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Short-term prognosis and influencing factors of patients with acute kidney injury treated with prolonged intermittent renal replacement therapy

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Abstract

Background: Studies assessing prognosis after prolonged intermittent renal replacement therapy (PIRRT) for acute kidney injury (AKI) are scarce.

Aim: To assess the impact of PIRRT on AKI and factors associated with short-term prognosis.

Methods: In this retrospective nested case-control study, AKI patients administered PIRRT in Shanghai General Hospital from 01/2012 to 10/2018 were assigned to the 30-day survivor and death groups. Surviving patients were further divided into the kidney recovery and non-recovery groups at 30 and 90 days post-discharge, respectively. Propensity score matching was performed.

Results: Totally 576 patients were included in the non-matched study population, mortality and kidney recovery rates were 51.7% and 33.4%, respectively. After propensity score matching, there were 250 patients in each of the death and survival groups. Low PIRRT frequency (OR = 2.165, 95% CI = 1.178-3.978), infection (OR = 0.447, 95% CI = 0.251-0.795), number of damaged vital organs (OR = 0.478, 95% CI = 0.346-0.661), sodium (OR = 0.958, 95% CI = 0.928-0.988), total protein (OR = 1.047, 95% CI = 1.022-1.072), pre-dialysis thrombin time (TT; OR = 0.959, 95% CI = 0.936-0.983), pre-discharge glomerular filtration rate (GFR; OR = 1.024, 95% CI = 1.017-1.031) and admission ward [reference: renal ward; intensive care unit (OR = 0.042, 95% CI = 0.008-0.211); surgery (OR = 0.092, 95% CI = 0.018-0.465); medical (OR = 0.049, 95% C% CI = 0.009-0.259); other (OR = 0.097, 95% CI = 0.016-0.572)] independently predicted 30-day mortality. Peripherally inserted central catheter (OR = 13.970, 95% CI = 1.439-135.589), urea nitrogen (OR = 0.961, 95% CI = 0.933-0.990) and pre-discharge GFR (OR = 1.102, 95% CI = 1.067-1.137) independently predicted 30-day kidney recovery. Pre-dialysis Scr (OR = 0.997, 95% CI = 0.995-0.999), urea nitrogen (OR = 0.948, 95% CI = 0.912-0.986) and pre-discharge GFR (OR = 1.137 95% CI = 1.088-1.189) independently predicted 90-day kidney recovery.

Conclusions: PIRRT improves survival and kidney function recovery in AKI patients. In patients with previous GFR ≥ 30 mL/(min-1.73 m^2) and no prior maintenance dialysis, PIRRT at 3-5 sessions/week might be appropriate.

1 | INTRODUCTION

The morbidity of acute kidney injury (AKI) in hospitalised patients is high and shows a marked upward trend. Poor prognosis, high mortality and low renal recovery in AKI makes it an independent risk factor for end-stage renal disease.[1,2] A systematic review of 154 cohort studies (3 585 911 patients) performed to estimate the global incidence of AKI suggested a pooled incidence of 21.6% in adults, with a pooled AKI-associated mortality rate of 23.9%.[3]

Renal replacement therapy (RRT) is pivotal to AKI treatment. Prolonged intermittent renal replacement therapy (PIRRT), a new RRT pattern, combines the characteristics of continuous renal replacement therapy (CRRT) and intermittent haemodialysis (IHD) and is being used in clinic, including in adults and children.[4-6] There is currently no uniform and clear definition of PIRRT. It seems that any haemodialysis or haemodiafiltration that combines the characteristics of IHD and CRRT for prolonged, intermittent periods may be classified as PIRRT. The definition of PIRRT by Australian scholars includes: (a) use of modified or standard dialyser; (b) use of diffusion and/or convection; (c) solute clearance strength lower than IHD; (d) duration of treatment more than 3-4 hours ("prolonged"), but less than 8-12 hours ("intermittent").[4] PIRRT has some advantages over CRRT: (a) mild water and solute clearance; (b) stable haemodynamics; (c) interval treatment can allow for other procedures, including diagnosis, treatment and patient transportation; (d) reduced use of anticoagulants; and (e) low cost.[4,7,8]

Few studies have focused on the prognosis of AKI patients treated with PIRRT; therefore, the present study aimed to assess survival and renal recovery in these patients and to determine the associated risk factors.

What's known

- As a combined pattern of continuous renal replacement therapy (CRRT) and intermittent haemodialysis (IHD), PIRRT is effectively applied in acute kidney injury (AKI).
- Studies assessing the prognosis and associated factors of PIRRT in AKI patients are scarce.

What's new

- PIRRT improves 30-day survival and kidney function recovery in AKI patients. The effectiveness of PIRRT might be similar to that of CRRT.
- Thrombin time, total protein, Na+, number of organs injured, pre-discharge GFR, ward type, infection and dialysis frequency are associated with mortality. Pre-discharge GFR and pre-dialysis Scr are associated with renal recovery.
- In patients with previous GFR ≥ 30 mL/(min-1.73 m^2) and no prior maintenance dialysis, PIRRT at 3-5 sessions per week might be appropriate.

2 | MATERIALS AND METHODS

2.1 | Study design and patients

This was a retrospective case-control study of patients with AKI hospitalised based on electronic medical records in Shanghai General Hospital, Shanghai Jiao Tong University School of Medicine, from January 2012 to October 2018. Inclusion criteria were: (1) diagnosis according to the KDIGO 2012 definition of AKI, that is, at least one of the following: (i) serum creatinine (Scr) >26.5 μmol/L for 48 h; (ii) Scr > 1.5 times the baseline (confirmed or suspected) occurring within 7 days; and (iii) urine volume < 0.5 ml/kg/h for > 6 h after excluding obstructive nephropathy or dehydration; and (2) PIRRT after AKI occurrence. Exclusion criteria were: (a) diagnosis of chronic kidney disease (CKD) stage 4-5 or maintenance dialysis before AKI; (b) other types of RRT after AKI, such as CRRT and IHD; (c) loss to follow-up within 30 days post-discharge; or (d) incomplete data. The subjects were assigned to the 30-day survivor and death groups. Surviving patients were further divided into the kidney recovery and non-recovery groups according to renal function recovery status. The study was approved by the ethics committee of Shanghai General Hospital, Shanghai Jiao Tong University School of Medicine. The need for individual consent was waived by the committee.

