Acute Kidney Injury in Hospitalized Kidney Transplant Recipients

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ABSTRACT

Background. Acute kidney injury (AKI) is a main cause of morbidity, hospitalization, and hospital readmission in kidney transplant recipients. We aimed to determine AKI incidence and risk factors following kidney transplant to assess outcomes such as renal function and graft loss after AKI.

Methods. We conducted a retrospective cohort study with hospitalized kidney transplant recipients during 2016 to 2017. Clinical data of 179 patients were reviewed. The primary outcome was AKI incidence and risk factors. To determine AKI occurrence, we based it on creatinine criteria from Acute Kidney Injury Network classification.

Results. We documented a total of 179 hospital admissions; AKI was diagnosed in 104 patients (58.1%). Recipients with higher baseline serum creatinine (odds ratio, 2.6; confidence interval [CI], 1.5-4.5; $P < .001$) and hospital admission because of infections (odds ratio, 2.4; CI, 1.1-5.2; $P = .020$) were more likely to experience AKI. A total of 19 recipients (10.6%) had graft loss with a significant AKI association ($P = .003$) at 12 months after admission. Intensive care unit length of stay ($P = .63$) and hospital stay ($P = .55$) were not different in patients with AKI compared with the control group.

Conclusions. As a main clinical finding, we concluded that infections and higher serum creatinine baseline level were associated with the development of AKI.

KIDNEY transplant is the best therapeutic option available as end-stage kidney disease treatment [1,2]. Acute kidney injury (AKI) is defined as an abrupt loss in renal function, and it has a wide variety of clinical etiologies [3]. Thus, the renal graft is predisposed to several acute insults related to ischemia-reperfusion injury, surgical complications, immunologic injury, and medication toxicity [4]. Afterward, AKI increases immune dysfunction risk, water overload, and adverse drug effects [5]. Likely, kidney transplant recipients have a "low renal reserve" according to several bibliographies, and it is a consequence of the reduced number of functioning glomeruli and the use of calcineurin inhibitors making them more susceptible to any renal injury [6,7]. Although there are no specific recommendations for the diagnosis of AKI in kidney transplant, it is recognized as an essential cause of morbidity and graft loss [8]. Mehrotra et al assessed the relationship between AKI episodes in 3066 kidney transplant recipients admitted to the hospital and their subsequent graft dysfunction. They found AKI was independently associated with increased graft loss from any cause (hazard ratio, 2.74; 95% confidence interval [CI], 2.56-2.92) and death with a functioning transplant (hazard ratio, 2.36; 95% CI, 2.14-2.60) [9].

We performed this study to determine AKI incidence and risk factors in hospitalized transplant recipients and their assessment of renal function and graft loss at 12-month follow-up.

MATERIALS AND METHODS

Definitions

AKI was defined as an increase in serum creatinine higher than 1.5 times the baseline serum creatinine. This baseline usually is

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measured within 7 days prior to hospital admission. Moreover, AKI classification stages were according to the Kidney Disease: Improving Global Outcomes (KDIGO) guideline [10]. All patients have been monitored by a nephrologist monthly at Colombiana de Transplantes. The urine output was not used as a criterion because of most of the patients had no indwelling bladder catheter, hindering reliable analysis of urine volume. Graft loss was defined as a return to dialysis or graft removal within the first year of follow-up. Urinary tract infection (UTI) was defined as a positive urine culture in a patient with urinary symptoms [11]. Acute diarrhea (AD) was defined as the presence of 3 or more loose stools per day (or increased defecation within a normal routine patient) [12]. Pneumonia was defined as an acute infection in the lung parenchyma [13].

Outcomes

Primary outcomes were graft loss, graft function at 12 months, and mortality rate. Secondary outcomes included incidence of AKI and all-cause hospitalizations.

Study Population and Design

We performed a retrospective analysis in kidney transplant recipients older than 18 years and with hospital admission. Our follow-up was from January 1, 2016, to December 31, 2017, at Colombiana de Transplantes (Bogotá, Colombia). The exclusion criteria were pregnancy, being within the first 3 months after kidney transplant, or having received a multiple solid organ transplant. AKI was appraised on hospital admission. Patients who underwent contrast media received nephroprotection to prevent contrast-induced nephropathy. Routinely, dosage adjustments of tacrolimus and cyclosporine were made to maintain target levels at 5 to 7 ng/mL and 200 to 400 ng/mL, respectively [14]. During an AKI episode, calcineurin inhibitors were withdrawn only in those patients under mechanical ventilation or with a Sequential Organ Failure Assessment (SOFA) score higher than 2 points [15]. In fact, these patients were maintained only with steroids in the AKI management [15].

