ORIGINAL PAPER

Nagoya J. Med. Sci. **88**. 477–486, 2026
doi:10.18999/nagjms.88.3.477

Analysis of risk factors for acute kidney injury during extracorporeal membrane oxygenation in children

Jiaqi Li[1,2], Liting Hou[1,2], Qianqian Liu[1,2], Bin Yan[2] and Weikai Wang[2]

[1]*Department of The First Clinical Medical College, Gansu University of Chinese Medicine, Lanzhou, China*
[2]*The Second Department of the PICU, Gansu Maternal and Child-Care Hospital, Lanzhou, China*

ABSTRACT

This study aimed to identify the risk factors for acute kidney injury (AKI) in children receiving extracorporeal membrane oxygenation (ECMO) treatment. We retrospectively analyzed data of 76 children who were treated with ECMO. The analysis factors included sex, age, primary disease type, ECMO treatment mode and flow, the vasoactive–inotropic score (VIS) after ECMO initiation, and relevant laboratory data before and after ECMO initiation. Patients who developed AKI were assigned to the observation group, while those who did not develop AKI were assigned to the control group. We compared relevant indicators between the two groups. Binary logistic regression was used to determine independent risk factors for AKI during ECMO treatment. AKI occurred in 43 patients (56.6%). Univariate analysis revealed significant differences in the intensive care unit (ICU) stay length, mechanical ventilation duration, combined continuous renal replacement therapy (CRRT) time, ECMO treatment duration, lactate level before ECMO initiation, VIS after ECMO initiation, and output within 24 hours after ECMO initiation. Multivariate analysis revealed that high lactate levels before ECMO initiation (OR = 1.828; 95% CI, 1.163–2.874; P = 0.009), a high VIS after ECMO initiation (OR = 1.038; 95% CI, 1.000–1.077; P = 0.047), and a prolonged ECMO duration (OR = 1.022; 95% CI, 1.005–1.039; P = 0.012) were independent risk factors for AKI. The incidence of AKI during ECMO treatment is high. A high VIS, an elevated lactate level before ECMO initiation, and an extended ECMO duration are independent risk factors for AKI in these patients.

Keywords: children, extracorporeal membrane oxygenation, acute kidney injury, risk factors

INTRODUCTION

Extracorporeal membrane oxygenation (ECMO) is an advanced form of extracorporeal cardiopulmonary circulation that is used for life support when treating conditions such as fulminant myocarditis, acute respiratory distress syndrome, and cardiopulmonary decompensation from primary diseases in other systems.[1-3] The primary function of ECMO is to mimic cardiopulmonary circulation outside the body, allowing time for the treatment of the child's primary condition or serving as a bridge to cardiopulmonary surgery. ECMO ensures effective blood perfusion and

Received: March 11, 2026; Accepted: April 3, 2026
Corresponding Author: Weikai Wang, MD
The Second Department of the PICU, Gansu Maternal and Child-Care Hospital,
No. 999, Mogao Avenue, Anning District, Lanzhou 730050, China
E-mail: wweikaiwwpicu@outlook.com

oxygen supply to tissues and organs. However, complications can occur, including elevated liver enzyme levels from liver damage, hemoglobinuria from kidney damage, and gastrointestinal bleeding due to impaired digestive function. Acute kidney injury (AKI) is a prevalent complication of ECMO and significantly affects patients' quality of life. A study reported that the incidence of AKI post-ECMO was 64.8% in 68 neonates and 61.4% in 105 children.[4] The mortality rate for AKI can exceed 50%.[5] Some studies suggest that the vasoactive–inotropic score (VIS) may predict AKI in infants undergoing ECMO,[6] although these studies often have small sample sizes. This study aimed to identify independent predictors of ECMO-related AKI in children by increasing the sample size and systematically including potential related factors, such as the VIS and blood lactate level, to provide a reference for clinical prevention and treatment.

