Contrast-induced Nephropathy in Kidney Transplant Recipients: A Single-center Experience

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Keywords. contrast media, nephrotoxicity, risk factors, contrast nephropathy, contrast agent, renal transplant

Introduction. Data regarding contrast-induced nephropathy (CIN) in kidney transplant (KT) recipients are scarce despite the distinct risk factors such as the use of immunosuppressive agents, sympathetic denervation, glomerular hyperfiltration, and high prevalence of the cardiovascular disease. This study aimed to determine the prevalence of CIN in KT recipients who received low-osmolality iodine-based contrast material (CM) for radiological assessment.
Methods. Between 2010 and 2020, 79 of the 3180 KT recipients followed at Hamed Al-Essa organ transplant center received low-osmolality iodine-based contrast for radiological assessment for various indications. Preventive measures including holding metformin, intravenous hydration, sodium bicarbonate and N-acetylcysteine were given before contrast administration. CIN was defined as an increase in serum creatinine of 25% from the baseline within 72 hours.
Results. The enrolled patients were divided into two groups: those who developed CIN (n = 7) and those with no increase in serum creatinine level (n = 72). The mean age of the patients was 52.1 ± 12.3 years; 44 of them were males, and the cause of end-stage kidney disease was mostly diabetic nephropathy. The pre-transplant demographics were comparable between the two groups. Forty-seven cases received contrast for coronary angiography, and 32 received it for a CT scan. The graft function deteriorated in group 1, but no significant difference was found between the two groups at the end of the study.
Conclusion. CIN is not uncommon in KT recipients receiving CM, especially with ischemic heart disease. Risk stratification, optimizing hemodynamics, and avoiding potential nephrotoxins are essential before performing CM-enhanced studies in KT recipients.

IJKD 2023;17:47-53
www.ijkd.org
DOI: 10.52547/ijkd.7165

Original Paper

INTRODUCTION

Contrast-induced nephropathy (CIN) has been recognized for more than 70 years. There are ongoing efforts to chemically modify iodine contrast media (CM) to reduce their nephrotoxicity.[1] Use of computed tomography (CT) scanning and coronary angiography have increased by about 800% and 400%, respectively.[2,3] Furthermore, the risk factors

for CIN, such as chronic kidney disease (CKD), diabetes mellitus, and congestive heart failure, have also increased. More than 27 million people are currently estimated to have chronic kidney disease in the USA, and 200 million people are diabetics.[4,5] The combined increase in CM administration and greater prevalence of at-risk patients will likely result in a continuing increase in CIN events. Contrast induced nephropathy occurring in native kidneys is associated with a significant increase in mortality and morbidity.[6] In a study including 1,826 patients, who underwent coronary artery intervention procedures, CIN was reported in 14% of patients and 1% required dialysis. Mortality was 1% in patients without CIN vs. 7% in cases with CIN and increased to 36% in the dialysis-treated CIN group.[7] Moreover, other studies also support the link between CIN and increased in-hospital and long-term mortalities.[8–10]

Most of the published papers studied CIN in native kidneys, but data on CIN in renal allografts is relatively scarce.[11–15] Moreover, it is probable that kidney transplant recipients are at a significantly increased risk for developing CIN due to kidney transplant-specific factors such as the use of immunosuppressive agents such as calcineurin inhibitors (CNIs), sympathetic denervation, glomerular hyperfiltration, and the higher risk of cardiovascular disease. Therefore, a better understanding of the incidence and predictors of CIN in renal transplant recipients is warranted.[16] Nephrotoxicity caused by CNIs is multifactorial and includes vasoconstriction and hyalinosis of the afferent arterioles, isometric vacuolization of tubular epithelial cells, interstitial fibrosis, and thrombotic microangiopathy, all of which might predispose to CIN.[17]

CIN is caused by the interaction of a number of factors, the most important of which are increased vasoconstriction and diminished arteriolar vasodilatation. This combination ultimately results in renal medullary hypoxia and acute tubular necrosis (ATN). In addition, the administration of contrast media highlights the production of reactive oxygen species (ROS), which itself causes direct damage to the tubular epithelial cells and scavenges nitric oxide (NO), leading to further arteriolar vasoconstriction.[18,19]

