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ORIGINAL ARTICLE

Retrospective Study

Effect of extracorporeal membrane oxygenation combined with hemoperfusion on inflammatory factors in patients with cardiogenic shock

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Abstract

BACKGROUND

Data on adsorptive extracorporeal membrane oxygenation (ECMO) (combined with HA380 hemoperfusion column) on the inflammatory factors in patients with cardiogenic shock (CS) remains limited.

AIM

To investigate the effects of adsorptive ECMO on the inflammatory factors in patients with CS.

METHODS

A retrospective analysis was performed on 81 patients with CS caused by acute myocardial infarction, fulminant myocarditis, or cardiac surgery who required venoarterial ECMO support at TEDA International Cardiovascular Hospital from December 2020 to December 2024. Patients were divided into the conventional ECMO group (42 cases) and the adsorptive ECMO group (ECMO combined with hemoperfusion, 39 cases). The adsorptive ECMO group received 2 columns of HA380 initiation on the first day (the first column connected within 2 hours of ECMO and the second after 12 hours of ECMO), followed by 1 column each day, with each column used for 4–6 hours, totaling 24–30 hours of treatment. Baseline data were compared between the two groups: Inflammatory factor levels (at 0, 6, 12, 24, 48, and 72 hours after ECMO or hemoperfusion initiation); ECMO support

duration; successful weaning rate; continuous renal replacement therapy (CRRT) utilization; Sequential Organ Failure Assessment (SOFA) score; Vasoactive-Inotropic Score (VIS); systemic inflammatory response syndrome (SIRS) incidence; and in-hospital survival and 30-/90-day survival after discharge.

RESULTS

The adsorptive ECMO group showed significantly lower levels of C-reactive protein, interleukin (IL)-6, tumor necrosis factor (TNF)-α, and lactate from 6 to 72 hours compared with the conventional ECMO group (all $P < 0.05$), with IL-6 decreasing by 94.4% and tumour necrosis factor alpha by 70.1% from baseline at 72 hours. The adsorptive ECMO group had a significantly shorter ECMO duration [114.0 (75.0–139.0) hours *vs* 135.0 (73.0–199.3) hours, $P = 0.032$]; higher successful weaning rate (66.7% *vs* 42.9%, $P = 0.032$); a trend toward lower CRRT utilization (54.8% *vs* 38.5%, $P = 0.070$); lower post-weaning SOFA score [7 (6–8) *vs* 9 (8–10), $P < 0.001$]; significantly reduced VIS (8.4 ± 1.3 *vs* 9.8 ± 1.6, $P < 0.001$); and a trend toward lower SIRS incidence (10.3% *vs* 26.2%, $P = 0.065$). There were no significant differences in complications, in-hospital survival (64.1% *vs* 52.4%, $P = 0.285$); or 30-/90-day survival between the two groups (all $P > 0.05$).

CONCLUSION

Adsorptive ECMO efficiently clears IL-6 and TNF-α, significantly improving ECMO weaning success rate and hemodynamics. However, it has no significant impact on survival, and its efficacy requires validation through prospective studies.

Key Words: Adsorptive extracorporeal membrane oxygenation; Cardiogenic shock; Inflammatory factors; Hemoperfusion column; Therapeutic outcomes

Core Tip: This study evaluates the efficacy of adsorptive extracorporeal membrane oxygenation (ECMO) (ECMO combined with HA380 hemoperfusion column) in reducing inflammatory burden in cardiogenic shock patients. Our single-center retrospective study ($n = 78$) demonstrates that adsorptive ECMO efficiently clears pro-inflammatory cytokines (interleukin-6: 94.4% reduction at 72 hours; tumour necrosis factor alpha: 70.1% reduction) and improves hemodynamics, including shorter ECMO duration (114 *vs* 141 hours, $P = 0.046$) and higher weaning success rate (66.7% *vs* 38.5%, $P = 0.013$).

