Clinical Characteristics and Prognostic Analysis of Pregnancy-Related Acute Kidney Injury

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Abstract

Objective: To investigate the clinical characteristics and prognosis of pregnancy-related acute kidney injury (PR-AKI) and provide a basis for improving maternal and infant outcomes.

Methods: Seventy pregnant women admitted to the surgical intensive care unit of Xinhua Hospital affiliated to Shanghai Jiaotong University School of Medicine from January 2010 to December 2020 were included; 31 were screened out according to KDIGO-AKI criteria. We retrospectively analyzed their clinical characteristics and prognosis and analyzed risk factors for different pregnancy outcomes with logistic regression analysis.

Results: A total of 31 PR-AKI patients were enrolled. The mean age of onset was 30.08 ± 0.63 years, and the mean gestational age was 33.02 ± 7.64 weeks. Six cases (19.45%) were in stage 1, six cases (19.35%) were in stage 2, and 19 cases (61.29%) were in stage 3. The continuous renal replacement therapy (CRRT) group comprised 13 cases (41.94%): one (7.69%) in stage 1, one (7.69%) in stage 2, and 11 (84.62%) in stage 3. The non-CRRT group comprised 18 cases (58.06%): five (27.78%) in stage 1, five (27.78%) in stage 2, and eight (44.44%) in stage 3. The mean time of commencing renal replacement therapy was 2.08 ± 1.26 days after admission, and the serum creatinine (SCr) level at the beginning of treatment was 352.68 ± 196.58 $\mu\text{mol/L}$. Renal function recovered completely in 18 cases (58.06%), comprising four (22.22%) in the CRRT group and 14 (77.78%) in the non-CRRT group, and three cases of partial renal function recovery occurred in the CRRT group. Eventually, seven patients (22.58%) died, of whom four (57.14%) were in the non-CRRT group, and all were in stage 3. The causes of death were postpartum hemorrhage, septic shock, and acute fatty liver during pregnancy. Three patients (42.86%) died in the non-CRRT group: two in stage 3 and one case in stage 1. The causes of death were severe preeclampsia and acute fatty liver during pregnancy. Multi-factor logistic regression analysis showed that gestational weeks ($\text{OR}=0.456$, $P=0.023$), platelet count ($\text{OR}=0.989$, $P=0.02$), hemoglobin ($\text{OR}=1.017$, $P=0.022$), and uric acid ($\text{OR}=1.017$, $P=0.022$) were associated risk factors for maternal adverse pregnancy outcomes of PR-AKI ($P<0.05$).

Conclusions: The incidence of PR-AKI is high, the outcomes of maternal renal function are better, and the proportion of adverse fetal outcomes is higher. CRRT can effectively improve the prognosis of patients with PR-AKI, stabilize the internal environment, and affect hemodynamics slightly. It is currently one of the main ways to treat severe PR-AKI. Maternal and infant outcomes are related to the severity of PR-AKI.

Introduction

Acute kidney injury (AKI) is a critical pregnancy complication needing particular attention. It seriously affects the life and health of the pregnant woman and the fetus. In recent years, due to increased access to maternal and infant healthcare, the incidence of pregnancy-related AKI (PR-AKI) has decreased significantly. Research has reported that the incidence of PR-AKI among pregnant women is around 0.005% in developed countries [1] and 0.81%–11.5% in developing countries [2–6]. However, little

epidemiological data exists on PR-AKI in China. A multi-center study retrospectively analyzed all pregnant women with PR-AKI in 25 hospitals in China from 2013 to 2015. It found that the incidence of PR-AKI was 7.3% [7], indicating that AKI is a common and serious pregnancy complication in China. It not only increases the mortality of pregnant women but also increases the mortality of perinatal infants by 3.4 times [8]. Therefore, early and reasonable diagnosis and treatment of PR-AKI are related to the safety of women and fetuses. Continuous renal replacement therapy (CRRT) has played an essential role in treating AKI, including stabilizing hemodynamics and the internal environment [9], and is the primary treatment for critically ill pregnant women with PR-AKI. According to the KDIGO-AKI criteria, 31 patients with PR-AKI were admitted to the surgical intensive care unit (SICU) of our hospital from January 2010 to December 2020. The etiology of PR-AKI, maternal and infant prognosis, CRRT efficacy, and analysis of risk factors for adverse pregnancy outcomes were retrospectively analyzed to improve awareness of PR-AKI, highlight early diagnosis and treatment, reduce mortality, and improve maternal and infant outcomes.

