Hipp et al measured the incidence and risk factors for contrast-

induced nephropathy (CIN) in trauma patients. They conducted a retrospective review of a prospectively collected trauma database. Authors found no significant ($P > 0.05$) difference in base-deficit, lactate, and Injury Severity Score between CIN and non-CIN patients.⁷ Hence; under the light of above mentioned evidence; we planned the present study to assess the effect of contrast agents on renal functions based on serum creatinine and creatinine clearance in patients with hypertension who are well controlled on medications.

MATERIALS & METHODS

We planned the present study in the department of radio-diagnosis of medical institute and included assessment of effect of contrast agents on renal functions based on serum creatinine and creatinine clearance in patients with hypertension who are well controlled on medications. We included a total of 60 patients and divided them broadly into two groups; Group 1 and group 2. Ethical approval was taken from institutional ethical committee and written consent was obtained after explaining in detail the

entire research protocol. Group 1: All patients without a pre-existing renal disease non diabetic non hypertensive. Group 2: Known Hypertensive patients on treatment but not known diabetic.

Exclusion criteria:

- Patient not willing to give consent.
- Patients with pre-existing renal disease if creatinine more than 1.6 mg%.
- Patients allergic to contrast media
- Patients with history of any other systemic illness
- Pregnant patients
- Any other disease or drug treatment affecting renal function.

We noticed all the demographic details of the patients included in the present study. We carried out the blood serum investigations for observing the contrast induced nephropathy and before the procedure blood urea levels and serum creatinine levels was done. Patient was injected with low osmolarnonionic contrast media intravenously in the dose of 1.5 ml/kg body weight. After the 48-72 hrs of procedure repeat creatinine and creatinine clearance was measured. Depending upon the predefined criteria, patients were defined for diagnosis of CIN either 25% increase in serum creatinine or 0.5 mg% increases in absolute value. Incidence rate of CIN was calculated for each group. Risk difference was calculated after comparing the group 2 incidences with control (group 1). All rates were expressed as proportions. Relative risk was calculated based on exposed as patients with CIN and non-exposed as without CIN.

STATISTICAL ANALYSIS

All the results were analyzed by SPSS software. Chi-square test was used for assessment of level of significance. P- value of less than 0.05 was taken as significant. P- value of less than 0.05 was taken as significant.

RESULTS

A total of 60 patients were included in the present study. The total incidence of CIN in the group 1 was 13.3% and 26.7 in group 2. We observed non- significant results while comparing the serum creatinine values in between group 1 and group 2.

Table 1: CIN distribution in the study groups

	Group 1 (No comorbidity)	Group 2 (with hypertension)
CIN (Number)	4	8
Proportion	13.3%	26.7%

Table 2: Comparison of mean plasma creatinine post-operative values in between various groups

Group comparison	Mean plasma creatinine post-operative values	p-value
Group 1	0.91	0.22
Group 2	1.03	

Table 3: Comparison of mean creatinine clearance post-operative values in between various groups

Group comparison	Mean creatinine clearance post-operative values	p-value
Group 1	94.86	0.84
Group 2	81.36	

DISCUSSION

Contrast-induced nephropathy (CIN) is defined as the impairment of renal function and is measured as either a 25% increase in serum creatinine (SCr) from baseline or 0.5 mg/dL (44 μmol/L) increase in absolute value, within 48-72 hours of intravenous contrast administration.⁸ In one study of the effect of CIN on long-term mortality after percutaneous coronary intervention in patients with or without CKD, CIN was found to be significantly correlated with long-term mortality in the entire cohort (hazard ratio [HR] 2.26, 95% confidence interval [CI] 1.62 to 2.29, P < 0.0001) and in patients with CKD (HR 2.62, 95% CI 1.91 to 3.57, P < 0.0001) but not in patients without CKD (HR 1.23, 95% CI 0.47 to 2.62, P = 0.6). The rate of CIN in patients with CKD was 11% and 2% in patients without CKD.⁹ Gadolinium-based CM (used for magnetic resonance imaging [MRI]), when compared with iodine-based CM, have a similar, if not worse, adverse effect profile in patients with moderate CKD and eGFR of less than 30 mL/min. Their use has been implicated in the development of nephrogenic systemic fibrosis, a chronic debilitating condition with no cure.¹⁰ A review of 3 series and 4 case reports suggested that the risk of renal insufficiency with gadolinium is similar to that of iodinated radiocontrast dye. The reported incidence varies from 4% in stage 3 CKD to 20% in stage 4 CKD. It may even be worse, as suggested by some investigators. A prospective study of 57 patients found that acute renal failure was seen in 28% of patients in the gadolinium group, compared with 6.5% of patients in the iodine group, despite prophylactic saline and N-acetylcysteine (NAC). The risk factor profile is similar to that for iodinated CM; increased incidence of acute renal failure is seen in older patients and in those with lower baseline creatinine clearance, diabetic nephropathy, anemia, and hypoalbuminemia¹¹. Hipp et al measured the incidence and risk factors for contrast-induced nephropathy (CIN) in trauma patients. They conducted a retrospective review of a prospectively collected trauma database. We studied injured patients who received a contrast-enhanced computer tomography with an initial and repeat serum creatinine after 48 h. Exclusion criteria were patients on dialysis. CIN was defined as a 25% rise in creatinine or an increase in creatinine > or =0.5 mg/dl from baseline 48 h after contrast. In total, 235 patients were studied with an average age of 44+/-20 (13-92 years) (80% men), 79% of whom had blunt injuries. CIN incidence was 5.1% [95% confidence interval (CI), 2.9-8.8%]. No patients in the CIN or non-CIN groups died, or required in-patient/chronic dialysis. CIN patients were significantly (P=0.003) older (61 vs. 43 years). For age > or =75 years, the relative risk was 7.7 and the number