2.2 | Representative PIRRT scheme

All subjects were treated with a Prismaflex CRRT system (Prismaflex M60 Set), using disposable haemodialysis filters and supporting tubes. The dialysis membrane area was 0.6 m^2 (AN69 membrane, Baxter, Deerfield, IL, USA). Dialysis was performed at a frequency of 3-7 times per week for 4-6 hours each time. The blood flow rate was set at 150-200 mL/min, while the displacement fluid flow rate was 25-30 mL/kg/h; the dialysate flow rate was 30-35 mL/min in the post-dilution mode. The dialysate contained sodium (135-145 mmol/L), potassium (2.0-4.0 mmol/L), calcium (1.25-1.5 mmol/L) and bicarbonate (25-35 mmol/L). Bacterial count was <0.1 CFU/mL and endotoxin levels were <0.03 EU/mL. The temperature of the dialysate was maintained at 35.5-36.5°C.

2.3 | Data collection and definition

Demographic characteristics (sex, age, AKI aetiology and AKI stage), clinical history (hypertension, diabetes mellitus (DM), coronary atherosclerotic heart disease (CAD), percutaneous transluminal coronary intervention (PCI), chronic obstructive pulmonary disease (COPD), severe hepatic diseases, malignant tumours and kidney diseases), pre-PIRRT laboratory tests (serum potassium (K^+), serum sodium (Na^+), serum chlorine (Cl^-), total protein, albumin, globulin, serum uric acid

(UA), urea nitrogen (BUN), Scr, glomerular filtration rate (GFR), serum calcium (Ca^{2+}), serum magnesium (Mg^{2+}), serum phosphorus (P), prothrombin time (PT), thrombin time (TT), haemoglobin, haematocrit and platelet count), pre-discharge laboratory tests (Scr and GFR), treatments (diuretics, angiotensin-converting enzyme inhibitor drugs (ACEI), angiotensin II receptor blockers (ARB), calcium channel blockers (CCB), β-blockers, lipid-lowering drugs, glucocorticoids and vasoactive agents) and other factors (peripherally inserted central catheter (PICC) during hospitalisation, albumin supplementation during dialysis, parenteral nutrition, PIRRT frequency, ward type, mechanical ventilation, infection and number of important organs injured (besides the kidney) were collected from the medical charts.

Infection was determined based on clinical manifestations and laboratory examination results such as fever, chills, elevated white blood cells (WBC), C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), interleukin (IL)-6 and procalcitonin (PCT). Ward types included the nephrology ward, ICU, surgical wards, internal medicine wards (except the nephrology ward) and other types. GFR was calculated by the EPI formula. High-frequency PIRRT was considered for six or seven dialysis sessions per week. Low-frequency PIRRT was reflected by 3-5 dialysis sessions per week. Mixed frequency indicated that patients had undergone both high and low-frequency dialysis sessions successively. Other frequency meant continuous dialysis for 2 days, especially.

2.4 | Outcomes and follow-up

The main outcome was the 30-day death, which was defined as death that occurred between AKI onset and 30 days post-discharge. The other outcomes included the recovery of renal function at 30 and 90 days post-discharge, respectively. Recovery of renal function was defined as stable decrease in serum creatinine (within the range of normal creatinine or 1.5 times of basal creatinine) combined with discontinuation of any form of RRT or urine volume more than 800 mL/24 h at 30 and 90 days post-discharge, respectively.[9]

Follow-up was performed at 30 and 90 days post-discharge, respectively, by phone and outpatient visits to determine the vital status and the recovery of renal function.

2.5 | Statistical analysis

SPSS 17.0 (IBM, Armonk, NY, USA) and R software (www.r-project.org) were used for statistical analysis. Continuous variables were tested for normality of distribution by the Kolmogorov-Smirnov test. Continuous variables with normal distribution were presented as mean ± standard deviation (SD) and analysed using the Student's *t* test. Categorical data were presented as frequency and analysed by the chi-square test. Continuous variables with skewed distribution were presented as median (Q1, Q3). Univariable and multivariable logistic regression analyses were performed to assess the short-term prognosis of patients with AKI after PIRRT and to determine the main factors affecting prognosis. $P < .05$ was considered statistically significant.

The propensity scores were estimated using logistic regression analysis and adjusted for covariates that were not examined in this study, including age, sex, hypertension, diabetes, CAD, malignant tumour, COPD, coronary intervention and liver cirrhosis. The matching was performed using an adjacent caliper value of 0.4 with a 1:1 ratio for the 30-day death and survival group. The MatchIt package of the R software (www.r-project.org) was used for propensity score matching (PSM).

3 | RESULTS

3.1 | Characteristics of the patients

Before the PSM, 576 subjects were included (Table 1). In total, the subjects had 3372 PIRRT sessions, with an average of 5.85 sessions per person. After the PSM, there were 250 patients in each group. The subjects experienced a total of 2993 PIRRT sessions, among which the survival group experienced a total of 1463 PIRRT sessions, with an average of 5.85 sessions per person. The death group had a total of 1530 PIRRT sessions, with an average of 6.12 sessions per person. There were no significant differences between the two groups in sex, age, hypertension, diabetes, CAD, malignant tumour, COPD, coronary intervention and liver cirrhosis (all $P > .05$) (Table 1).

3.2 | Factors associated with 30-day mortality

In this study, the aetiologies of AKI included severe infection (n = 76, 15.2%), drugs (n = 15, 3.0%), cardiorenal syndrome (n = 75, 15.0%), operation or trauma (n = 115, 23.0%), multiple organ dysfunction syndrome (MODS) (n = 32, 6.4%), urinary obstruction (n = 26, 5.2%), rhabdomyolysis (n = 19, 3.8%), renal parenchymal diseases (n = 22, 4.4%), haematological diseases (n = 17, 3.4%), poisoning (including organophosphorus poisoning, alcoholism, fish gall poisoning and scorpion poisoning; n = 17, 3.4%), hepatorenal syndrome (n = 14, 2.8%), severe water and electrolyte disturbance (n = 23, 4.6%), severe acute pancreatitis (SAP) (n = 32, 6.4%) and contrast medium (n = 4, 0.8%). The frequencies of severe infection, cardiorenal syndrome, operation/trauma and MODS were higher in the death group compared with the surviving patients (all $P < .05$) (Table 2).