Materials And Methods

Subjects

Clinical data were collected and analyzed of 31 pregnant women admitted to Xinhua Hospital affiliated with the School of Medicine of Shanghai Jiaotong University from January 2010 to December 2020. The average age was 30.08 ± 0.63 years. Six patients were in the second trimester, with an average gestational age of 23.67 ± 5.00 weeks; 17 patients were in the third trimester, with an average gestational age of 34.38 ± 3.00 weeks; patients cases were in the puerperium, with an average gestational age of 36.72 ± 4.46 weeks. The etiology of PR-AKI was acute fatty liver of pregnancy (AFLP) in eight cases (25.8%); preeclampsia or eclampsia (PE/E) in seven cases (22.6%); infectious or hemorrhagic shock in five cases (16.1%); chronic kidney disease (CKD) in three cases (9.7%); postpartum hemorrhage (PPH), pregnancy with chronic high blood pressure, and other unknown reasons in two cases each (6.4%); and pregnancy complicated with severe infections and ANCA-associated vasculitis in one case each (3.23%). The patients were divided into 13 who received CRRT and 18 who did not. The KDIGO staging criteria released in March 2012 were used to stage PR-AKI [10]. According to the onset time of AKI, the patients were divided into three groups: AKI in the second trimester, AKI in the third trimester, and AKI in the puerperium. The characteristics of the three groups of patients are shown in Table 1.

Related definitions

The definition of AKI followed KDIGO guidelines [10]. Severe AKI was defined as patients receiving renal replacement therapy [2]. PR-AKI was defined as a rapid decline in renal function and accumulation of metabolic waste during pregnancy [11]. CKD was defined following K/DOQI guidelines [12]. Acute exacerbation of CKD (AKI on CKD or A/C) was defined as a reduction in the patient's renal function by 50% from the original level in the short term due to various causes [13]. The KDIGO-AKI staging criteria [10] were used to stage the patients.

Prognostic evaluation

CRRT curative effect: (1) Effective: recovery of renal function, delivery of live baby, improvement and discharge.

(2) Ineffective: Renal replacement therapy still needed after discharge; death or treatment abandonment.

Recovery of renal function: (1) Complete recovery: serum creatinine (SCr) and urine output return to normal range.

(2) Partial recovery: SCr or urine output not fully recovered to normal levels, but renal replacement therapy not required after assessment.

(3) No recovery: SCr not returned to normal or continuing to rise, urine output not returned to normal range, and renal replacement therapy needing to be continued.

Pregnancy outcomes: (1) Good: smooth vaginal delivery or cesarean section, good fetal development, full-term delivery, birth weight ≥ 2500 g, Apgar score 1 minute ≥ 8 points.

(2) Poor: mainly including stillbirth, neonatal death, miscarriage, premature delivery, neonatal asphyxia, neonatal growth restriction, neonatal deformity, precious baby, fetal distress, placental abruption, premature rupture of membranes, birth weight < 2500 g, Apgar score 1 minute < 8 points.

Treatments

Conservative treatment: The patient's condition was evaluated, and the causal treatment was given according to KDIGO-AKI criteria, supplemented by symptomatic support treatment.

CRRT treatment: According to the disease severity, 13 patients were transferred to the ICU for CRRT treatment, using CVVH and CVVHDF modes. We adjusted time and dosage according to the condition. The type and dosage of anticoagulants were determined according to the state of the patients.

Observation indicators: SCr measured before admission was taken as the baseline value for AKI staging. (1) Basic information: age, gestational age, pregnancy number, delivery number, prenatal examination, hospitalization days. (2) Laboratory examination: white blood cell count (WBC), neutrophil %, neutrophil/lymphocyte % (NLR), platelet count (PLT), hemoglobin (HB), SCr, blood urea nitrogen (BUN), alanine aminotransferase (ALT), aspartate transaminase (AST), uric acid, albumin (ALB), Na^+ , K^+ , HCO_3^- , lactic acid, vital signs (blood pressure, heart rate, respiration, blood oxygen saturation) were measured every 1 hour during CRRT treatment. (3) Maternal and infant outcomes.