MATERIALS AND METHODS

Participants

In this study, we retrospectively collected data of 98 patients who initially underwent ECMO at the Gansu Provincial Maternity and Child-Care Hospital and Xi'an Children's Hospital in Shanxi Province between September 2020 and September 2025. Ultimately, 76 patients (36 males and 40 females) were included in the study, while 22 patients were excluded. The exclusion criteria were an ECMO treatment duration of less than 24 hours (n = 7), preexisting AKI before ECMO initiation (n = 6), and incomplete clinical data (n = 9). The inclusion criteria were as follows: (1) admission to the intensive care units (ICUs) of the two hospitals for ECMO treatment from September 2020 to September 2025; (2) age under 18 years; and (3) ECMO treatment lasting more than 24 hours, with complete clinical data. The exclusion criteria included the following: (1) a history of AKI before ECMO treatment and (2) a history of kidney-related diseases (such as renal tumors, kidney stones, and postrenal oliguria). On the basis of the AKI clinical guidelines from the Kidney Disease: Improving Global Outcomes (KDIGO),[7] the patients were categorized into non-AKI and AKI groups. AKI was diagnosed if any of the following conditions were met: an increase in the serum creatinine concentration by ≥ 26.5 μmol/L (0.3 mg/dL) within 48 hours, a urine output of less than 0.5 mL/kg/hour for 6 consecutive hours, or a known or presumed increase in the serum creatinine concentration to more than 1.5 times the baseline value within 7 days. This article establishes 48 hours post-initiation of ECMO as a critical time point for assessing AKI in children, thereby excluding patients with AKI who fall outside this designated milestone. This decision is informed by several clinical and methodological considerations: (1) The 48-hour mark post-initiation of ECMO represents a crucial period for hemodynamic adaptation, the activation of inflammatory responses, and the opportunity for early intervention; (2) This time frame has been extensively utilized in prior studies examining AKI associated with ECMO, thereby enhancing the comparability of results; (3) Within the retrospective data, the records of all indicators during this time window are the most comprehensive.

This study was conducted in accordance with Declaration of Helsinki. This research study was conducted retrospectively from data obtained for clinical purposes. The Ethics Committee of the Gansu Provincial Maternal and Child Health Hospital has reviewed and approved this research (file no. 2026 GSFY Lunshen 002).

Clinical data

Upon admission to the ICU, comprehensive clinical data were gathered for each patient. These data included sex, age (in days), body mass index (BMI [kg/m^2]), type of primary disease, length of the ICU stay (in days), duration of mechanical ventilation (in hours), ECMO mode

and initial flow, duration of ECMO (in hours), and fluid intake and output within 24 hours post-ECMO initiation, including the fluid overload percentage (FO%).[8] Additionally, the duration of combined continuous renal replacement therapy (CRRT) (in hours) and the VIS following ECMO initiation were recorded. The study by Gaies et al[9] provided coefficients to integrate all vasoactive drugs, allowing for the calculation of the final VIS during ECMO treatment for each patient. Laboratory data primarily included results of routine blood tests (such as white blood cell counts, hemoglobin levels, and platelet counts), blood biochemistry (including the levels of albumin, transaminases, blood urea nitrogen, and procalcitonin), blood gas analysis (covering the pH value and the levels of various ions and lactic acid) within 24 hours prior to ECMO initiation, and the left ventricular ejection fraction of the heart.

Statistical methods

Statistical analyses were conducted using SPSS 25.0 software (IBM). Data with a nonnormal distribution are presented as the median and interquartile range (M [Q1, Q3]), whereas data with a normal distribution are expressed as the mean ± standard deviation ($\bar{X} \pm s$). For comparisons between the two groups, an independent sample *t* test was employed for normally distributed data, and a nonparametric rank sum test was used for nonnormally distributed data. Count data are presented as the number of cases (n), and comparisons between groups were made by using the χ undefined test. Variables with a *P* value less than 0.05 in the univariate analysis were included in the multivariate logistic regression analysis. The forward likelihood ratio method or enter method was utilized for variable selection, and the odds ratio (OR) and its 95% confidence interval (CI) were calculated. A difference was deemed statistically significant when *P* was less than 0.05.