The study protocol adhered to the principles of the Declaration of Helsinki, and an independent research ethics committee has approved it. Demographic, clinical, and laboratory data were collected from the medical records.

Analysis

Frequencies and percentages were used for categorical variables. The distribution of numerical variables was quantified using the Shapiro-Wilk test. Also, numerical variables were reported as medians and interquartile ranges (IQRs) in the descriptive statistics associated with the cohort.

Comparisons of the 2 groups (AKI and no AKI) were performed to determine graft loss and renal function at 12 months using Pearson χ^2 test and the Mann-Whitney nonparametric test as corresponded. Variables with P values less than or equal to .10 were included in the multiple logistic regression models. Logistic regression analysis was applied to model AKI development and mortality. The models were subjected to variable selection, and the variables with P values less than .05 were considered statistically significant.

RESULTS

Study Population

A total of 179 kidney transplant recipients were included in the study period. Although 270 hospital admissions occurred with 179 recipients, only the first admission was considered for AKI diagnosis. Demographic and clinical data are described in Table 1. The median age was 52 years (IQR, 22 years) over an age range from 19 to 83 years. Sex distribution was quite similar (50.8% were male and 49.1% were female). The glomerular disease was the most frequent end-stage kidney disease underlying etiology with an incidence of 17.3% (n = 30); other underlying causes were diabetes (13.1%), congenital (9.1%), and urinary tract obstruction (6.8%). The median body mass index was 22.7 kg/m^2, with an interval from 14.9 to 39.3 kg/m^2 (IQR, 5.5 kg/m^2). Furthermore, the basal serum creatinine levels were reported between 0.65 and 5.78 mg/dL with a median of 1.5 mg/dL (IQR, 1.1 mg/dL) compared with serum creatinine levels between 0.6 and 16.38 mg/dL with a median of 2.2 mg/dL (IQR, 1.9 mg/dL) at hospital admission. Likewise, infection was the most common cause of admission and AKI (Table 1).

Table 1. Demographic and Clinical Data

Variables	All Patients (N = 179)	AKI (n = 104)	No-AKI (n = 75)	P Value
Age, years (IQR)	52 (22)	50 (24)	55 (20)	.028*
Sex (%)				.519
Women	88 (49.1)	49 (47.1)	39 (52)	
Men	91 (50.8)	55 (52.8)	36 (48)	
Cause ESKD (%)				.172
Diabetes	23 (13.1)	10 (9.9)	13 (17.5)	
Glomerular	30 (17.3)	21 (20.7)	9 (12.1)	
Congenital	16 (9.1)	12 (11.8)	4 (5.4)	
Obstruction	12 (6.8)	8 (7.92)	4 (5.4)	
Unknown	90 (51.4)	47 (46.5)	43 (58.1)	
Other	4 (2.2)	3 (2.9)	1 (1.3)	
Baseline serum creatinine, mg/dL (IQR)	1.4 (0.9)	1.5 (1.3)	1.2 (0.6)	.000*
Creatinine at admission, mg/dL (IQR)	2.6 (2)	3.5 (3.0)	1.4 (1.3)	.000*
Transplant time, years (%)				.138
3 months-1 year	36 (20.1)	11 (14.6)	25 (24)	
1-5 years	41 (22.9)	15 (20)	26 (25)	
>5 years	102 (56.9)	49 (65.3)	53 (50.9)	
ICU admission (%)	15 (8.3)	9 (8.6)	6 (8)	.876
ICU stay, days (IQR)	10 (10)	10.8 (10)	9 (8)	.635
Hospital stay, days (IQR)	7.3 (4)	7.6 (5)	6.8 (3)	.559
Infection (%)	121 (67.6)	81 (77.8)	40 (53.3)	.001*
Urinary tract infection	52 (42.9)	39 (48.1)	13 (32.5)	.102
Acute diarrhea	32 (26.4)	24 (29.6)	8 (20)	.259
Pneumonia	12 (6.7)	5 (4.8)	7 (9.3)	.232
Surgical (%)	7 (3.9)	6 (5.7)	1 (1.3)	.131
Cancer (%)	5 (2.7)	2 (1.9)	3 (4)	.405
CVD (%)	13 (7.2)	3 (2.8)	10 (13.3)	.008*

Abbreviations: BMI, body mass index; CVD, cardiovascular disease; DGF, delayed graft function; ESKD, end-stage Kidney Disease; ICU, intensive care unit; IQR, interquartile range.
*Statistically significant.