Another issue is the significant sodium and water retention in the proximal convoluted tubules of denervated renal allograft. As a result, transplant recipients may be prone to hemodynamically-mediated acute allograft dysfunction, secondary to a decrease in effective arterial circulating volume. Intrarenal hemodynamics of the kidney allografts has been assessed in a study which compared the resistive index (RI), measured by Doppler method, in the donors' kidneys with their subsequent RI in allograft recipients. Resistive indices improved after transplant, which reflected an increase in renal blood flow to maintain GFR.[20]

Ardalan and Tarzamni explained their findings by sympathetic denervation in kidney allografts. Denervated kidney grafts may have a different CIN vulnerability profile compared to native kidneys due to the effect of the sympathetic nervous system on systemic and renal hemodynamics.

Renal allografts undergo hyperfiltration, hemodynamic stress and develop maladaptive structural changes. Therefore, renal allografts often have significantly lower renal reserve despite maintaining a near-normal GFR by utilizing all available compensatory mechanisms. This makes them more prone to renal insults, including CIN. In addition to the impact of cardiovascular diseases, which frequently necessitate coronary and peripheral angiography, kidney transplant recipients also have an increased risk of infection and malignancy due to immunosuppression, which may need CM-enhanced studies.[11-15]

The goal of this study was to evaluate the prevalence of contrast-induced nephropathy and possible risk factors in renal transplant recipients who received low-osmolality iodine-based contrast material for radiological assessment.

MATERIALS AND METHODS

Out of 3180 renal transplant recipients followed at the Hamed Al-Essa Organ Transplant Center, 79 patients who received low-osmolality iodine-based contrast for radiological assessment with various indications were enrolled in this retrospective study, between 2010 and 2020. According to our protocol, all patients received the following preventive measures before contrast administration: A) For elective patients, oral hydration was started 2 days before the procedure and intravenous (IV) fluids were started 6 hours before contrast administration, including carbonated fluids (150 mL of 8.4% sodium bicarbonate diluted in 350 mL of 5% Dextrose or

half normal saline, to be infused at a rate of 3 mL/kg for one hour pre-contrast, then 1 mL/kg/h for the next 6 hours) and oral N-acetylcysteine (ACC) with a dose of 600 mg twice per day, 2 days before and 2 days after the contrast, and holding metformin in diabetic patients; B) In emergency situations, only IV hydration with carbonated fluids and ACC were given. For the angiographic studies (renal, pulmonary, coronary, or aortic), the contrast agent was visipaque TM 270 with a dose of 100 mL, if the patient's weight was less than 75 kg and 120 mL in patients weighing more than 75 kg. Before scanning, we used a 20- gauge cannula needle inserted in a peripheral vein with an injection rate of 4 mL per second. Contrast induced nephropathy was defined as an increase in serum creatinine levels by 0.3 mg/dL, or 25% from baseline values within one week of contrast exposure. We assessed serum creatinine after 72 hours of contrast exposure.

The data of the patients who participated in our study were collected after reviewing our institution's electronic medical records, including those of radiology and cardiac catheterization units. These data included clinical features, laboratory results, and associated comorbid conditions. Moreover, clinical information about the transplant and immunosuppression was extracted from the transplant master database.

Demographics, comorbid conditions (hypertension, diabetes, ischemic heart disease), type and volume of contrast agent, IV fluids, N-acetylcysteine, and the use of calcineurin inhibitors, angiotensin-converting enzyme inhibitors (ACEI), angiotensin receptor blockers and/or diuretics were all evaluated as potential risk factors for CIN.

Our immunosuppression protocol consists of five doses of anti-thymocyte globulin for high-risk patients or two doses of an IL-2 receptor blocker for low-risk patients for induction therapy. Maintenance therapy consists of prednisolone, mycophenolate mofetil, and a calcineurin inhibitor (CNI). The dose of CNI is gradually decreased to the lowest dose by the end of the first year, guided by a 12-hour trough level. We keep the cyclosporine A level between 200 and 250 ng/mL during the first month, at 150 to 200 ng/mL until the third month, at 125 to 150 ng/mL for the next two months, and at 75 to 125 ng/mL until the end of the first year. Similarly, we keep tacrolimus trough levels between 8 to 10 ng/mL during the first three months, and between 5 to 8 ng/mL afterwards. After three months of transplantation, maintenance immunosuppression with a sirolimus-based regimen is used for rejection-free patients with low immunological risk.