RESULTS

Incidence of AKI in ECMO patients and univariate analysis

The study ultimately included 76 patients, 43 (56.6%) of whom developed AKI. The primary infection sites among the children receiving ECMO-assisted therapy were predominantly the cardiovascular system, affecting 42 cases (55.2%). This group included 34 cases of fulminant myocarditis and 8 cases of congenital heart disease. The respiratory system was involved in 28 cases (36.8%), comprising 26 cases of acute respiratory distress syndrome complicated by severe pneumonia and 2 cases of bilateral pulmonary contusion and laceration. Additionally, there were 6 cases (8%) in other systems, which encompassed the digestive, blood, and nervous systems, with conditions such as pancreatitis, typhus, myelitis, leukemia, and sepsis.

Baseline characteristics prior to ECMO initiation indicated no statistically significant differences in age, sex, weight, or underlying diseases between the two patient groups ($P > 0.05$). Laboratory data collected within 24 hours before ECMO initiation revealed that the blood lactate levels in the AKI group were significantly higher than those in the non-AKI group ($P < 0.05$). However, no statistically significant differences were observed in other indicators, including white blood cells, red blood cells, platelets, albumin, and bilirubin ($P > 0.05$). Early exposure following the initiation of ECMO revealed that within 24 hours, the output of patients in the AKI group was significantly lower than that of the non-AKI group ($P < 0.05$). However, no statistically significant differences were observed between the two groups regarding ECMO flow and patterns ($P > 0.05$). A comparison of clinical data from ECMO initiation to weaning indicated that the mechanical ventilation duration, combined CRRT treatment duration, ECMO treatment duration, and VIS for patients in the AKI group were all significantly higher than those in the non-AKI group ($P < 0.05$), as detailed in Tables 1, 2-1, 2-2 and 2-3.

Table 1 Comparison of baseline data between AKI and non-AKI groups

Parameter	AKI Group (n = 43)	Non-AKI Group (n = 33)	T/Z/χ^2	*P*
Age (days)	2610 (690–3960)	2880 (1125–3585)	–0.079	0.940
BMI (kg/m^2)	16.74 (14.80–18.40)	15.70 (15.04–18.19)	–0.576	0.568
Sex, n (%)				
Male/female	21 (48.8)/22 (51.2)	15 (45.5)/18 (54.5)	0.068	0.770
Type of primary disease, n (%)				
Heart	22 (51.2)	20 (60.6)	3.071	0.215
Lung	19 (44.2)	9 (27.3)		
Others (digestive tract, blood, nervous system)	2 (4.6)	4 (12.1)		

AKI: acute kidney injury
BMI: body mass index

Table 2-1 Comparison of laboratory data between the two groups within 24 hours before the initiation of ECMO

Parameter	AKI Group (n = 43)	Non-AKI Group (n = 33)	T/Z	*P*
Systolic blood pressure (mmHg)	90.370 ± 25.294	90.550 ± 22.192	0.031	0.975
Diastolic blood pressure (mmHg)	56.930 ± 16.775	54.910 ± 11.223	–0.628	0.532
Mean arterial pressure (mmHg)	67.810 ± 18.316	66.450 ± 13.079	–0.361	0.719
pH	7.350 (7.297–7.440)	7.370 (7.285–7.425)	–0.320	0.752
PO_2 (mmHg)	81.6 (64–128)	97 (69–165)	–1.289	0.199
PCO_2 (mmHg)	37 (26–46)	34.9 (23.6–48.5)	–0.765	0.448
Na^+ (mmol/L)	140.495 ± 6.274	140.621 ± 4.804	0.096	0.924
Lac (mmol/L)	4.3 (3.3–6.6)	2.20 (1.55–3.45)	–4.492	<0.001
HCO_3^- (mmol/L)	20.0 (15.1–24.9)	17.90 (13.30–24.85)	–0.990	0.325
K^+ (mmol/L)	3.98 (3.67–4.51)	3.87 (3.58–4.185)	–1.116	0.264
WBCs ($\times 10^9$/L)	8.41 (5.50–13.33)	10.46 (6.78–15.63)	–1.472	0.142
RBCs ($\times 10^{12}$/L)	4.0230 ± 0.8241	4.0706 ± 0.8140	0.251	0.803
HB (g/L)	116.020 ± 22.453	116.330 ± 21.489	0.061	0.952
PLTs ($\times 10^9$/L)	225 (106–294)	168 (126–217.5)	–1.174	0.243
PCT (ng/mL)	0.71 (0.13–10.67)	0.500 (0.120–19.765)	–0.341	0.737
Albumin (g/L)	35.3950 ± 7.0068	37.1640 ± 6.8371	1.102	0.274
ALT (U/L)	51 (24–151)	29 (18.5–54.8)	–1.630	0.104
AST (U/L)	121.3 (62–351)	75.5 (39.5–203.5)	–1.305	0.194
Total bilirubin (μmol/L)	13.2 (9.4–20.7)	16.3 (9.65–33.2)	–0.938	0.352
LDH (U/L)	670 (375–1247)	449 (266.5–866.5)	–1.63	0.104
CK-MB (U/L)	53 (20.5–142)	55 (18.5–93.15)	–0.603	0.551
BUN (mmol/L)	5.48 (3.97–9.48)	5.58 (4.57–9.545)	–0.891	0.377