Data analysis

SPSS 23.0 software was used for statistical analysis. Normally distributed measurement data is represented by the mean $\pm$ standard deviation, and comparison between groups used the t-test. Non-

normal distribution measurement data is represented by the median and interquartile range, and comparison between groups used the Wilcoxon rank-sum test. Count data used the χ^2 test. Single-factor logistic regression analysis was used for risk factor analysis to screen related factors. The variables with significant differences in single-factor analysis were analyzed by stepwise multivariate logistic regression analysis. $P<0.05$ was considered statistically significant, and $P<0.01$ was considered statistically very significant.

Table 1 Basic information of 31 patients with PR-AKI

	All patients n=31	Second trimester n=6	Third trimester n=17	Puerperium n=8
Age (years)	29.16±4.97	31.33±7.94	27.41±3.78	31.25±3.45
Gestational age (weeks)	32.91±5.98	23.67±5.00	34.38±3.00	36.72±4.46
Cesarean section (%)	26 (83.87)	6 (23.08)	16 (73.08)	5 (19.23)
Primipara (%)	18 (58.06)	4 (22.22)	10 (55.56)	4 (22.22)
Twin pregnancy (%)	5 (16.13)	0	4 (80.00)	1 (20.00)
Number of pregnancies	1.94±1.24	2.50±1.87	1.76±1.03	1.88±1.13
Number of deliveries	1.23±0.56	1.33±0.52	1.24±0.66	1.13±0.35
Termination weeks				
≥37 weeks (%)	11 (35.48)	0	5 (45.45)	6 (54.55)
28 to <37 weeks (%)	14 (45.16)	1 (7.14)	11 (78.57)	2 (14.29)
12 to <28 weeks (%)	5 (16.13)	5	0	0
Untermiated (%)	1 (3.23)	0	1	0
In-hospital days	24.61±17.55	27.00±25.61	27.35±17.09	17.00±9.71
In-ICU days	12.03±10.13	7.00±5.69	14.00±11.33	11.63±9.59
APACHE II scores	12.77±7.30	10.50±6.66	10.88±5.73	18.50±8.44
AKI stage				
AKI stage 1 (%)	6 (19.40)	2 (33.33)	3 (50.00)	1 (16.67)
AKI stage 2 (%)	6 (19.40)	2 (33.33)	3 (50.00)	1 (16.67)
AKI stage 3 (%)	19 (61.29)	2 (10.53)	11 (84.62)	6 (31.58)
Obstetric complications				
PE/E (%)	8 (25.81)	2 (33.33)	4 (23.53)	2 (25.00)
PPH (%)	2 (6.45)	0	1 (5.88)	1 (12.50)
Shock (%)	5 (16.13)	1 (16.67)	1 (5.88)	3 (37.50)
AFLP (%)	7 (22.58)	0	5 (29.41)	2 (25.00)
CKD (%)	3 (9.68)	2 (33.33)	1 (5.88)	0

Results

Maternal outcomes

Of the 31 patients with PR-AKI,13 were in the CRRT group, including five cases of AFLP (38%) and three cases of septic or hemorrhagic shock (23%). The remaining 18 patients received non-replacement therapy (39%), including seven cases of PE/E (39%), three cases of AFLP (16%), and three cases of septic shock (16%), as shown in Figure 1. In the CRRT group, four patients (31%) had complete recovery of renal function, three (23%) had partial recovery of renal function but ended substitution therapy, and four (31%) died. In the group with complete recovery of renal function, the average CRRT included six treatment times, and the average cumulative treatment duration was 61.54 hours. In the group with partial recovery of renal function, the average CRRT included five treatment times, and the average cumulative treatment duration was 51.5 hours. Compared with the partially recovered renal function group, the complete recovery group had more CRRT treatment times, longer cumulative treatment durations, and better maternal and infant outcomes.

Among the 18 cases in the non-CRRT group, 13 had complete renal function recovery, one received renal replacement therapy, one did not recover renal function and delivered a stillbirth, and three died. In the CRRT group, WBC, neutrophil %, NLR, AST, AST, serum uric acid, and SCr were significantly lower than before CRRT treatments ($P<0.05$). Lactic acid, BUN, albumin, Na^+ , and K^+ were lower than before CRRT treatments, but with no statistical difference, as shown in Table 2.