Univariable analysis showed that the cause of AKI, high AKI stage, high PIRRT frequency, no kidney disease, infection, ward type, PICC use, no use of diuretics, ARB use, no CCB use, albumin supplementation, reduced pre-dialysis GFR, elevated pre-discharge Scr, reduced pre-discharge GFR, an elevated number of organs injured, high Na^+, higher Cl^-, low total proteins, low albumin, low globulin, high UA, high BUN, low Ca^{2+}, high PT, high TT and low

TABLE 1 Characteristics of the patients before and after PSM

	Before matching			After matching		
	30 d death group (n = 298)	30 d survival group (n = 278)	*P*	30 d death group (n = 250)	30 d survival group (n = 250)	*P*
Male, n (%)	200 (67.1)	191 (68.7)	.749	173 (69.1)	181 (72.3)	.491
Age, y, mean ± SD	64.8 ± 15.9	56.9 ± 17.2	<.001	62.5 ± 15.8	59.3 ± 16.3	.052
Age, n (%)			<.001			.202
20-29	12 (4.0)	25 (8.4)		12 (4.8)	20 (8.0)	
30-39	13 (4.4)	24 (8.1)		13 (5.2)	9 (3.6)	
40-49	25 (8.4)	43 (14.4)		25 (10.0)	36 (14.4)	
50-59	41 (13.8)	44 (14.8)		39 (15.6)	43 (17.2)	
60-69	74 (24.8)	70 (23.5)		65 (26.0)	70 (28.0)	
70-79	83 (27.9)	53 (17.8)		70 (28.0)	53 (21.2)	
≥80	50 (16.8)	19 (6.4)		26 (10.4)	19 (7.6)	
Hypertension	117 (39.3)	92 (33.1)	.147	91 (36.4)	91 (36.4)	1.000
Diabetes	69 (23.3)	52 (18.7)	.227	53 (21.2)	51 (20.4)	.912
CHD	34 (11.4)	30 (10.8)	.918	31 (12.4)	29 (11.6)	.891
COPD	9 (3.0)	8 (2.9)	1.000	6 (2.4)	8 (3.2)	.786
History of malignancy	59 (19.8)	48 (17.3)	.500	51 (20.4)	48 (19.2)	.822
Coronary intervention	10 (3.4)	13 (4.7)	.551	10 (4.0)	12 (4.8)	.827
Liver cirrhosis	30 (10.1)	11 (4.0)	.007	12 (4.8)	11 (4.3)	1.000
CKD			.030			.025
no	259 (86.9)	221 (79.4)		217 (86.8)	195 (78.0)	
1	8 (2.6)	16 (5.7)		6 (2.4)	16 (6.4)	
2	3 (1.0)	10 (3.5)		3 (1.2)	9 (3.6)	
3	28 (9.3)	31 (11.1)		24 (9.6)	30 (12.0)	
PIRRT frequency			<.001			<.001
High	136 (45.6)	104 (37.4)		115 (46.0)	94 (37.6)	
Low	45 (15.1)	82 (29.4)		39 (15.6)	78 (31.2)	
Mixed	32 (10.7)	31 (11.1)		27 (10.8)	28 (11.2)	
Other	85 (28.5)	61 (21.9)		69 (27.6)	50 (20.0)	

Abbreviations: AKI, acute kidney injury; CHD, coronary heart disease; CKD, chronic kidney disease; COPD, chronic obstructive pulmonary disease; PIRRT, prolonged intermittent renal replacement therapy; PSM, propensity score matching.

platelet count were significantly associated with 30-day mortality (all $P < .05$) (Table 2).

Multivariable analysis showed that low PIRRT frequency (OR = 2.165, 95% CI: 1.178-3.978, $P = .01$), infection (OR = 0.447, 95% CI: 0.251-0.795, $P = .006$), number of organs injured (OR = 0.478, 95% CI: 0.346-0.661, $P < .001$), ICU (OR = 0.042, 95% CI: 0.008-0.211, $P < .001$ vs. renal ward), surgery ward (OR = 0.092, 95% CI: 0.018-0.465, $P = .004$ vs. renal ward), medical ward (OR = 0.049, 95% CI: 0.009-0.259, $P < .001$ vs. renal ward), other ward (OR = 0.097, 95% CI: 0.016-0.572, $P = .01$ vs. renal ward), Na^+ (OR = 0.958, 95% CI: 0.928-0.988, $P = .006$), total protein (OR = 1.047, 95% CI: 1.022-1.072, $P < .001$), TT (OR = 0.959, 95% CI: 0.936-0.983, $P = .001$) and pre-discharge GFR (OR = 1.024, 95% CI: 1.017-1.031, $P < .001$) were independently associated with 30-day mortality in patients with AKI treated with PIRRT (Table 3).

3.3 | Factors associated with 30-day and 90-day renal recovery

Univariable analysis showed that cancer history, renal disease history, infection, PICC, ARB use, pre-dialysis Scr, pre-dialysis GFR, pre-dialysis BUN, pre-dialysis phosphorus, pre-dialysis HCT, pre-dialysis haemoglobin, pre-discharge Scr and pre-discharge GFR were associated with 90-day renal recovery (all $P < .05$) (Table 4).

Then, multivariable analysis showed that PICC (OR = 13.970, 95% CI: 1.439-135.589, $P = .023$) pre-dialysis BUN (OR = 0.961, 95% CI: 0.933-0.99, $P = .009$) and pre-discharge GFR (OR = 1.102, 95% CI: 1.067-1.137, $P < .001$) were independently associated with 30-day non-recovery of the kidney function in AKI after PIRRT (Table 5).

Multivariable analysis showed that pre-dialysis Scr (OR = 0.997, 95% CI: 0.995-0.999, $P = .006$), pre-dialysis BUN (OR = 0.948, 95%

TABLE 2 Comparison of clinical characteristic between survival and death patients