ECMO: extracorporeal membrane oxygenation
AKI: acute kidney injury

Lac: lactic acid
HCO3-: hydrogen carbonate
WBC: white blood cell
RBC: red blood cell
HB: hemoglobin
PLT: platelet
PCT: procalcitonin
ALT: alanine transaminase
AST: aspartate aminotransferase
LDH: lactate dehydrogenase
CK-MB: creatine kinase-MB isoenzyme
BUN: blood urea nitrogen

Table 2-2 Comparison of clinical data between the two groups within 24 hours after ECMO initiation

Parameter	AKI Group (n = 43)	Non-AKI Group (n = 33)	T/Z/χ^2	*P*
24-h intake after initiation (mL)	1741.764 ± 887.477	1925.530 ± 1034.045	0.833	0.408
24-h output volume after initiation (mL)	828 (324–1481)	1296 (720.5–1952)	–2.143	0.032
24-day FO (%) after initiation	31.22 (15.60–67.61)	22.39 (12.01–48.09)	–1.420	0.158
ECMO mode, n (%)				
V-V/V-A	16 (37.2)/27 (62.8)	9 (27.3)/24 (72.7)	0.835	0.361
ECMO initial flow (L/min)	1.56 (1.09–2.50)	1.700 (1.055–2.225)	–0.037	0.973

ECMO: extracorporeal membrane oxygenation
AKI: acute kidney injury
V-V: veno-venous
V-A: veno-arterial
ICU: intensive care unit
Fluid overload percentage (FO%) = Σ[daily intake (mL) − daily output (mL)]/body weight at ICU admission (kg) × 100%.

Table 2-3 Comparison of clinical data between the two groups from the initiation to the weaning of ECMO

Parameter	AKI Group (n = 43)	Non-AKI Group (n = 33)	T/Z/χ^2	*P*
Duration of ECMO (h)	219.810 ± 85.001	139.060 ± 48.907	–5.207	<0.001
LVEF (%)	48.3510 ± 24.5121	46.0830 ± 19.4696	–0.436	0.664
Duration of mechanical ventilation (h)	255 (192–360)	192 (94.5–253)	–1.991	0.046
CRRT use time (h)	207 (66–305)	134 (18.5–191)	–2.477	0.013
Vasoactive drugs (types)	2 (1–4)	2 (1–4)	–0.368	0.716
VIS	35.6 (23.0–79.2)	22.00 (14.80–33.25)	–3.511	<0.001
Other organ damages (cases)	4.910 ± 1.716	4.390 ± 1.298	–1.431	0.157
Clinical outcome, n (%)				
Survival/death	32 (74.4)/11 (25.6)	30 (90.9)/3 (9.1)	3.379	0.066

ECMO: extracorporeal membrane oxygenation
AKI: acute kidney injury

LVEF: left ventricular ejection fraction
CRRT: continuous renal replacement therapy
Vasopressor–inotrope score (VIS) = dopamine [μg/(kg·min)] + dobutamine [μg/(kg·min)] + 10 × milrinone [μg/(kg·min)] + 100 × epinephrine [μg/(kg·min)] + 100 × norepinephrine [μg/(kg·min)] + 10000 × vasopressin [μg/(kg·min)].