needed to harm was 5. An elevated creatinine (more than 1.5 mg/dl) was significantly ($P=0.007$) associated with CIN. For creatinine greater than 1.5 mg/dl, the relative risk was 6.4 and the number needed to harm was 6. CIN was significantly ($P=0.02$) more likely in patients with glomerular filtration rate less than 60 ml/min/1.73 m. Authors found no significant ($P>0.05$) difference in base-deficit, lactate, and Injury Severity Score between CIN and non-CIN patients.¹² **Rashid et al** conducted a study with an the aim to determine the incidence of CIN in an intensive care unit (ICU) setting and describe the prevalence of associated risk factors. Authors performed a retrospective analysis by review of electronic laboratory database and manual chart review of all patients in two tertiary intensive care units in Newcastle, New South Wales who underwent CT with intravenous contrast during their ICU stay in 2006. CIN was defined as an absolute increment in serum creatinine of 44.2 micromol/l or a relative increment of 25% from baseline at 48 to 72 hours following intravenous contrast. Patients' demographic, biochemical and contrast media data, physiological parameters, fluid and drug administrations and previously described as well as ICU specific risk factors were analysed. Authors compared CIN positive and CIN negative patients to identify risk factors associated with CIN. In total, 2043 patients were admitted to ICU during 2006 and 509 CT studies were performed. One hundred and forty-one of these included administration of intravenous contrast and 139 charts were reviewed. Sixteen out of 139 patients developed CIN (11.5%). More than 70% of patients had two or more risk factors. Age was the only risk factor found to be significantly associated with the development of CIN in a multivariate analysis (P value 0.04, OR 1.041, 95% confidence interval 1.002 to 1.081). Mortality was higher in CIN positive patients (31 vs 13%, P value 0.068). ICU and hospital length of stay was not significantly different in CIN positive and negative patients and persisting renal impairment was not found in CIN positive survivors.¹³

Nough et al studied incidence and main determinants of contrast-induced nephropathy following coronary angiography or subsequent balloon angioplasty. CIN following coronary angiography or angioplasty appeared in 12.8% of the cases. A myocardial infarction before the procedure (OR = 2.121, p = 0.036) and a prior history of hypertension (OR = 2.789, p = 0.025) predicted the appearance of acute renal failure following angiography or subsequent angioplasty. A low estimated glomerular filtration rate at baseline slightly predicted CIN after these interventions.¹⁴

CONCLUSION

From the above results, the authors concluded that hypertension is associated with significantly increased risk of contrast nephropathy. The contrast medium utilization also leads to transient increase in plasma creatinine of all patients. However; future studies are recommended.

REFERENCES

1. Parfrey PS, Griffiths SM, Barrett BJ, et al. Contrast material-induced renal failure in patients with diabetes mellitus, renal insufficiency, or both. *N Engl J Med* 1989; 320:143–15.
2. Gleeson TG, Bulugahapitiya S. Contrast-Induced Nephropathy. *Am J Roentgenol*. 2004;183(6):1673-1689.
3. Katzberg RW. Urography into the 21st century: new contrast media, renal handling, imaging characteristics, and nephrotoxicity. *Radiology* 1997; 204:297–312.
4. Kolonko A, Wiecek A. Contrast-associated nephropathy: old clinical problem and new therapeutic perspectives. *Nephrol Dial Transplant* 1998; 13:803–806.
5. Juncos R, Garvin JL. Superoxide enhances Na-K-2Cl cotransporter activity in the thick ascending limb. *American Journal of Physiology Renal Physiology*. 2005;288(5):982–987.
6. Barbieri L, Verdoia M, Schaffer A, et al. Pre-diabetes and the risk of contrast induced nephropathy in patients undergoing coronary angiography or percutaneous intervention. *Diabetes Res Clin Pract*. 2014;106(3):458-464.
7. McCullough PA, Wolyn R, Rocher LL, Levin RN, O'Neill WW. Acute renal failure after coronary intervention: incidence, risk factors, and relationship to mortality. *Am J Med*. 1997;103(5):368-375.
8. Heyman SN, Rosenberger C, Rosen S, Khamaisi M. Why is diabetes mellitus a risk factor for contrast-induced nephropathy? *Biomed Res Int*. 2013;2013:123589.
9. Nough H, Eghbal F, Soltani M, et al. Incidence and Main Determinants of Contrast-Induced Nephropathy following Coronary Angiography or Subsequent Balloon Angioplasty. *Cardiorenal Med*. 2013;3(2):128-135.
10. Rahman MM, Haque HS, Banerjee SK, et al. Contrast induced nephropathy in diabetic and non-diabetic patients during coronary angiogram and angioplasty. *Mymensingh Med J*. 2010;19(3):372-376.
11. Hipp A, Desai S, Lopez C, Sinert R. The incidence of contrast-induced nephropathy in trauma patients. *Eur J Emerg Med*. 2008;15(3):134-139.
12. Wong PCY, Li Z, Guo J, Zhang A. Pathophysiology of contrast-induced nephropathy. *Int J Cardiol*. 2012;158(2):186-192.
13. Nough H, Eghbal F, Soltani M, et al. Incidence and Main Determinants of Contrast-Induced Nephropathy following Coronary Angiography or Subsequent Balloon Angioplasty. *Cardiorenal Med*. 2013;3(2):128-135.

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