Table 2. Risk Factors for Acute Kidney Injury: Multivariate Analysis

AKI Risk Factors	Odds Ratio	*P* Value	95% CI	
Baseline creatinine	2.6	< .001	1.5	4.54
Admission because of infection	2.4	.020	1.14	5.21
CVD	0.41	.294	0.09	1.91
Age	0.28	.120	0.06	1.38

Abbreviations: AKI, acute kidney injury; CVD, cardiovascular disease.

Risk Factors for AKI Development

AKI incidence in hospitalized renal recipients who received diagnosis by KDIGO criteria was a total of 58.1% (n = 104) categorized as follows: 52 patients (50%) were classified as KDIGO stage 1, 20 (19.2%) were classified as stage 2, and 32 (30.7%) were classified as stage 3. In a bivariate analysis, we identified the following variables associated with AKI: younger age ($P = .028$; AKI 50 vs no AKI 55 years), higher baseline serum creatinine ($P < .001$; AKI 1.5 vs no AKI 1.2 mg /dL), hospital admission because of infections ($P < .001$; AKI 77% vs no AKI 53%), and hospital admission because of cardiovascular disease (CVD) ($P = .001$, AKI 2.8% vs no AKI 13.3%) (Table 1). As a result, in a multivariate analysis of AKI risk factors, higher baseline serum creatinine (odds ratio, 2.6; CI, 1.5-4.5; $P < .001$) and hospital admission because of infections were significantly associated (odds ratio, 2.4; CI, 1.1-5.2; $P = .020$) (Table 2).

Factors for Severe AKI

From a total of 179 hospital admissions, 32 patients (17.8%) developed AKI 3 during their hospital stay. From the 32 patients with AKI 3, we identified in a bivariate analysis the following variables were significant: admission because of AD (n = 10; 43.4%; $P = .040$) and UTI (n = 5; 2.7%; $P = .022$).

Hospital Admission Causes

Overall, the length of hospital stay was from 1 to 63 days, with a median stay of 4 days (IQR, 5 days). During hospitalization, 15 patients (8.3%) were admitted to the intensive care unit (ICU). Among these patients, 9 (60%) developed AKI. ICU median length of stay was 10 days (IQR, 5 days), with an interval rate from 2 to 25 days.

Infections were the most prevalent cause of hospital admission (n = 121, 67.6%). Its etiology was in the first place UTI (n = 52; 42.9%) followed by AD (n = 32; 26.4%). Most of the urinary cultures isolated *Escherichia coli* (53.8%) as main UTI cause. Other UTI etiologies are shown in Table 3. In patients who had a UTI, we found 34 cases (27.6%) with extended-spectrum β-lactamases (ESBLs) and 15 cases (12.1%) cases with carbapenem-resistant Enterobacteriaceae (CRE). In kidney transplant recipients with AD, *Entamoeba histolytica* (n = 8; 13%) was isolated by microscopic stool examination. However, no other micro-organisms were isolated. Additional admission causes were surgical, cardiovascular, and oncologic conditions with 3.9%, 2.7%, and 7.2%, respectively. Finally, Table 4 summarizes the top 10 common causes of hospital admission.

Table 3. Microorganisms Isolated in Urinary Culture

Microorganism	n = 52	%
Escherichia coli	28	53.8
ESBL	12	
β-lactamases	9	
No resistance	7	
Klebsiella pneumoniae	6	11.5
CRE	5	
No resistance	1	
AmpC group	6	11.5
Candida glabrata	1	1.9
Other	8	15.3
No isolation	3	5.7

Abbreviations: CRE, carbapenem resistant Enterobacteriaceae; ESBL, extended spectrum B lactamases.