Multivariate analysis between the AKI group and the non-AKI group

Variables showing statistically significant differences in the univariate analysis were further analyzed via binary logistic regression. The results indicated that the ECMO treatment duration, VIS, and blood lactate levels prior to ECMO initiation were independent related factors for developing AKI during ECMO treatment (OR > 1, $P < 0.05$). In contrast, multivariate analysis revealed that an increase in the output volume within 24 hours post-ECMO initiation was linked to a reduced risk of AKI (OR = 0.999). However, the OR value was nearly 1, and the lower limit of the 95% confidence interval (0.998) was also close to 1, suggesting that the effect size of this association might be very weak (Table 3).

Table 3 Multivariate logistic analysis

Risk factors	B	Wald	OR	95% CI	*P*
Duration of ECMO (h)	0.022	6.369	1.022	1.005–1.039	0.012
VIS	0.037	3.944	1.038	1–1.077	0.047
24-h output volume after initiation (mL)	–0.001	4.266	0.999	0.998–1	0.039
Lac before initiation (mmol/L)	0.603	6.832	1.828	1.163–2.874	0.009
Duration of mechanical ventilation (h)	–0.002	0.533	0.998	0.992–1.004	0.465
CRRT use time (h)	0.002	0.234	1.002	0.995–1.009	0.628

ECMO: extracorporeal membrane oxygenation
VIS: vasopressor–inotrope score
CRRT: continuous renal replacement therapy

DISCUSSION

ECMO is considered a crucial alternative treatment for patients with critical and severe illnesses, as it can improve survival outcomes. However, significant complications during ECMO treatment, such as AKI, acute gastrointestinal injury, bleeding, thrombosis, and nosocomial infections, can negatively impact prognosis and survival rates. This study performed a retrospective analysis involving 76 children who underwent ECMO. The results indicated that the incidence of AKI during ECMO treatment reached 56.6%. Factors such as the duration of ECMO treatment, pre-initiation lactate levels, and the score of vasoactive drugs may be independently associated with the development of AKI. These findings suggest a hypothesis: in pediatric patients undergoing ECMO, reducing pre-initiation lactate levels, controlling the intensity of vasoactive drugs early, and minimizing the duration of ECMO treatment could potentially decrease the risk of AKI. This outcome corroborates the research findings of other scholars.[2,10]

In the present multivariate analysis, long-term ECMO treatment in patients may be associated with an increased risk of AKI. The continuous exposure of the blood to foreign substances in ECMO circuits can trigger a severe systemic inflammatory response, resulting in the release of