	Total (n = 500)	Death (n = 250)	Survival (n = 250)	*P*
AKI causes				
Severe infection	76 (15.2)	51 (20.4)	25 (10.0)	<.001
Drugs	18 (3.6)	4 (1.6)	14 (5.6)	
Cardiorenal syndrome	75 (15.0)	43 (17.2)	32 (12.8)	
Operation or trauma	115 (23.0)	60 (24.0)	55 (22.0)	
MODS	32 (6.4)	24 (9.6)	8 (3.2)	
Urinary obstruction	26 (5.2)	4 (1.6)	22 (8.8)	
Rhabdomyolysis	19 (3.8)	2 (0.8)	17 (6.8)	
Renal parenchymal diseases	22 (4.4)	2 (0.8)	20(8.0)	
Haematological diseases	17 (3.4)	8 (3.2)	9 (3.6)	
Poisoning	17 (3.4)	9 (3.6)	8 (3.2)	
Hepatorenal syndrome	14 (2.8)	8 (3.2)	6 (2.4)	
Massive fluid loss (massive gastrointestinal bleeding, nausea, vomiting or diarrhoea)	23 (4.6)	11 (4.4)	12 (4.8)	
SAP	33 (6.4)	17 (6.8)	15 (6.0)	
Contrast medium	4 (0.8)	2 (0.8)	2 (0.8)	
Severe water and electrolyte disturbance	10 (2.0)	5 (2.0)	5 (2.0)	
AKI stage				
Stage 3	283 (56.6)	140 (56.0)	143 (57.2)	.001
Stage 2	178 (35.6)	101 (40.4)	77 (30.8)	
Stage 1	39 (7.8)	9 (3.6)	30 (12.0)	
PIRRT frequency				
High	209 (41.8)	115 (46.0)	94 (37.6)	.001
Low	117 (23.4)	39 (15.6)	78 (31.2)	
Mixed	55 (11.0)	27 (10.8)	28 (11.2)	
Other	119 (23.8)	69 (27.6)	50 (20.0)	
Kidney disease	88 (17.6)	33 (13.2)	55 (22.0)	.01
Infection	374 (74.8)	203 (81.2)	171 (68.4)	.001
Ward				<.001
Nephrology ward	44 (8.8)	2 (0.8)	42 (16.8)	
ICU	201 (40.2)	122 (48.8)	79 (31.6)	
Surgery Ward	141 (28.2)	72 (28.8)	69 (27.6)	
Medical ward (non-nephrology)	71 (14.2)	39 (15.6)	32 (12.8)	
Other	43 (8.6)	15 (6.0)	28 (11.2)	
PICC	54 (10.8)	34 (13.6)	20 (8.0)	.046
Mechanical ventilation	34 (6.8)	22 (8.8)	12 (4.8)	.08
Diuretics	100 (20.0)	39 (15.6)	61 (24.4)	.015
ACEI	12 (2.4)	6 (2.4)	6 (2.4)	1.000
ARB	36 (7.2)	12 (4.8)	24 (9.6)	.042
CCB	143 (28.6)	57 (22.8)	86 (34.4)	.004
Albumin supplementation	295 (59.0)	162 (64.8)	133 (53.2)	.009
PIRRT times	6.0 (2, 7)	6.1 (2, 9)	5.9 (2, 9)	.671
Pre-dialysis Scr (μmol/L)	406.4 (222.2, 548.7)	389.4 (233.7, 513.1)	423.4 (209.8, 608.0)	.126
Pre-dialysis GFR (ml/min)	25.8 (8.0, 24.9)	21.7 (8.5, 24.2)	29.9 (7.3, 27.2)	.008
Pre-discharge Scr (μmol/L)	312.7 (115.1, 419.1)	348.4 (193.5, 455.7)	277.1 (80.2, 371.3)	.002

(Continues)

TABLE 2 (Continued)

	Total (n = 500)	Death (n = 250)	Survival (n = 250)	*P*
Pre-discharge GFR (ml/min)	43.6 (11.1, 54.4)	25.8 (10.7, 28.0)	61.4 (12.9 ,87.2)	<.001
Number of organs injured (except renal)	0.8 (0, 1)	1.0 (1, 2)	0.5 (0, 1)	<.001
K^+ (mmol/L)	4.5 ± 1.7	4.6 ± 2.1	4.4 ± 1.1	.174
Na^+ (mmol/L)	139.8 ± 7.8	141.4 ± 8.4	138.1 ± 6.9	<.001
Cl^- (mmol/L)	103.3 ± 8.0	104.0 ± 8.5	102.6 ± 7.5	.047
Ca^{2+} (mmol/L)	1.9 ± 0.4	1.9 ± 0.4	2.0 ± 0.3	.007
Mg^{2+} (mmol/L)	1.0 ± 0.3	0.9 ± 0.3	1.0 ± 0.3	.237
P (mmol/L)	1.8 ± 1.2	1.8 ± 1.1	1.8 ± 1.2	.966
Total protein (g/L)	56.1 ± 10.9	54.1 ± 11.1	58.2 ± 10.3	<.001
Albumin (g/L)	29.5 ± 6.8	28.5 ± 6.7	30.6 ± 6.8	.001
Globulin (g/L)	26.6 ± 6.9	25.6 ± 7.2	27.6 ± 6.5	.001
UA (μmol/L)	483.6 ± 228.8	509.8 (341.5, 635.2)	457.5 (319.0, 588.8)	.012
BUN (mmol/L)	24.8 (14.8, 32.2)	27.0 (15.1, 36.4)	22.6 (14.4, 29.3)	.004
PT(s)	17.8 (12.5, 17.8)	19.5 (13.0, 19.0)	16.1 (12.0, 15.9)	.003
TT(s)	22.6(17.0, 21.9)	25.0 (17.6, 23.7)	20.3 (16.7, 20.7)	.001
Thrombocytocrit	0.14(0.07, 0.19)	0.13 (0.06, 0.17)	0.16 (0.09, 0.20)	.002
30-day recovery		—	151	
90-day recovery		—	166	

Abbreviations: AKI, acute kidney injury; ARB, angiotensin receptor blocker; BUN, urea nitrogen; CCB, calcium channel blocker; GFR, glomerular filtration rate; ICU, intensive care unit; MODS, multi-organ dysfunction syndrome; PICC, peripherally inserted central catheter; PIRRT, prolonged intermittent renal replacement therapy; PT, prothrombin time; SAP, severe acute pancreatitis; Scr, serum creatinine; TT, thrombin time; UA, serum uric acid.

CI: 0.912-0.986, $P = .008$) and pre-discharge GFR (OR = 1.137, 95% CI: 1.088-1.189, $P < .001$) were independently associated with 90-day non-recovery of the kidney function in AKI after PIRRT (Table 6).

4 | DISCUSSION

In the non-PSM population, the present study suggests that the mortality rate of AKI patients treated with PIRRT was 51.7%, with a 30-day kidney recovery rate of 30.6% and a 90-day kidney recovery rate of 33.4%. This is almost consistent with the mortality and renal recovery rates reported in AKI patients treated with CRRT.[10] Actually, numerous prospective studies have confirmed that PIRRT and CRRT achieve similar outcomes in patients with AKI.[9,11] A meta-analysis showed no significant differences between PIRRT and CRRT in mortality (RR = 0.90; 95% CI: 0.74-1.11) and renal function recovery (RR = 1.12, 95% CI: 0.83-1.76).[11] The largest RCT so far revealed a mortality rate in the PIRRT group of 55.6%, similar to that of the present study, while mortality in the CRRT group was 49.6%, without significant difference.[12] Studies even suggested that the renal recovery rate after PIRRT is higher than that obtained with CRRT.[13] A retrospective study by Khanal et al compared the efficacies and clinical outcomes of CRRT, IHD and PIRRT in 146 patients with severe AKI and suggested that PIRRT should be the first choice for patients with severe AKI.[14] Since PIRRT has reduced costs and financial burden compared with CRRT,[7] it may be a more suitable RRT for developing countries, such as China.

According to the above regression analysis, the final GFR before discharge was one of the main factors affecting survival in AKI patients receiving PIRRT. For 30-day renal function recovery, pre-dialysis BUN and final GFR before discharge were the main influencing factors; pre-dialysis Scr, BNU and pre-discharge GFR were associated with 90-day renal recovery. Undoubtedly, Scr, GFR and BUN were associated with renal function and renal recovery and timely and adequate PIRRT results in improved prognosis.[15,16] However, the exact timing of dialysis initiation remains unstandardised[17,18] and few studies have been conducted to determine the optimal initiation time for PIRRT. Therefore, further RCTs are needed to explore this matter.