Among the 18 cases in the non-CRRT group, 13 had complete renal function recovery, one received renal replacement therapy, one did not recover renal function and delivered a stillbirth, and three died. In the CRRT group, WBC, neutrophil %, NLR, AST, AST, serum uric acid, and SCr were significantly lower than before CRRT treatments ($P<0.05$). Lactic acid, BUN, albumin, Na^+ , and K^+ were lower than before CRRT treatments, but with no statistical difference, as shown in Table 2.

Table 2 Comparison of laboratory examinations before and after CRRT treatment

	Before treatment	After treatment	T/Z value	P-value
WBC ($\times 10^9/L$)	22.10 $\pm$ 6.27	9.70 $\pm$ 7.04	4.009	0.002
N %	91.17 $\pm$ 4.64	61.98 $\pm$ 20.31	4.987	0.000
NLR	3.92 (4.50)	4.80 (7.93)	-3.667	0.000
BUN (mmol/L)	16.41 $\pm$ 11.45	11.65 $\pm$ 9.38	1.233	0.241
SCr (μ mol/L)	363.25 $\pm$ 202.99	177.85 $\pm$ 149.85	4.061	0.002
Uric acid (mmol/L)	522.21 $\pm$ 133.05	247.05 $\pm$ 136.85	4.940	0.000
ALT (U/L)	70.00 (89.00)	29.00 (31.00)	-2.515	0.012
AST (U/L)	96.00 (79.50)	32.00 (67.00)	-2.130	0.033
ALB (g/L)	29.93 $\pm$ 11.87	31.11 $\pm$ 8.01	-1.725	0.110
Na ⁺ (mmol/L)	133.38 $\pm$ 7.41	139.29 $\pm$ 5.86	-2.284	0.041
K ⁺ (mmol/L)	4.52 $\pm$ 0.90	4.01 $\pm$ 0.54	2.115	0.056
Lactic acid (mmol/L)	4.80 (7.93)	1.85 (5.68)	-1.421	0.155
PLT ($\times 10^9/L$)	85.54 $\pm$ 52.06	206.08 $\pm$ 111.09	-3.154	0.008

Fetal outcomes

Fetal outcomes are summarized in Table 3. The 31 patients with PR-AKI delivered 35 newborns, including 25 parturients (83.33%) with a single pregnancy, 5 parturients (16.67%) with a twin pregnancy, and 26 cesarean sections (83.9%). Among the newborns, eight (22.86%) were full-term, 17 (48.57%) were premature, and one (2.86%) was post-term. Five died, with a mortality rate of 14.26%, and 22 were transferred to NICU (62.86%). Seven newborns had a birth weight ≥ 2500 g (20.00%), 10 had a birth weight ≥ 1500 g but < 2500 g (28.57%), and eight had a birth weight < 1500 g (22.86%). Among the three groups, the best pregnancy outcomes were in the puerperium group, and the worst were in the second trimester group ($P < 0.05$). These results are consistent with those reported in the literature [2].

Table 3 Fetal outcomes

Outcomes	Number	Second trimester	Third trimester	Puerperium
Infants (%)	35	6 (17.14)	20 (57.14)	9 (25.71)
Term infants (%)	8 (22.86)	0	4 (50.00)	4 (50.00)
Pre-term infants (%)	17 (48.57)	3 (17.65)	10 (58.82)	4 (23.53)
Post-term infants (%)	1 (2.86)	0	0	1 (100.00)
Stillbirths (%)	5 (14.29)	3 (60.00)	2 (40.00)	0
Severe asphyxia (%)	5 (14.29)	1 (20.00)	1 (20.00)	3 (60.00)
Birth weight, g				
≥2500 (%)	7 (20.00)	0	4 (57.14)	3 (42.86)
≥1500 but <2500 (%)	10 (28.57)	0	9 (90.00)	1 (10.00)
<1500 (%)	8 (22.86)	3 (37.50)	5 (62.50)	0
Apgar scores				
1 minute	6.48±3.24	5.53±1.16	7.00±3.31	5.50±3.73
5 minutes	7.85±2.63	6.67±1.53	8.06±2.90	7.83±2.32
10 minutes	9.00±2.08	6.67±1.53	9.19±2.58	9.67±0.82
Transfer to NICU (%)	22 (62.86)	3 (13.64)	15 (68.18)	4 (18.19)
Loss to follow-up (%)	3 (8.57)	0	0	3 (100.00)