Table 4. Most Common Causes of Hospital Admission for Kidney Transplant Recipients

Cause of Hospital Admission	%	n = 179
Urinary tract infection	42.9	52
Acute diarrhea	26.4	32
Pneumonia	6.7	12
Uremia	5.5	10
Myocardial infarction	2.7	5
Acute bronchitis	2.7	5
Graft Rejection	2.2	4
Abdominal pain	2.2	4
Unstable angina	2.2	4
Soft tissue infections	1.6	3
Anemia	1.6	3
Other	28.3	45

Mortality

Seven individuals (3.93%) died during their hospital stay; 4 were diagnosed as having AKI. Mortality was not related to AKI diagnosis ($P = .9$) or severity ($P = .292$). We found in a bivariate analysis the following factors associated with mortality: CVD as admission cause ($P < .001$), ICU admission ($P < .001$), and length of hospital stay ($P = .002$). The independent risk variables for mortality in a multivariate analysis are shown in Table 5.

Assessment of Renal Function at 12 Months

Comparing the groups with and without AKI, patients with AKI had an increased media change in creatinine levels at 12 months compared with individuals without AKI (15.7%

Table 5. Multivariate Analysis of Factors Associated With Mortality

Mortality	Odds Ratio	*P* Value	95% CI	
Length of stay	1.0	.831	0.934	1.08
ICU admission	14.2	.010	1.89	106.6
CVD	9.08	.019	1.42	57.7

Abbreviations: CVD, cardiovascular disease; ICU, intensive care unit.

Table 6. Creatinine Evaluation in Patients With and Without AKI*

Creatinine mg/dL	No-AKI	AKI
Baseline (n = 179)	1.24 (1-1.6)	1.54 (1.2-2.5)
Admission (n = 179)	1.38 (1-1.8)	3.0 (2.0-4.2)
12 months (n = 164)	1.24 (1.0-1.8)	2.15 (1.3-3.6)

Abbreviations: AKI, acute kidney injury; n, number of patients.
*$P < .000$; Median (IQR, 25-75th).

vs 4.1%, $P = .023$). Data from serum creatinine evolution are shown in Table 6.

Graft Loss at 12 Months

Graft loss was found in 19 recipients (10.6%), which was significantly higher in the AKI group ($P = .003$). Of the 19 patients with graft loss, 17 had AKI, and that included 12 who developed AKI stage 3 (37.5%) ($P < .001$) (Table 7).

DISCUSSION

AKI is a common entity in kidney transplant. Renal transplant recipients are more susceptible to AKI than the general population, including more severe forms and AKI requiring dialysis [16]. In this study, we calculated AKI incidence related to hospitalized kidney transplant recipients providing a useful AKI burden.

AKI incidence in our study was 58.1%. Recent publications have shown different AKI incidence from 11% to 82.3% [8,16]. It depends on some measurement conditions, such as hospitalized individuals or outpatient and AKI diagnosis by KDIGO or Risk, Injury, Failure, Loss of Kidney Function, End-Stage Kidney Disease criteria. In comparison with previous studies that recruited hospitalized recipients, Filiponi et al reported 82.3% parallel to other studies with 33.8% (184.621 recipients) and 21% of AKI incidence [16]. Despite, publications that included outpatient clinics in their AKI incidence, calculations were not compared with our findings [17]. Besides, we found a lower AKI risk in older transplant recipients and in patients with cardiovascular disease as a cause of hospital admission, although these findings were not significant. Moreover, younger age was not significant for AKI incidence in our population comparable with other research [8,16]. Nevertheless, Charbonney et al described a significance on younger transplant recipients for AKI in comparison with subjects who did not receive transplants [18]. These findings will need further discussion.

Table 7. Graft Loss and AKI at 12 Months

	AKI		
Graft Loss	No	Yes	Total
No	73 (45.6)	87 (54.3)	160
Yes	2 (10.5)	17 (87.4)	19
Total	75	104	179
$P = .003$			

Abbreviation: AKI, acute kidney injury.

Infection-related hospitalization was an independent risk factor for AKI development. UTI was most common in recipients, but there was not significant evidence for AKI development. Certainly, recent research with 198 UTI admissions including kidney transplant recipients with pyelonephritis did not observe statistical significance for AKI incidence in a 4-year follow-up [11]. Conversely, a prospective clinical cohort described higher AKI risk in transplant recipients with pyelonephritis ($P = .001$) [19]. Of note, the proportion of resistant bacteria generating ESBLs and carbapenem-resistant Enterobacteriaceae was 39.7% in our population, which resembles local findings (ESBLs = 42%) [20].

Some scientific studies have published relevant data reference to AD that increases AKI occurrence, graft loss, and mortality in solid organ transplant recipients [21,22]. Furthermore, a retrospective study in Taiwan showed higher serum creatinine levels in renal transplant recipients with longer diarrhea period (> 14 days) [23]. In fact, AD was significantly associated with AKI risk in our patients. We identified the AD etiologic agent in 13% of the total cases in comparison with 39.1% and 47% reported by other medical centers [21,22].