Inclusion / Exclusion Criteria

All kidney transplant recipients with a functioning renal allograft, who underwent interventional studies during their hospitalization at the Hammed Al-Essa organ transplant center between 2010 and 2020 and were exposed to contrast media for vascular radiological assessment (with cardiac indications or for assessment of peripheral arterial disease), computed tomography scans with IV contrast (chest, abdomen, or brain), or other indications, were included in this study. We excluded patients who had allergies to contrast agents or those who refused to receive them.

Statistical Analysis

Statistical analyses were performed by using SPSS software version 20 (SPSS, Chicago, IL, USA). Qualitative data were presented as numbers and percentages, and quantitative data were presented as means and standard deviation. The student's t-test was used to compare the means and standard deviations of the studied groups. We compared categorical variables using the chi-squared test. *P* value was considered significant if it was less than .05.

RESULTS

Out of 3180 renal transplants, which are followed up in the Hamed Al-Essa organ transplant center, 116 patients received low-osmolality iodine-based contrast before radiological assessment for different indications between 2010 and 2020. A total of 79 patients were enrolled in this retrospective study and were divided into two groups: group 1 (n = 7) with contrast-induced nephropathy (8.8%), and group 2 (n = 72) representing the control group.

The mean age of patients was 56.8 ± 10.8 years, with no significant difference between the two groups. Forty-four of them were males, 59 were Kuwaitis, and most of them developed end-stage kidney disease as a result of diabetic nephropathy or hypertension (33 and 15, respectively).

Hemodialysis was the most widely used type of renal replacement therapy (79.7%) before transplantation. Sixty-three patients received an allograft from a living donor, out of whom 58 (73.4%) had delayed graft function. Many patients (46.8%) received thymoglobulin for induction therapy, and 50.6% were maintained on a tacrolimus-based regimen. Table 1 showed other pre-transplant demographics (hypertension, diabetes, ischemic heart disease, treated tuberculosis), virology status including hepatitis C virus (HCV) and cytomegalovirus (CMV), and HLA typing. The two groups were comparable in their demographics ($P > .05$) (Table 1).

Six of the 7 patients in group 1 had ischemic heart disease (IHD) but were hemodynamically stable (Table 2), which a significantly higher percentage compared to group 2 (40 out of 72) ($P < .05$). However, the patients who underwent coronary angiography and stenting were comparable in both groups (3 out of 7, 42.8 % vs. 40 out of 72, 55.5%). The majority of patients in both groups were diabetics without any significant difference ($P > .05$) (Table 2). BK viremia was reported in 11% of patients, while nephropathy was documented in 1.2% of cases. Forty-seven patients received

Table 1. Demographics of the 2 Groups

Variables	Total cases (n = 79)	CIN Group (n = 7)	Non CIN (n = 72)	*P*
Age, y (Mean ± SD)	56.8 ± 10.8	51.57 ± 12.4	52.1 ± 12.3	> .05
Sex (Male / Female)	44 / 35	4 / 3	40 / 32	> .05
Nationality (Kuwaiti / Non-Kuwaiti)	59 / 20	4 / 3	55 / 17	> .05
Original Kidney Disease				
Diabetic Nephropathy	33	4	29	
Glomerulonephritis	15	1	14	> .05
Hypertension	4	0	4	
Others	27	2	25	
Dialysis Modality				
Hemodialysis	63	5	58	
Peritoneal Dialysis	5	0	5	> .05
Preemptive	11	2	9	
Donor Type (Live / Cadaveric)	63 / 16	5 / 2	58 / 14	> .05
Graft Function				
Immediate	16	1	15	
Slow	58	6	52	> .05
Delayed	5	0	5	
Induction				
Basiliximab	27	5	22	
Thymoglobulin	37	1	36	> .05
Others	15	1	14	
Immunosuppressant at Time of Contrast				
Tacrolimus	40	4	36	
Neoral	27	3	24	> .05
Others	12	0	12	
HCV (Positive)	3	0	3	> .05
CMV (IgG Positive)	73	6	67	> .05
Pre-transplant Hypertension	72	5	67	> .05
Pre-transplant Diabetes	44	4	40	> .05
Pre-transplant Ischemic Heart Disease	31	4	27	> .05
Treated Tuberculosis Pre-transplant	39	4	35	> .05
Graft Outcome				
Functioning	68	7	61	> .05
Failed	11	0	11	
Patient survival				
Living	75	7	68	> .05
Dead	4	0	4	