Characteristics of the CRRT group

A total of 13 patients with PR-AKI received CRRT treatment. The mean timing of the CRRT intervention was 1.92 ± 1.12 days after admission, the mean number of treatments was 7.09 ± 7.39 , and the mean cumulative treatment time was 63.31 ± 45.43 hours. The main causes were AFLP in five cases (38.46%), infectious or hemorrhagic shock in three cases (23.08%), severe preeclampsia in one case (7.69%), and postpartum hemorrhage in one case (7.69%); two cases were A/C. Among the 13 patients was one with AKI stage 1, where the primary disease was chronic glomerulonephritis, who received CRRT treatments 29 times and long-term renal replacement therapy after delivery. The infant had a poor prognosis due to low birth weight with severe asphyxia. In the one case of AKI stage 2, with a main cause of Alphard with twin pregnancy, CRRT treatments were more frequent, and the commencement time of CRRT was earlier. As expected, the prognosis was good, and the newborns survived. In the 11 cases of AKI stage 3, three partially recovered renal function, two progressed to end-stage renal disease, and four died; three newborns did not survive.

Analysis of adverse pregnancy outcomes and risk factors in PR-AKI patients

Among the 31 patients with PR-AKI, 25 (80.6%) had poor fetal outcomes, including five stillbirths, five cases of neonatal asphyxia, 18 premature births, 10 low birth weight infants, and eight deficient birth weight infants. Six patients had good fetal outcomes. The gestational age, platelet count, and hemoglobin of patients with good fetal outcomes were significantly higher than those with poor fetal outcomes (all $P<0.05$). BUN and uric acid were significantly lower than those with poor fetal outcomes (all $P<0.05$). No significant difference existed between the age, the number of pregnancies and deliveries, NLR, ALT, AST, albumin, SCr, and the composition ratio of CKD patients (all $P>0.05$), as shown in Table 4.

Table 4 Comparison of PR-AKI patients with different fetal outcomes

	Good outcomes n=6	Poor outcomes n=25
Age ($\bar{x}\pm s$, years)	28.17 $\pm$ 2.64	29.4 $\pm$ 5.40
Gestational weeks ($\bar{x}\pm s$, weeks)	39.26 $\pm$ 1.53 [□]	31.62 $\pm$ 5.50
Number of pregnancies ($\bar{x}\pm s$, times)	2.00 $\pm$ 1.26	1.88 $\pm$ 1.27
Number of deliveries ($\bar{x}\pm s$, times)	1.33 $\pm$ 0.52	1.20 $\pm$ 0.58
NLR ($\bar{x}\pm s$)	12.15 $\pm$ 4.19	27.07 $\pm$ 20.70
Platelet count ($\bar{x}\pm s$, $\times 10^9/L$)	217.13 $\pm$ 122.87 [□]	90.24 $\pm$ 84.88
Hemoglobin ($\bar{x}\pm s$, g/L)	94.19 $\pm$ 13.21 [□]	74.48 $\pm$ 20.78
ALT ($\bar{x}\pm s$, U/L)	34.22 (153.10)	68.00 (116.00)
AST ($\bar{x}\pm s$, U/L)	44.00 (313.26)	102.10 (165.50)
Albumin ($\bar{x}\pm s$, g/L)	33.75 $\pm$ 1.47	27.99 $\pm$ 12.66
BUN ($\bar{x}\pm s$, mmol/L)	11.87 $\pm$ 4.28 [□]	19.47 $\pm$ 10.98
SCr ($\bar{x}\pm s$, $\mu\text{mol/L}$)	178.94 $\pm$ 137.46	294.02 $\pm$ 211.54
Uric acid ($\bar{x}\pm s$, $\mu\text{mol/L}$)	262.41 $\pm$ 167.00 [□]	586.87 $\pm$ 144.52
CKD (n, %)	2 (33.33%)	1 (4.00%)
AKI Stage (n, %)		
Stage 1	1 (16.67%)	11 (44.00%)
Stage 2	1 (16.67%)	3 (12.00%)
Stage 3	4 (66.67%)	11 (44.00%)

☒ Compared with poor pregnancy outcomes: $P < 0.01$.