Concerning dialysis frequency, this study showed that patients with high frequency (6-7 times/wk) PIRRT unexpectedly did not manifest better prognosis compared with those with low-frequency treatment (3-5 times/wk), while the impact of frequency on renal recovery was not statistically significant. This may be because critically ill patients tend to have higher rates of PIRRT, especially those who die, but PIRRT is only a kidney replacement treatment, not a cure and does not alter the outcome of such patients. Thus, for AKI patients with previous renal function grade 1-3 and no prior maintenance dialysis (this was one of the inclusion criteria in this study), administering PIRRT 3-5 times per week (4-6 h/session) with PIRRT correction at each session seems to be

TABLE 3 Univariable and multivariable analysis of factors associated with 30-day mortality

	Univariable				Multivariable			
	OR	95% CI		*P*	OR	95% CI		*P*
Male	0.856	0.582	1.260	.432				
AKI causes				<.001				
Severe infection	1.000							
Drugs	7.140	2.129	23.94	.001				
Cardiorenal syndrome	1.518	0.783	2.943	.217				
Operation or trauma	1.870	1.024	3.415	.042				
MODS	0.680	0.268	1.727	.418				
Urinary obstruction	11.220	3.490	36.074	<.001				
Rhabdomyolysis	17.340	3.712	80.991	<.001				
Renal parenchymal diseases	20.400	4.416	94.238	<.001				
Haematological diseases	2.295	0.790	6.663	.127				
Poisoning	1.813	0.625	5.265	.274				
Hepatorenal syndrome	1.530	0.479	4.889	.473				
Massive fluid loss (massive gastrointestinal bleeding, nausea, vomiting, diarrhoea)	2.225	0.863	5.742	.098				
SAP	1.800	0.775	4.183	.172				
Contrast medium	2.040	0.271	15.34	.489				
Severe water and electrolyte disturbance	2.040	0.540	7.704	.293				
AKI stage				.001				
Stage 3	1.000							
Stage 2	0.746	0.512	1.088	.128				
Stage 1	3.263	1.495	7.123	.003				
PIRRT frequency				.001				
High	1.000				1.000			
Low	2.447	1.527	3.920	<.001	2.165	1.178	3.978	**.013**
Mixed	1.269	0.700	2.300	.433	1.626	0.794	3.329	.184
Other	0.887	0.563	1.397	.604	0.729	0.404	1.316	.295
Renal disease history	1.855	1.156	2.976	.010				
Infection	0.501	0.331	0.758	.001	0.447	0.251	0.795	**.006**
Number of damage to vital organs	0.369	0.278	0.489	<.001	0.478	0.346	0.661	**<.001**
Ward				<.001				
Nephrology ward	1.000				1.000			
ICU	0.031	0.007	0.131	<.001	0.042	0.008	0.211	**<.001**
Surgery Ward	0.046	0.011	0.196	<.001	0.092	0.018	0.465	**.004**
Medical ward (non-nephrology)	0.039	0.009	0.174	<.001	0.049	0.009	0.259	**<.001**
Other	0.089	0.019	0.419	.002	0.097	0.016	0.572	**.01**
PCI	1.210	0.513	2.854	.663				
PICC	0.552	0.308	0.989	.046				
Mechanical ventilation	0.523	0.253	1.080	.080				
Uretic	1.746	1.117	2.731	.015				
ACEI	1.000	0.318	3.144	1.000				
ARB	2.106	1.029	4.312	.042				

(Continues)

TABLE 3 (Continued)

	Univariable				Multivariable			
	OR	95% CI		P	OR	95% CI		P
CCB	1.776	1.197	2.634	.004				
β-blocker	1.255	0.807	1.952	.313				
Albumin supplement	0.617	0.431	0.884	.009				
Antiuric acid therapy	2.531	0.486	13.168	.270				
Lipid - lowering agent	1.348	0.833	2.182	.224				
Parenteral nutrition	1.115	0.767	1.620	.568				
PIRRT times	0.995	0.970	1.020	.671				
Pre-dialysis								
Thrombocytocrit	17.757	2.79	113.007	.002				
HCT	2.171	0.265	17.796	.470				
Haemoglobin	1.003	0.997	1.009	.334				
Scr	1.000	1.000	1.001	.489				
GFR	1.008	1.002	1.013	.008				
K^+	0.899	0.772	1.048	.174				
Na^+	0.945	0.922	0.968	<.001	0.958	0.928	0.988	**.006**
Cl^-	0.977	0.956	1.000	.047				
Total protein	1.037	1.019	1.055	<.001	1.047	1.022	1.072	**<.001**
Albumin	1.047	1.019	1.076	.001				
Globulin	1.045	1.018	1.073	.001				
Lactic dehydrogenase	1.000	1.000	1.000	.317				
UA	0.999	0.998	1.000	.012				
BUN	0.981	0.969	0.994	.004				
Ca^{2+}	2.275	1.246	4.153	.007				
Mg^{2+}	1.499	0.767	2.930	.237				
P	0.997	0.850	1.169	.966				
PT	0.971	0.952	0.990	.003				
TT	0.967	0.947	0.987	.001	0.959	0.936	0.983	**.001**
Pre-discharge								
Scr	0.999	0.998	1.000	.002				
GFR	1.018	1.013	1.024	<.001	1.024	1.017	1.031	**<.001**

Note: The multivariable analysis used the forward stepwise method.

The adjustment variables included in the multivariable analysis: AKI causes, AKI stage, PIRRT frequency, renal disease history, infection, number of damage to vital organs, ward, PICC, uretic, ARB, CCB, albumin supplement, thrombocytocrit, pre-dialysis GFR, Na^+, Cl^-, total protein, albumin, globulin, UA, BUN, Ca^{2+}, PT,TT and pre-discharge Scr and GFR.