Table 2. Shows Post-transplant Complications and Risk Factors for Contrast Induced Nephropathy

Variables	Total Cases (n = 79)	CIN Group (n = 7)	Non CIN (n = 72)	*P*
Post-transplant Ischemic Heart Disease	46	6 (85)	40 (55.5)	< .05
Post-transplant Diabetes	63	7	57	> .05
BK Viremia	9 (11.3%)	1	8	> .05
BK Nephropathy	1 (1.2%)	0	1	> .05
Contrast Indication				
Coronary Angiography	47	3	44	
Coronary Angiography and Stenting	43	3	40	> .05
CT with Contrast	32	4	28	
ACEI Usage	34 (43%)	2	32	> .05
Diuretic Usage	35 (44%)	2	33	> .05

contrast for coronary angiography, while 32 patients received contrast for CT studies (chest, abdomen, brain). About 44 % of patients received ACEI and or diuretics without a significant difference between the two groups ($P > .05$).

As represented by mean serum creatinine, renal function was comparable between the two groups at different time intervals ($P > .05$). Despite the rise in serum creatinine in group 1 at one week and one month post-contrast, the rise was not statistically significant. ($P > .05$). However, when compared to baseline values, the percentage of the post-contrast increase in serum creatinine was significantly higher in group 1 ($P < .001$) (Table 3).

DISCUSSION

Contrast-induced nephropathy is defined as development of an impairment in kidney function, as either a 25% increase in serum creatinine from the basal value or an increase in the absolute serum creatinine value by 0.5 mg/dL (44 µmol/L) within 48 to 72 hours after intravenous contrast administration.[21]

To attribute renal insufficiency to contrast administration, it must be acute, occur within seven days (usually 2 to 3 days) of contrast use and after excluding all other identifiable causes of kidney dysfunction.

Contrast-induced nephropathy has been extensively studied in native kidneys, but only a few small retrospective studies have addressed CIN in renal allografts.[11-15] In a retrospective study of 35 kidney transplant recipients who were treated with cyclosporine, the incidence of CIN, defined as a creatinine rise > 25%, was 21%.[12] This incidence is higher than that reported in native

Table 3. Renal Function and Laboratory Parameters Post-transplant

Variables	Total Cases (Mean ± SD)	CIN (Mean ± SD)	Non CIN (Mean ± SD)	*P*
Age, y (Mean ± SD)	56.8 ± 10.8	51.57 ± 12.4	52.1 ± 12.3	
Graft Function as Estimated by eGFR (by MDRD Formula)				
Pre-contrast	71.5 ± 31.9	64 ± 27	72.3 ± 32.4	.51
After 1 week	70 ± 32.2	47.2 ± 15.5	72.2 ± 32.6	.05
After 1 month	71.8 ± 31.8	57.5 ± 18.6	73.2 ± 32.4	.21
At Last Follow-up (6 months)	62.8 ± 35.2	52.4 ± 27.7	63.8 ± 35.9	.41
Serum creatinine (Mean ± SD)				
Pre-contrast	133.7 ± 72.7	141.1 ± 99.7	133 ± 70.5	
After 1 week	137.1 ± 71.3	174.8 ± 88.1	133.3 ± 69.1	> .05
After 1 month	136.2 ± 84.9	151.7 ± 101.7	134.7 ± 83.3	
At last follow up (6 months)	124.2 ± 61	139.1 ± 77.2	121.5 ± 59.6	
Creatinine Rise Compared to Basal (%)	4.6 ± 18.1	44.6 ± 19.9	0.55 ± 13.3	< .001
Pre-contrast S Albumin	32.8 ± 4.5	31.8 ± 4.5	33 ± 4.8	> .05
Pre-contrast Hemoglobin	116.1 ± 17.4	105.5 ± 8.4	117.1 ± 17.8	> .05
Pre-contrast Weight	79.37 ± 17.9	83 ± 21.6	79 ± 17.65	> .05
Pre-contrast Height	162 ± 9.8	161.6 ± 11.2	162 ± 9.8	> .05
Pre-contrast BMI (Mean ± SD)	30.6 ± 7.1	31.4 ± 10	30.6 ± 6.9	> .05

kidneys (13%); however, six out of seven cases who developed CIN had received half normal saline or no IV fluids for CIN prophylaxis rather than the standard prophylactic care of isotonic fluid administration.[22]