inflammatory mediators such as tumor necrosis factor α and interleukins. These mediators can damage the renal capillary basement membrane,[11,12] which leads to the leakage of macromolecules and development of AKI. Additionally, the mechanical shear force and high rotational speed of the ECMO pump head can damage red blood cells, causing hemolysis and increasing the concentration of free hemoglobin,[13] which can obstruct renal tubules. Heme from free hemoglobin can induce cell apoptosis and disrupt the aerobic respiratory chain, directly or indirectly contributing to AKI. Despite anticoagulant use, microthrombi may still form in long-term ECMO circuits, potentially embolizing into renal artery branches and causing focal renal infarction, which is another significant factor in AKI. Although ECMO can improve hypoxia and organ hypoperfusion, prolonged treatment can lead to rapid hemodynamic fluctuations and fluid intake, causing changes in renal blood flow and ischemia–reperfusion injury-related AKI. Studies indicate that AKI is more likely to occur in the veno-arterial (V-A) ECMO[14] mode because nonpulsatile blood flow from the ECMO pump can affect renal perfusion.[12] Thus, extended ECMO treatment increases the risk of AKI, necessitating dynamic monitoring of vital signs and laboratory indicators to withdraw ECMO as soon as the condition is controlled. Additionally, several factors identified in this study suggest that elevated lactate levels prior to ECMO initiation may be associated with an increased risk of AKI. The level of lactate, a metabolite of anaerobic glycolysis, increases when oxygen delivery or blood perfusion to tissues is insufficient, leading to metabolic acidosis and hyperlactatemia. The kidney, with its rich blood flow, is sensitive to ischemia and hypoxia. In an acidic environment, organelles such as mitochondria in renal tubular epithelial cells can be damaged, exacerbating cell damage and death and leading to AKI. Studies suggest that high lactate levels often correlate with a poor prognosis.[15] Furthermore, the multivariate regression analysis indicates that a high vasoactive drug score during ECMO treatment might be associated with an increased risk of AKI. The kidneys of infants typically exhibit low perfusion pressure and high renal vascular resistance.[6] Vasoactive drugs can increase vascular resistance, accelerate hemodynamic fluctuations, and reduce renal blood flow, leading to ischemic necrosis of renal tubules and reperfusion injury-related AKI. During ECMO, the ability of kidneys to self-regulate blood flow may be impaired. To maintain vital signs in critically ill patients, continuous intravenous administration of one or more vasoactive drugs may be necessary, causing strong vasoconstriction and disrupting renal regulatory balance, reducing renal blood flow and the glomerular filtration rate, and increasing the incidence of AKI. Therefore, prolonged or high-dose vasoactive drug use causes significant damage to the renal microcirculation. Building on previous mechanistic studies, we hypothesize that prolonged ECMO treatment, elevated lactate levels, and increased vasoactive drug scores contribute to the risk of AKI through multiple pathways. However, these hypotheses require direct validation in future research.

The present univariate analysis indicated that prolonged mechanical ventilation might be a potential risk factor for developing AKI during ECMO treatment. Extended positive-pressure ventilation increases thoracoabdominal pressure, potentially compressing the vena cava, reducing venous return, and consequently decreasing renal blood flow and perfusion pressure,[16] which can lead to AKI. Additionally, inflammatory factors that are released because of lung injury can enter the kidneys through the bloodstream, directly damaging renal tubular epithelial cells and causing AKI. The univariate analysis in this study revealed that prolonged combined CRRT was a potential risk factor for AKI. This association may occur because patients who undergo combined CRRT typically present with more severe conditions and are prone to developing multiple organ dysfunction syndrome. Additionally, substantial fluid infusions, such as rehydration and blood transfusions, which are administered during combined therapy to maintain ECMO flow stability, could increase the risk of circulatory volume overload, thereby further impairing organ perfusion function.[17,18] While univariate analysis revealed a correlation of the duration of

mechanical ventilation and CRRT with AKI occurrence, this correlation was not observed in the multivariate analysis. These findings suggest that these factors may primarily reflect confounding influences, such as the severity of the underlying disease in pediatric patients, rather than serve as independent risk factors for AKI.

Some studies suggest that FO can predict AKI early, indicating that fluid management plays a role in AKI prevention. However, no significant difference in FO% was found between the groups. The clinical importance of the finding that an increased output within 24 hours post-ECMO treatment was negatively correlated with AKI remains unclear. Excessive intake of blood products, various drugs, and volume resuscitation may significantly elevate central venous pressure, resulting in venous congestion in multiple tissues and organs. Congestion of the renal vein increases renal venous pressure, which impedes blood flow into the kidneys. This obstruction can lead to a cascade of consequences, including decreased renal perfusion pressure, reduced renal blood flow, and diminished glomerular filtration rate. Furthermore, a decrease in urine output indicates insufficient renal perfusion or impaired systemic hemodynamics, ultimately contributing to ischemic injury of the renal vasculature. While an increased output might indicate better renal perfusion and functional reserve, an OR value near 1 suggested a very weak association. The limited sample size of this study resulted in an events per variable (EPV) of 7.2 for the multivariate analysis, falling below the recommended standard of 10–15. This raises concerns about the risk of overfitting, which could result in inaccurate parameter estimates or inflated effect sizes. Consequently, the patient output findings in this study should be considered exploratory. Future research should aim to increase the sample size or validate the results through multi-center studies. Additionally, measuring urine output in infants is prone to errors, and this study lacked data on diuretic use, which could have influenced the interpretation of the results. Pre-ECMO urine volume, along with other identified risk factors like blood lactate levels and vasoactive drug scores, can be used to categorize patients based on AKI diagnostic criteria (low, medium, and high risk) and develop a straightforward bedside risk assessment tool. However, the effectiveness of this tool in terms of sensitivity and specificity requires validation through larger prospective studies. A limitation of this study is the absence of a statistical analysis of vasoactive drug doses at fixed time points. The VISs of patients who use short-term drugs may match those of patients who use long-term maintenance drugs, yet their hemodynamics could differ significantly. Currently, no studies have explored how the dosage and duration of vasoactive drugs contribute to AKI. Finally, retrospective studies inherently cannot establish causal relationships, establishing only associations between variables. The sample size in this study was relatively small, and including multiple variables in the multivariate analysis might have affected model stability. Since the data were collected from only two centers, selection bias may exist, and the conclusions require further validation.