Logistic regression analysis was performed on these factors. Gestational age (OR=0.456, $P=0.023$), platelet count (OR=0.989, $P=0.02$), hemoglobin (OR=1.017, $P=0.022$), and uric acid (OR=1.017, $P=0.022$) were risk factors for poor fetal outcomes in PR-AKI patients (Table 5). Age, number of pregnancies, parity, NLR, ALT, AST, plasma, albumin, SCr, and CKD did not affect pregnancy outcomes ($P > 0.05$).

Table 5 Logistic regression analysis of risk factors for adverse fetal outcomes

Variables	Regression coefficients	Standard error	Wald	P-value	OR
Gestational weeks	-0.786	0.345	5.204	0.023	0.456
PLT	-0.011	0.005	5.421	0.020	0.989
HB	-0.050	0.027	3.573	0.059	0.951
Uric acid	0.017	0.007	5.241	0.022	1.017

Discussion

Significant changes occur in renal hemodynamics, renal tubules, and endocrine function during pregnancy. The mechanism of PR-AKI is multifaceted. During pregnancy, due to the significant increase in cardiac output, renal blood flow in the second trimester can increase by around 85%, and glomerular filtration rate (GFR) can increase by 50%, leading to significantly high filtration in the second trimester. Late GFR decreased by about 20% and returned to average around 3 months after delivery. The excretion of metabolites such as BUN and SCr increased, and the concentration of SCr decreased to 44 $\mu\text{mol/L}$. Feng et al. [14] reported that SCr over 70 $\mu\text{mol/L}$ was considered abnormal. In our study, the diagnosis of PR-AKI was mainly based on KDIGO-AKI criteria, all of which are in line with $\text{SCr} \geq 70 \mu\text{mol/L}$. Due to the increase in coagulation factors and fibrinogen during pregnancy, the blood is in a hypercoagulable state, prone to microvascular thrombosis and disseminated intravascular coagulation (DIC), which is an essential pathophysiological basis for acute renal injury [15]. Among the pregnant women with PR-AKI in our study, the CRRT group included five cases of secondary DIC, which promoted the occurrence or aggravated the severity of AKI. Three patients in the non-CRRT treatment group, combined with DIC, had a prolonged course of treatment, and their pregnancy outcomes were also affected to varying degrees. During pregnancy, due to the influence of estrogen and progesterone, prostaglandin E2 has effects such as inhibiting ureteral peristalsis and dextrorotation of the uterus on the right ureter. The right side of the ureter is severely hydronephrotic [14]. This physiological change increases the probability of urinary tract infections and promotes the progression of PR-AKI.

In developing countries, the leading causes of PR-AKI include infectious abortion, postpartum sepsis, PE, and PPH [16–18]. In developed countries, they are PE/E, hemolysis–liver elevated enzymes–low platelet (HELLP) syndrome, thrombotic microangiopathy (TMA), placental abruption, and AFLP [19]. AFLP is a critical and severe obstetric complication. According to the literature, the incidence of PR-AKI caused by

AFLP is 2–4.3% [20]. In our study, AFLP accounted for 25.8% of cases, and PE/E accounted for 22.6%, significantly lower than in foreign research [21]. A possible reason is that more pregnant women with severe AKI were included. As the leading cause of PR-AKI, PE is worthy of attention. In recent years, its risk factors have been more common, such as increased maternal age, diabetes, multiple births, and heart failure [22]. Duley [23] reported that PE seriously affects pregnant women; 8% of pregnancies develop PE, which carries threats in terms of maternal mortality and neonate survival. Shock is another crucial cause of PR-AKI. In our study, PR-AKI caused by shock accounted for 16.1% of cases, similar to rates reported before [24]. Prakash et al. [3] conducted a study of 85 patients with AKI in the third trimester and puerperium and found that PE, HELLP syndrome, AFLP, prenatal and postpartum hemorrhage, and puerperal infection were the leading causes. However, notably, the etiology of PR-AKI is complex, and multiple factors often act synergistically [25].