In this study, patients with higher baseline creatinine (1.5 mg/dL) had increased AKI risk 2.6 times vs individuals with lower baseline creatinine. This finding was coherent with Pinheiro et al and Filiponi et al who found higher baseline creatinine could increase AKI incidence 2.7 times, and it was a predictor for AKI severity adjacent to dialysis requirement [8,24]. Creatinine recovery after AKI was observed in kidney transplant recipients, although there was a baseline creatinine rise compared with patients without AKI. It could be classified as a partial renal recovery after the AKI event [25]. Transplant recipients with AKI diagnosis have decreased their glomerular filtration rate (GFR) with higher media change in creatinine levels at 12 months compared with those individuals without AKI (15.7% vs 4.1%, $P = .023$). Our data are equivalent to other clinical results; a cohort with 659 transplant recipients has issued increasing creatinine media levels and higher GFR in patients with AKI vs the control group [26]. Indeed, lower GFR has been linked to dialysis requirement and graft loss [8,27]. Hence, we detected a significant association between AKI incidence and graft loss risk ($P = .003$) and incidence (10.6%). For instance, these findings were confirmed for recent publications reporting higher graft loss incidence (3.7% and 20.4%) in the AKI group than the non-AKI group [8,16,28].

Finally, 4 patients (2.2%) with AKI died in our cohort, and this was not correlated to AKI. These data are compatible with other mortality rates (2.1% and 4.4%) where AKI was not associated with death. Factors such as ICU admission, length of hospital stay, or CVD medical history were the main risk factors for mortality in our epidemiologic analysis and other research groups [8,16]. Likewise, mortality in organ solid transplant recipients with AKI has been decreasing over the time in a recent hospitalization database [16].

Our study has some limitations. First, it was a single-center, retrospective cohort study, which is susceptible to bias. Secondly, we did not report other risk factors for AKI, such as exposure to contrast media, fluid types, and others. Also, our examination was only in the first AKI episode instead of several AKI events. Regarding the outcomes, we did not measure dialysis requirement and potential renal recovery in our sample.

CONCLUSION

In summary, we demonstrated that AKI is a common complication in hospitalized kidney transplant recipients, it is significantly associated with hospital admission because of infections and high levels of basal serum creatinine, and it causes decreased renal function and graft survival at 12 months. Because infectious diseases are the leading cause of AKI, we should make every effort in the prevention and early detection of these pathologies to avoid the occurrence of AKI.

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REFERENCES

[1] Hart A, Smith JM, Skeans MA, Gustafson SK, Stewart DE, Cherikh WS, et al. OPTN/SRTR 2015 Annual Data Report: Kidney. Am J Transplant 2017;17(Suppl. 1):21–116.

[2] Rosselli D, Rueda JD, Diaz CE. Cost-effectiveness of kidney transplantation compared with chronic dialysis in end-stage renal disease. Saudi J Kidney Dis Transpl 2015;26:733–8.

[3] Fortrie G, De Geus HRH, Betjes MGH. The aftermath of acute kidney injury: a narrative review of long-term mortality and renal function. Crit Care 2019;23:24.

[4] Cooper JE, Wiseman AC. Acute kidney injury in kidney transplantation. Curr Opin Nephrol Hypertens 2013;22:698–703.

[5] Kellum JA, Murugan R. Effects of non-severe acute kidney injury on clinical outcomes in critically ill patients. Crit Care 2016;20:159.

[6] Musso CG. Renal reserve test: its methodology and significance. Saudi J Kidney Dis Transpl 2011;22:990–3.

[7] Husain-Syed F, Ferrari F, Sharma A, Danesi TH, Bezerra P, Lopez-Giacoman S, et al. Preoperative renal functional reserve predicts risk of acute kidney injury after cardiac operation. Ann Thorac Surg 2018;105:1094–101.

[8] Filiponi TC, Requião-Moura LR, Tonato EJ, Carvalho de Matos AC, E Silva-Filho AP, de Souza Durão Junior M. Hospital admission following acute kidney injury in kidney transplant recipients is associated with a negative impact on graft function after 1-year. PLoS One 2015;10:1–12.