CONCLUSION

In conclusion, factors potentially associated with an elevated risk of AKI during ECMO treatment include the vasoactive drug score, lactate levels prior to ECMO initiation, and the duration of ECMO. Active interventions should focus on these risk factors and appropriately time the weaning from ECMO once the patient's condition is effectively managed to reduce the incidence of AKI and improve patient prognosis. However, the conclusions of this study do not constitute definitive evidence. The aforementioned assumptions require further validation through large-sample, multi-center, and prospective studies. Future research should implement standardized data collection protocols, include a broader range of potential confounding factors, and investigate

the predictive value of novel biomarkers to furnish more reliable evidence for the prevention and management of ECMO-related AKI in children.

DECLARATIONS

Authors' contributions

Jiaqi Li and Liting Hou contributed equally to this paper, both as the first authors.

(1) Jiaqi Li, Liting Hou and Weikai Wang was conceiving and designing the study;
(2) Jiaqi Li, Liting Hou, Qianqian Liu and Bin Yan, collecting the data;
(3) Jiaqi Li, Liting Hou, Qianqian Liu and Bin Yan, analyzing and interpreting the data;
(4) Jiaqi Li and Liting Hou, writing the manuscript;
(5) Weikai Wang, providing critical revisions that are important for the intellectual content;
(6) All authors approved the final version of the manuscript.

Conflict of interest

All authors declare that they have no conflict of interest.

Funding

The authors declared that financial support was received for this work and/or its publication. This work was supported by the Gansu Provincial Science and Technology Major Project (grant no. 22ZD6FA034): "Research on Key Technologies and Application of Early Identification and Precision Diagnosis and Treatment of Severe Pediatric Infections."

Availability of data and materials

The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

Acknowledgement

The authors would like to thank the staff of the Gansu Provincial Maternity and Child-Care Hospital, Xi'an Children's Hospital, for their support during data collection.

REFERENCES

1 Papazian L, Herridge M, Combes A. Focus on veno-venous ECMO in adults with severe ARDS. *Intensive Care Med.* 2016;42(11):1655–1657. doi:10.1007/s00134-016-4398-0
2 Liao XZ, Cheng Z, Wang LQ, Li BF. Identified risk factors for acute renal failure in patients undergoing extracorporeal membrane oxygenation. Article in Chinese. *J Clin Anesthesiol.* 2018;34(3):241–245.
3 Gristina G, Nardi G, Orazi D, et al. Prone Positioning and Intravenous Zanamivir may Represent Effective Alternatives for Patients with Severe ARDS Virus A (H1N1) Related Pneumonia in Hospitals with no Access to ECMO. *Crit Care Res Pract.* 2010;2010:146456. doi:10.1155/2010/146456
4 Liao MT, Tsai IJ, Lin FH, et al. Risk factors for in-hospital mortality and acute kidney injury in neonatal-pediatric patients receiving extracorporeal membrane oxygenation. *J Formos Med Assoc.* 2021;120(9):1758–1767. doi:10.1016/j.jfma.2021.03.004
5 Pilarczyk K, Huenges K, Bewig B, et al. Acute Kidney Injury in Patients with Severe ARDS Requiring Extracorporeal Membrane Oxygenation: Incidence, Prognostic Impact and Risk Factors. *J Clin Med.* 2022;11(4):1079. doi:10.3390/jcm11041079
6 Cui YL, Liu JP, Zhao J, Feng ZY, Long C, Qian XY. The predictive value of the vasoactive-inotropic score for acute kidney injury in infants undergoing extracorporeal membrane oxygenation. Article in Chinese. *China Med Her.* 2013;10(18):79–81.