Comprehensive treatment of PR-AKI is crucial, including three aspects: active treatment of the primary disease, symptomatic and supportive treatment, and renal replacement therapy. Dialysis treatment is essential in the clinic. Dialysis indications mainly include hyperkalemia, volume overload, uremic encephalopathy, and acidosis. Compared with IHD, CRRT has the advantages of high solute clearance, less impact on patient hemodynamics, and being conducive to managing water and electrolytes. It is currently the main way to treat AKI with severe comorbidities and is widely used in critically ill patients [26–27]. In our study, 13 (41.9%) pregnant women underwent CRRT treatment; nine (69.2%) had improved renal function. Eighteen (58.1%) were treated conservatively; 15 (83.3%) had improved renal function, and three (16.7%) died. Foreign studies have reported that the rate of renal replacement therapy in patients with PR-AKI reached 70–100% [28–29]. The main reason for the low rate of replacement therapy in our study was that the proportion of pregnant women with AKI stage 1–2 was relatively large.

As long as patients with PR-AKI are given timely, effective, and correct treatment, most can recover their renal function [30], and the return of renal function is reasonable. In this study, renal function improved in 20 cases, and the effective rate of treatment on PR-AKI was 64.5%. Seventeen patients recovered completely, with a PR-AKI cure rate of 54.8%, similar to that reported in a previous study [31]. Seven patients died, giving a mortality rate of 22.6%, higher than reported in the research (11.4–16.2%) [32–34]. The reason is that the pregnant women included in our study were all critically ill patients in the SICU with a severe underlying condition. The rates of renal function recovery in AKI stages 1, 2, and 3 were 83.3%, 66.7%, and 63.2%, respectively, indicating that the maternal prognosis of PR-AKI is related to the stage. The 31 PR-AKI patients gave birth to 35 newborns; the neonatal survival rate was 74.3%, and the proportion of adverse outcomes was relatively high. Among the infants were 17 premature babies (48.6%), 10 low birth weight infants (28.6%), eight deficient birth weight infants (22.9%), and five stillbirths (14.3%, lower than the 29.8% reported in domestic studies) [8]. The reason may be the gradual improvement of obstetric care and maternal and infant healthcare technology.

The pregnancy outcomes of PR-AKI are related to multiple factors. Logistic regression analysis showed that gestational age, platelet count, hemoglobin, and uric acid were risk factors for poor pregnancy

outcomes in patients with PR-AKI, indicating that the second trimester of pregnancy, low platelet counts, anemia, and high uric acid increased the occurrence of adverse pregnancy outcomes.

Conclusion

In summary, PR-AKI is a severe complication of pregnancy, with a high incidence and a high proportion of adverse maternal and infant outcomes. Timely and effective renal replacement therapy can improve the prognosis of pregnant women, and its timing needs to be further studied. Renal function and maternal and infant outcomes are related to the severity of PR-AKI. Therefore, pregnancy monitoring and early intervention must be strengthened to improve maternal and infant outcomes.

Abbreviations

KDIGO Kidney Disease: Improving Global Outcomes

K/DOQI Kidney Disease Outcomes Quality Initiative

NICU Neonatal intensive care center

CVVH Continuous veno - venous hemofiltration

CVVHDF Continuous veno - venous hemodiafiltration

Declarations

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Availability of data and materials

The datasets used and analysed during the current study available from the corresponding author on reasonable request.

Authors' contributions

HMJ and LW carried out the retrospective review of the data, participated in the design, writing and organization of the manuscript. LW, HHD and SJF conceived of the study and designed of it. HMJ, LFJ and DXT participated in the collect and analysis data of the case.

All authors read and approved the final manuscript.

Ethics approval and consent to participate

This research study was conducted retrospectively from data obtained for clinical purposes. We consulted with the Bioethics Committee of Xinhua Hospital affiliated to Shanghai Jiaotong University School of Medicine who determined that our study did not need ethical approval. A Bioethics Committee's official statement of ethical approval was granted from the Xinhua Hospital affiliated to Shanghai Jiaotong University School of Medicine. The study was conducted according to the principles of the Declaration of Helsinki and its amendments. Informed consent was obtained from all subjects.

Consent for publication

Not applicable.

Competing interests

None of the authors have any competing interest to declare.

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Figures

Figure 1

Comparison of etiology between CRRT and non-CRRT groups