[9] Mehrotra A, Rose C, Pannu N, Gill J, Tonelli M, Gill JS. Incidence and consequences of acute kidney injury in kidney transplant recipients. Am J Kidney Dis 2012;59:558–65.

[10] KDIGO. KDIGO clinical practice guideline for acute kidney injury. Kidney Int Suppl 2012;2.

[11] Al Midani A, Elands S, Collier S, Harber M, Shendi AM. Impact of urinary tract infections in kidney transplant recipients: a 4-year single-center experience. Transplant Proc 2018;50:3351–5.

[12] Maes B, Hadaya K, De Moor B, Cambier P, Peeters P, De Meester J, et al. Severe diarrhea in renal transplant patients: results of the DIDACT study. Am J Transplant 2006;6:1466–72.

[13] Dizdar OS, Ersoy A, Akalin H. Pneumonia after kidney transplant: incidence, risk factors, and mortality. Exp Clin Transplant 2014;12:205–11.

[14] KDIGO group. KDIGO clinical practice gudeline for the care of kidney transplant recipients. Am J Transplant 2008;8:1084.

[15] De Carvalho MA, Freitas FGR, Silva HT, Bafi AT, Machado FR, Pestana JOM. Mortality predictors in renal transplant recipients with severe sepsis and septic shock. PLoS One 2014;9:e111610.

[16] Garg N, Kumar N, Singh T, Parajuli S, Astor BC, Mandelbrot D, et al. Hospitalization trends for acute kidney injury in kidney transplant recipients in the United States, 2004-2014. Transplantation 2019;103:2405–12.

[17] Nakamura M, Seki G, Iwadoh K, Nakajima I, Fuchinoue S, Fujita T, et al. Acute kidney injury as defined by the RIFLE criteria is a risk factor for kidney transplant graft failure. Clin Transplant 2012;26:520–8.

[18] Charbonney E, Saudan P, Triverio PA, Quinn K, Mentha G, Martin PY. Prognosis of acute kidney injury requiring renal replacement therapy in solid organ transplanted patients. Transpl Int 2009;22:1058–63.

[19] Rice JC, Peng T, Kuo YF, Pendyala S, Simmons L, Boughton J, et al. Renal allograft injury is associated with urinary tract infection caused by Escherichia coli bearing adherence factors. Am J Transplant 2006;6:2375–83.

[20] Giraldo-Ramírez S, Díaz-Portilla OE, Miranda-Arboleda AF, Henao-Sierra J, Echeverri-Toro LM, Jaimes F. Urinary tract infection leading to hospital admission during the first year after kidney transplantation: a retrospective cohort study. Transplant Rep 2016;1:18–22.

[21] Echenique IA, Penugonda S, Stosor V, Ison MG, Angarone MP. Diagnostic yields in solid organ transplant recipients admitted with diarrhea. Clin Infect Dis 2015;60:729–37.

[22] Devresse A, Morin L, Aulagnon F, Taupin JL, Scemla A, Lanternier F, et al. Baseline graft status is a critical predictor of kidney graft failure after diarrhoea. Nephrol Dial Transplant 2019:1–8.

[23] Wang HY, Yeh MK, Tian YF, Huang YB. Effect of prolonged diarrhea in renal transplant recipients at a single center in Taiwan. Transplant Proc 2016;48:870–3.

[24] Konigsfeld HP, Viana TG, Pereira SC, Oliveira T, Dos C, Kirsztajn GM, et al. Acute kidney injury in hospitalized patients who underwent percutaneous kidney biopsy for histological diagnosis of their renal disease. BMC Nephrol 2019;20:315.

[25] Doyle JF, Forni LG. Acute kidney injury: short-term and long-term effects. Crit Care 2016;20:1–7.

[26] van Dijk TY, Rahamimov R, Chagnac A, van Dijk DJ, Mor E, Shlomai A, et al. The effect of cause, timing, kidney function recovery, and recurrent events on the prognosis of acute kidney injury in kidney transplant recipients. Clin Transplant 2018;32:e13398.

[27] Wu J, Li H, Huang H, Wang R, Wang Y, He Q, et al. Slope of changes in renal function in the first year post-transplantation and one-yr estimated glomerular filtration rate together predict long-term renal allograft survival. Clin Transplant 2010;24:862–8.

[28] Nagarajan M, Ramanathan S, Dhanapriya J, Dineshkumar T, Bala Subramaniyan T, Gopalakrishnan N. Impact of acute kidney injury on renal allograft survival. Ren Fail 2017;39:40–4.