Abbreviations: ACEI, angiotensin-converting enzyme inhibitor; AKI, acute kidney injury; ARB, angiotensin receptor blocker; BUN, urea nitrogen; CCB, calcium channel blocker; CI, confidence interval; GFR, glomerular filtration rate; HCT, haematocrit; ICU, intensive care unit; MODS, multi-organ dysfunction syndrome; OR, odds ratio; PCI, percutaneous coronary intervention; PICC, peripherally inserted central catheter; PIRRT, prolonged intermittent renal replacement therapy; PT, prothrombin time; SAP, severe acute pancreatitis; TT, thrombin time; UA, serum uric acid.

sufficient. Although several studies showed that increasing the dialysis doses in patients with AKI (such as prolonging dialysis duration or increasing its frequency) can improve survival and kidney recovery,[18,19] the present study yielded discrepant results. In fact, some studies have come to the same conclusion as we have. An American study showed that intensive renal support in critically ill patients with AKI did not decrease mortality, improve the recovery of the kidney function or reduced the rate of non-kidney organ failure compared with less intensive therapy.[20] In addition, the Hannover Dialysis Outcome study compared the short-term mortality and renal recovery in 156 patients with AKI randomised to intensified extended dialysis (IED) and standard extended dialysis (SED); there were no significant differences in survival (70.4% in the IED group vs 70.7% in the SED group, $P = .97$) and renal recovery between the two groups.[21] Therefore, for AKI patients with good basal renal function in developing countries (previous renal

TABLE 4 Comparison of clinical characteristic between the patients with 90-day recovery or not

	90-d non-recovery (n = 84)	90-d recovery (n = 166)	*P*
Cancer history	24 (28.5)	24 (14.4)	.008
Renal disease history	33 (39.2)	22 (13.2)	<.001
Infection	50 (59.5)	121 (72.8)	.033
Number of organs injured (except renal)	0.5 ± 0.7	0.5 ± 0.6	.672
PICC	2 (2.3)	18 (10.8)	.034
Diuretics	15 (17.8)	46 (27.7)	.089
ARB	2 (2.3)	22 (13.2)	.015
CCB	31 (36.9)	55 (33.1)	.553
AKI stage			<.001
Stage 3	1 (1.1)	29 (17.4)	
Stage 2	22 (26.1)	55 (33.1)	
Stage 1	61 (72.6)	82 (49.3)	
Ward			
Nephrology ward	20 (23.8)	22 (13.2)	.069
ICU	20 (23.8)	59 (35.5)	
Surgery Ward	27 (32.1)	42 (25.3)	
Medical ward (non-nephrology)	11 (13.0)	21 (12.6)	
Other	6 (7.1)	22 (13.2)	
PIRRT times	6.4 (3, 9)	5.6 (2, 7)	.307
PIRRT frequency			
High	25 (29.7)	69 (41.5)	<.001
Low	41 (48.8)	37 (22.2)	
Mixed	8 (9.5)	20 (12.0)	
Other	10 (11.9)	40 (24.0)	
Pre-dialysis			
Scr (μmol/L)	610.6 (417.9, 796.9)	328.6 (122.3, 465.7)	<.001
GFR (ml/min)	10.1 (5.4, 11.9)	39.9 (9.8, 47.7)	<.001
UA (μmol/L)	475.5 (380.2, 590.5)	448.3 (295.0, 587.8)	.339
BUN (mmol/L)	27.8 (21.4, 32.8)	19.9 (9.3, 25.9)	<.001
Na^+ (mmol/L)	138.0 ± 6.2	138.2 ± 7.2	.82
P(mmol/L)	2.26 ± 1.52	1.61 ± 0.93	.001
HCT	0.29 ± 0.07	0.32 ± 0.09	.004
Haemoglobin g/L	94.9 ± 21.2	107.4 ± 29.5	.001
TT(s)	20.1 ± 7.0	20.4 ± 10.1	.805
Pre-discharge			
Scr (μmol/L)	556.1 (307.5, 737.7)	135.9 (65.3, 156.9)	<.001
GFR (ml/min)	12.3 (5.8, 16.6)	86.2 (38.2, 116.7)	<.001

Abbreviations: AKI, acute kidney injury; ARB, angiotensin receptor blocker; BUN, urea nitrogen; CCB, calcium channel blocker; GFR, glomerular filtration rate; HCT, haematocrit; ICU, intensive care unit; PICC, peripherally inserted central catheter; PIRRT, prolonged intermittent renal replacement therapy; PT, prothrombin time; Scr, serum creatinine; TT, thrombin time; UA, serum uric acid.

function grade 1-3 and no maintenance dialysis before), we tend to recommend low-frequency dialysis.

As for equipment, the dialyser used in this study was a Prismaflex continuous blood purification device, which is a CRRT machine. Fresenius (2008 H, 4008F, 4008S ArRT-Plus) and Gambro (AK200S Ultra 1, Integra) dialysers are mostly employed routinely.[4,11,12,14] In fact, few studies have employed CRRT machines for PIRRT. In consideration of our findings were similar to those obtained with PIRRT instruments. This may suggest that CRRT machines can be used directly for PIRRT.

Regarding specific dialysis schemes, the duration of a single PIRRT dialysis in previous studies ranged from 6 to 12 hours, with an average of 8 hours,[10,11] while dialysis in the present study lasted 4-6 hours. To our knowledge, this is the retrospective study with the

TABLE 5 Univariable and multivariable analyses of factors associated with 30-day renal recovery

	Univariable				Multivariable			
	OR	95% CI		*P*	OR	95% CI		*P*
Male	0.822	0.463	1.458	.502				
Age, n (%)				.138				
20-29	1.000							
30-39	0.389	0.045	3.324	.388				
40-49	0.175	0.035	0.871	.033				
50-59	0.140	0.029	0.681	.015				
60-69	0.157	0.034	0.730	.018				
70-79	0.157	0.033	0.745	.020				
≥80	0.081	0.014	0.452	.004				
Hypertension	0.526	0.311	0.890	.017				
Diabetes	0.680	0.366	1.265	.224				
CHD	0.670	0.308	1.457	.312				
COPD	0.646	0.158	2.646	.544				
Liver cirrhosis	6.950	0.876	55.175	.067				
Cancer	0.532	0.282	1.003	.051				
Infection	1.435	0.836	2.465	.190				
Number of damage to vital organs	0.840	0.562	1.256	.396				
Renal disease history	0.182	0.095	0.352	<.001				
PICC	6.564	1.488	28.954	.013	13.970	1.439	135.589	0.023
Mechanical ventilation	0.914	0.282	2.964	.881				
Uretic	1.014	0.562	1.831	.963				
ACEI	3.356	0.386	29.167	.272				
ARB	2.098	0.802	5.485	.131				
CCB	0.601	0.353	1.021	.060				
Albumin supplement	1.118	0.673	1.859	.666				
AKI causes				.722				
Severe infection	1.000							
Drugs	20.258	14210.360	.	.999				
Cardiorenal syndrome	1.944	0.192	19.741	.574				
Operation or trauma	0.778	0.176	3.431	.740				
MODS	6.28E+08	0.000	.	.998				
Urinary obstruction	2.41E-10	0.000	.	.999				
Rhabdomyolysis	0.583	0.08	4.271	.596				
Renal parenchymal diseases	0.700	0.173	2.836	.617				
Haematological diseases	0.500	0.163	1.529	.224				
Poisoning	0.502	0.181	1.397	.187				
Hepatorenal syndrome	0.233	0.044	1.248	.089				
Massive fluid loss (massive gastrointestinal bleeding, nausea, vomiting or diarrhoea)	0.324	0.097	1.088	.068				
SAP	0.556	0.151	2.043	.376				
Contrast medium	0.475	0.138	1.643	.240				
Severe water and electrolyte disturbance	0.111	0.018	0.671	.017				