7 Kellum JA, Lameire N. Diagnosis, evaluation, and management of acute kidney injury: a KDIGO summary (Part 1). *Crit Care.* 2013;17(1):204. doi:10.1186/cc11454
8 Murphy HJ, Gien J, Sahay R, et al. Acute Kidney Injury, Fluid Overload, and Renal Replacement Therapy Differ by Underlying Diagnosis in Neonatal Extracorporeal Support and Impact Mortality Disparately. *Blood Purif.* 2021;50(6):808–817. doi:10.1159/000512538
9 Gaies MG, Gurney JG, Yen AH, et al. Vasoactive-inotropic score as a predictor of morbidity and mortality in infants after cardiopulmonary bypass. *Pediatr Crit Care Med.* 2010;11(2):234–238. doi:10.1097/PCC.0b013e3181b806fc
10 Haneya A, Diez C, Philipp A, et al. Impact of Acute Kidney Injury on Outcome in Patients With Severe Acute Respiratory Failure Receiving Extracorporeal Membrane Oxygenation. *Crit Care Med.* 2015;43(9):1898–1906. doi:10.1097/ccm.0000000000001141
11 Hepokoski M, Englert JA, Baron RM, et al. Ventilator-induced lung injury increases expression of endothelial inflammatory mediators in the kidney. *Am J Physiol Renal Physiol.* 2017;312(4):F654–F660. doi:10.1152/ajprenal.00523.2016
12 Li YK, Hei FL. Advances in research on extracorporeal membrane oxygenation–associated acute kidney injury. Article in Chinese. *Chin Clin Res.* 2022;35(01):105–109+115.
13 Lyu L, Long C, Hei F, et al. Plasma Free Hemoglobin Is a Predictor of Acute Renal Failure During Adult Venous-Arterial Extracorporeal Membrane Oxygenation Support. *J Cardiothorac Vasc Anesth.* 2016;30(4):891–895. doi:10.1053/j.jvca.2016.02.011
14 Thongprayoon C, Cheungpasitporn W, Lertjitbanjong P, et al. Incidence and Impact of Acute Kidney Injury in Patients Receiving Extracorporeal Membrane Oxygenation: A Meta-Analysis. *J Clin Med.* 2019;8(7):981. doi:10.3390/jcm8070981
15 Lamsfus-Prieto JÁ, de Castro-Fernández R, Hernández-García AM, Marcano-Rodriguez G. Prognostic value of gasometric parameters of carbon dioxide in resuscitation of septic patients. A bibliography review. *Rev Esp Anestesiol Reanim.* 2016;63(4):220–230. doi:10.1016/j.redar.2015.11.005
16 Benites MH, Suarez-Sipmann F, Kattan E, Cruces P, Retamal J. Ventilation-induced acute kidney injury in acute respiratory failure: Do PEEP levels matter? *Crit Care.* 2025;29(1):130. doi:10.1186/s13054-025-05343-5
17 Ahlqvist M, Forsman P, Morberg P, Larsson M, Broman LM, Akram S. Extracorporeal membrane oxygenation in trauma: a single-center retrospective observational study. *Eur J Trauma Emerg Surg.* 2025;51(1):88. doi:10.1007/s00068-024-02734-1
18 Selewski DT, Wille KM. Continuous renal replacement therapy in patients treated with extracorporeal membrane oxygenation. *Semin Dial.* 2021;34(6):537–549. doi:10.1111/sdi.12965