In another study that included 57 patients undergoing coronary angiography, CIN was reported in 16% of the patients.[11] In univariate analysis, the use of N-acetylcysteine and iso-osmolar contrast media (CM) were found to be protective against CIN. However, logistic regression analysis revealed that only the administration of low-osmolarity CM was associated with CIN compared to iso-osmolar CM (odds ratio of ~7.7), while type of renal allograft, preexisting co-morbidities, and immunosuppressive medications were not associated with CIN. Neither of the two mentioned studies evaluated complex outcomes such as death-censored graft loss or death. More recently, three small studies reported a CIN incidence of 6 to 13%.[13–15] In one study by Bostock *et al.*, CIN was reported in 13% of renal transplant recipients who underwent endovascular repair of an aortic aneurysm, compared to 5% in a non-transplanted group.[14]

In our study, which included kidney transplant recipients who underwent contrast-mediated computed tomography or coronary angiography, the prevalence of CIN was 8.8% (7 out of 79), which was similar to that reported in other recent studies (6-13%) in both native and transplant kidneys.[11,13,14,22] The routine use of hypo-osmolar CM along with proper hydration, intravenous bicarbonate, and N-acetylcysteine in all cases might explain the relatively lower prevalence of CIN in our cohort.

Regarding the risk factors for CIN, a strong correlation has been found between the risk of CIN and pre-existing renal impairment, diabetes mellitus, advanced age, peri-procedural dehydration, congestive heart failure, the volume and type of administered CM, and the concomitant use of other nephrotoxins.[6,7,24]

The two groups of patients in our cohort were comparable in terms of preexisting renal function, the prevalence of diabetes, mean age, mean dose of contrast used for different indications, and the type of maintenance immunosuppression. However, ischemic heart disease was significantly higher in group 1 ($P < .05$). This finding was concordant with the report of Pan *et al.*, who indicated that IHD is a risk factor for CIN and the degree of coronary artery stenosis was significantly higher in the group who developed CIN compared to the those without CIN ($P < .05$).[22]

Anemia can exacerbate hemodynamic stress and ischemia-reperfusion-related acute kidney injury, particularly in renal allografts with already compromised hemodynamic compensatory mechanisms. Considering anemia as a risk factor for CIN, there was no statistically significant difference in hemoglobin level between the two groups, although hemoglobin was relatively higher in group 2 (11.71 ± 1.78 vs. 10.55 ± 0.84 g/dL, $P > .05$), which was inconsistent with previous reports.[24]

We did not find any protective effect with the use of CNIs (either tacrolimus or cyclosporin) and mycophenolate mofetil in CIN ($P > .05$). An observation that contradicted the findings of Abu Jawdeh *et al.*, who demonstrated that CNIs might have some protective effect on the development of CIN.[16]

In our cohort, all patients in both groups received N-acetylcysteine. Abu Jawdeh *et al.* found that the use of N-acetylcysteine was positively correlated with the development of CIN, which contradicted the findings of multiple previous studies that showed a protective effect for N-acetylcysteine in preventing CIN.[16,25,26] However the protective effect of N-acetylcysteine was not replicated in multiple recent trials such as in a prospective randomized controlled trial of 2,308 patients with risk factors for CIN, where N-acetylcysteine failed to exert a protective effect.[22]

We acknowledge that our study has some limitations. First, it is a retrospective study with a small sample size. Second, we did not capture CM-enhanced procedures done at the outpatient department or outside our hospital.

CONCLUSION

CIN is not unusual in KT recipients receiving CM, especially with ischemic heart disease. Risk stratification, hemodynamic optimization, and avoidance of potential nephrotoxins are important before performing a CM-enhanced study in a KT recipient. It is appropriate to conduct prospective, randomized, controlled studies to assess CIN in transplant settings.

CONFLICT OF INTEREST

The authors have no conflict of interest to disclose.

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Received August 2022
Revised October 2022
Accepted December 2022