(Continues)

TABLE 5 (Continued)

	Univariable				Multivariable			
	OR	95% CI		P	OR	95% CI		P
AKI stage				.004				
Stage 3	1.000							
Stage 2	1.420	0.809	2.495	.222				
Stage 1	26.293	3.487	198.268	.002				
Ward				.119				
Nephrology ward	1.000							
ICU	2.242	1.042	4.824	.039				
Surgery Ward	1.272	0.590	2.743	.540				
Medical ward (non-nephrology)	2.100	0.814	5.420	.125				
Other	2.750	0.992	7.621	.052				
PIRRT times	0.980	0.941	1.019	.311				
PIRRT frequency				.003				
High	1.000							
Low	0.380	0.204	0.707	.002				
Mixed	0.760	0.318	1.818	.538				
Other	1.400	0.652	3.008	.388				
Pre-dialysis								
Scr	0.997	0.996	0.998	<.001				
GFR	1.060	1.032	1.088	<.001				
K^+	0.850	0.665	1.085	.191				
Na^+	1.002	0.965	1.039	.930				
Cl^-	1.004	0.97	1.038	.840				
Total protein	0.985	0.961	1.010	.238				
Albumin	0.976	0.94	1.014	.215				
Globulin	0.989	0.95	1.028	.566				
Lactic dehydrogenase	1.000	1.000	1.000	.416				
UA	0.999	0.998	1.000	.174				
BUN	0.959	0.937	0.981	<.001	0.961	0.933	0.99	.009
Ca^{2+}	0.564	0.246	1.297	.178				
Mg^{2+}	0.309	0.117	0.822	.019				
P	0.619	0.448	0.855	.004				
Thrombocytocrit	1.203	0.081	17.793	.893				
HCT	214.912	7.628	6054.914	.002				
Haemoglobin	1.018	1.008	1.029	.001				
PT(s)	1.016	0.984	1.048	.328				
TT(s)	1.008	0.978	1.039	.594				
Pre-discharge								
Scr	0.990	0.987	0.993	<.001				
GFR	1.097	1.069	1.126	<.001	1.102	1.067	1.137	<.001

Note: The multivariable analysis used the forward stepwise method.

The adjustment variables included in the multivariable analysis: hypertension, renal disease history, PICC, AKI stage, PIRRT frequency, pre-dialysis Scr, pre-dialysis GFR, BUN, Mg^{2+}, P, HCT, haemoglobin and pre-discharge Scr and GFR.

Abbreviations: ACEI, angiotensin-converting enzyme inhibitor; AKI, acute kidney injury; ARB, angiotensin receptor blocker; BUN, urea nitrogen; CCB, calcium channel blocker; CI, confidence interval; GFR, glomerular filtration rate; HCT, haematocrit; ICU, intensive care unit; MODS, multi-organ dysfunction syndrome; OR, odds ratio; PCI, percutaneous coronary intervention; PICC, peripherally inserted central catheter; PIRRT, prolonged intermittent renal replacement therapy; PT, prothrombin time; SAP, severe acute pancreatitis; TT, thrombin time; UA, serum uric acid.

TABLE 6 Univariable and multivariable analyses of factors associated with 90-day renal recovery

	Univariable				Multivariable		
	OR	95% CI		*P*	OR	95% CI	*P*
Male	0.984	0.546	1.771	.956			
Age, n (%)				.236			
20-29	1.000						
30-39	0.889	0.070	11.280	.928			
40-49	0.253	0.05	1.281	.097			
50-59	0.154	0.032	0.750	.021			
60-69	0.200	0.043	0.933	.041			
70-79	0.199	0.042	0.951	.043			
≥80	0.153	0.027	0.854	.032			
Hypertension	0.769	0.448	1.321	.341			
Diabetes	1.015	0.529	1.949	.964			
CHD	0.807	0.362	1.798	.600			
COPD	0.494	0.120	2.026	.327			
Liver cirrhosis	5.321	0.669	42.282	.114			
Cancer history	0.423	0.223	0.802	.008			
Infection	1.828	1.051	3.182	.033			
Number of damage to vital organs	0.914	0.604	1.385	.672			
Renal disease history	0.236	0.126	0.442	<.001			
PICC	4.986	1.129	22.028	.034			
Mechanical ventilation	0.696	0.214	2.261	.546			
Uretic	1.763	0.917	3.390	.089			
ACEI	2.578	0.296	22.425	.391			
ARB	6.264	1.436	27.317	.015			
CCB	0.847	0.489	1.466	.553			
Albumin supplement	1.051	0.621	1.778	.854			
AKI causes				.567			
Severe infection	1.000						
Drugs	510149950.4	0.000	.	.999			
Cardiorenal syndrome	1.579	0.153	16.310	.701			
Operation or trauma	0.632	0.139	2.862	.551			
MODS	510149950.400	0.000	.	.998			
Urinary obstruction	0.000	0.000	.	.999			
Rhabdomyolysis	0.474	0.063	3.538	.466			
Renal parenchymal diseases	0.568	0.136	2.369	.438			
Haematological diseases	0.603	0.187	1.947	.398			
Poisoning	0.553	0.190	1.611	.277			
Hepatorenal syndrome	0.947	0.150	5.994	.954			
Massive fluid loss (massive gastrointestinal bleeding, nausea, vomiting or diarrhoea)	0.263	0.076	0.913	.035			
SAP	1.026	0.241	4.369	.972			
Contrast medium	0.474	0.131	1.707	.253			
Severe water and electrolyte disturbance	0.090	0.015	0.557	.010			

(Continues)

TABLE 6 (Continued)

	Univariable				Multivariable			
	OR	95% CI		P	OR	95% CI		P
Ward				.069				
Nephrology ward	1.000							
ICU	2.682	1.217	5.908	.014				
Surgery Ward	1.414	0.652	3.069	.381				
Medical ward (non-nephrology)	1.736	0.673	4.479	.254				
Other	3.333	1.124	9.884	.030				
AKI stage				.002				
Stage 3	1.000							
Stage 2	1.860	1.026	3.373	.041				
Stage 1	21.573	2.859	162.76	.003				
Continuous blood purification times	0.979	0.940	1.020	.307				
PIRRT frequency				<.001				
High	1.000							
Low	0.327	0.173	0.619	.001				
Mixed	0.906	0.354	2.316	.836				
Other	1.449	0.632	3.325	.381				
Pre-dialysis								
GFR	1.090	1.05	1.132	<.001				
Scr	0.996	0.995	0.997	<.001	0.997	0.995	0.999	.006
K^+	0.834	0.651	1.068	.151				
Na^+	1.004	0.967	1.044	.820				
Cl^-	1.024	0.986	1.064	.214				
Total protein	0.985	0.96	1.011	.247				
Albumin	0.970	0.932	1.008	.123				
Globulin	0.996	0.956	1.037	.829				
Lactic dehydrogenase	1.000	1.000	1.000	.371				
UA	0.999	0.998	1.001	.339				
BUN	0.954	0.931	0.977	<.001	0.948	0.912	0.986	.008
Ca^{2+}	0.623	0.266	1.459	.276				
Mg^{2+}	0.407	0.158	1.051	.063				
P	0.566	0.403	0.795	.001				
HCT	156.700	5.104	4810.838	.004				
Haemoglobin	1.017	1.007	1.028	.001				
PT	1.01	0.98	1.04	.533				
TT	1.004	0.974	1.034	.805				
Pre-discharge								
Scr	0.989	0.986	0.992	<.001				
GFR	1.139	1.096	1.183	<.001	1.137	1.088	1.189	<.001

Note: The multivariable analysis used the forward stepwise method.

The adjustment variables included in the multivariable analysis: cancer history, infection, renal disease history, PICC, ARB, AKI stage, PIRRT frequency, HCT, haemoglobin, pre-dialysis GFR, pre-dialysis Scr, BUN, P and pre-discharge Scr and GFR.

Abbreviations: ACEI, angiotensin-converting enzyme inhibitor; AKI, acute kidney injury; ARB, angiotensin receptor blocker; BUN, urea nitrogen; CCB, calcium channel blocker; CI, confidence interval; GFR, glomerular filtration rate; HCT, haematocrit; ICU, intensive care unit; MODS, multi-organ dysfunction syndrome; OR, odds ratio; PCI, percutaneous coronary intervention; PICC, peripherally inserted central catheter; PIRRT, prolonged intermittent renal replacement therapy; PT, prothrombin time; SAP, severe acute pancreatitis; TT, thrombin time; UA, serum uric acid.

largest sample size in developing countries, but it was not designed to look specifically for the dialysis scheme. Nevertheless, considering that China is a developing country, the gap between huge medical needs and the limited medical human and material resources is significant. Although PIRRT costs less than CRRT, it is still an important financial burden for Chinese patients. Considering that in our research, survival and renal recovery rates were similar to those reported by other authors, a single 4-6-hours PIRRT seems to be a non-inferior and acceptable choice for Chinese patients. Indeed, some authors explored the relationship between the duration of a single PIRRT and its efficacy. Albino et al observed 75 subjects for 18 months and found no significant difference in prognosis between PIRRT dialysis durations (6 vs 10 hours).[19] If PIRRT duration could be shortened to 4-6 hours with guaranteed efficacy, it would greatly help reduce the risk associated with RRT and decrease the costs and allow more patients to be treated daily with a single machine. Nevertheless, additional studies have to be performed to address this matter.

According to the above multivariable analysis, TT, total protein, Na^+, number of organs injured, ward type and infection were independently associated with mortality; while PICC was related to renal recovery. The total protein is an index reflecting the nutritional status of patients. Pre-existing malnutrition is an independent risk factor for mortality in hospitalised patients and may increase the likelihood of inpatient mortality, complications and the use of medical resources in patients with AKI.[20] TT reflects the coagulation function. In dialysed patients, anticoagulants are often administered to reduce the risk of circulating coagulation, which, in turn, increases the risk of bleeding. Maintaining the balance between the risk of circulating coagulation and bleeding remains a major challenge in RRT.[21] Hypernatremia is common in critically ill patients, especially ICU patients. The prognosis of hypernatremia is poor and its mortality rate can reach 42%-75%.[22] Through a retrospective analysis, Mezones-Holguin et al[23] confirmed that hypernatremia is associated with mortality in patients with AKI. Gao et al[24] included 13 621 ICU AKI patients into a Cox proportional risk model and reached a similar conclusion. Venous access devices, such as PICC, are of pivotal importance for an increasing number of critically ill patients in a variety of disease states and in a variety of clinical settings (emergency, intensive care and surgery) and for different purposes (fluids or drugs infusions, parenteral nutrition, antibiotic therapy, hemodynamic monitoring, procedures of dialysis/apheresis).[25] Our research indicated that PICC was independently associated with 30-day renal recovery, while had nothing to do with 90-day renal recovery. More studies are needed for confirmation.

In the present study, the use of diuretics, ARB and CCB was associated with the survival group. The effects of diuretics in AKI are controversial and they might have benefits that are not related to the AKI, but rather could be used to manage pulmonary oedema secondary to volume overload.[26,27] On the contrary, ARBs are associated with an increased risk of AKI if they develop a condition associated with hypovolemia and hypertension.[28] In addition, the use of an ARB after AKI could reduce mortality.[29] CCBs have renal protective effects that could be associated with decreased mortality after AKI.[30] Nevertheless, those three drug classes were not associated with 30-day mortality in the above multivariable analysis. Additional studies are necessary to determine their roles in AKI.

In addition, the clinical outcomes of patients in non-nephrology wards were worse than those found in the nephrology wards, which may be related to the insidious onset of AKI. Early-stage AKI manifests only as elevated serum creatinine and oliguria or anuria and it is quite easy for non-nephrologists to neglect, thus, delaying RRT initiation. Meanwhile, non-nephrologists may not be able to adjust the dialysis scheme timely and reasonably according to the clinical reality. This calls for the enhancement of communication and cooperation among clinical departments and the improvement of the medical staff's ability to identify AKI.

4.1 | Limitations

This study had limitations. The actual death time of each patient was not considered in the current study and only the survival status was included. At the same time, in this study, 500 patients were included in the matched study population, with a total of 2993 PIRRT sessions. Patient records were made on special dialysis forms and there was no electronic version. The forms were sealed and stored after treatment. Therefore, some information could not be determined, such as the Kt/v. The study subjects were patients with no previous renal disease or CKD 1-3, except patients receiving maintenance RRT, excluding individuals with CKD 4-5. Meanwhile, each haemodialysis scheme of every patient was constantly changing based on the actual situation of the individual. Thus, most patients' single haemodialysis time was not fixed, making it much tougher to divide into subgroups. In addition, this study had all limitations inherent to retrospective studies and randomised controlled trials are needed to confirm the present findings.

5 | CONCLUSION

The effectiveness of PIRRT might be similar to CRRT. Thrombin time, total protein, Na+, number of organs injured, pre-discharge GFR, ward type, infection and PIRRT frequency were associated with 30-day mortality in AKI after PIRRT. Among survivors, pre-discharge GFR and pre-dialysis Scr were associated with renal recovery. For AKI patients with good basal renal function, PIRRT at 3-5 sessions per week may be sufficient.

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Not applicable.

DISCLOSURE

The authors have no conflicts of interest to declare.

DATA AVAILABILITY STATEMENT

Data are available upon reasonable request from corresponding author.

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