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INTRODUCTION

Cardiogenic shock (CS) is a critical condition characterized by tissue hypoperfusion and poor prognosis[1]. Venoarterial extracorporeal membrane oxygenation (VA-ECMO), a mechanical circulatory support device, is widely used in treating refractory CS, giving time to identify the etiology and restore cardiopulmonary function[2]. However, clinical practice shows that VA-ECMO support is associated with a high incidence of complications and mortality[3]. The pathophysiology involves multiple factors: Blood contact with nonbiological surfaces of the extracorporeal circuit, endotoxemia, and ischemia–reperfusion injury can all induce systemic inflammatory responses[4,5]. Exposure of blood to nonbiological surfaces activates immune, coagulation, and inflammatory cascades, further exacerbating inflammatory amplification[6,7]. When uncontrolled excessive inflammation persists and is not effectively regulated by compensatory anti-inflammatory mechanisms, it progresses to systemic inflammatory response syndrome (SIRS), end-organ dysfunction, and even death[8,9]. Therefore, exploring strategies to mitigate ECMO-related systemic inflammation is of clinical importance.

The HA380 hemoperfusion (HP) column, utilizing specific adsorptive materials, enables specific adsorption and removal of medium and large molecular substances [such as proinflammatory cytokines interleukin (IL)-6 and tumor necrosis factor (TNF)-α] from the blood, offering a new approach to controlling excessive inflammation in CS patients[10]. Although ECMO and HP have been studied separately in critical care, research on integrating HP columns as additional components into VA-ECMO systems (*i.e.*, adsorptive ECMO) for treating CS remains limited, particularly regarding critical data on dynamic changes in inflammatory factors[11]. We hypothesized that adsorptive ECMO would more efficiently clear pro-inflammatory cytokines (IL-6, TNF-α) and improve clinical outcomes (ECMO weaning success, hemodynamics) compared to conventional ECMO in patients with cardiogenic shock. Therefore, this retrospective study evaluated the inflammatory clearance efficacy and clinical outcomes of adsorptive ECMO in patients with CS caused by acute myocardial infarction, fulminant myocarditis, or cardiac surgery.

MATERIALS AND METHODS

Patient selection

A retrospective analysis included 81 patients with CS caused by acute myocardial infarction, fulminant myocarditis, or cardiac surgery who received VA-ECMO at TEDA International Cardiovascular Hospital from December 2020 to December 2024.

Inclusion criteria: (1) Age ≥ 18 years; (2) Diagnosis of CS defined by systolic blood pressure < 90 mmHg or dependence on vasoactive drugs to maintain blood pressure, cardiac index < 2.2 L/min/m², and pulmonary capillary wedge pressure ≥ 18 mmHg[12]; (3) ECMO initiation within 24 hours of shock onset; (4) Signed informed consent; and (5) Complete medical records.

Exclusion criteria: (1) History of chronic heart failure (NYHA class III/IV), awaiting heart transplantation, or requiring long-term mechanical circulatory support; (2) Concomitant malignant tumor or immunosuppressive status; (3) Severe infection (*e.g.*, sepsis); (4) Severe chronic obstructive pulmonary disease or chronic renal failure; and (5) Incomplete medical records. The study was approved by the Ethics Committee of TEDA International Cardiovascular Hospital (2022K034).

Grouping strategy

Patients were consecutively enrolled from our ECMO registry and grouped retrospectively by actual treatment received: Conventional ECMO group (standard VA-ECMO support only, n = 42); and adsorptive ECMO group [VA-ECMO combined with HP, n = 39]. Group allocation was strictly based on documented treatment protocols to ensure accuracy and objectivity.

Treatment protocols

Standard ECMO therapy: ECMO was initiated immediately after CS diagnosis (PLS BE-PLS 2050, Maquet, United States). Target parameters were maintained by adjusting flow and rotational speed: Mean arterial pressure (MAP) 65–75 mmHg; central venous oxygen saturation ≥ 70%; and oxygenation index (PaO_2/FiO_2) > 200 mmHg. Anticoagulation was achieved with intravenous heparin or argatroban, monitored every 4 hours by activated clotting time (target: 180–200 seconds) and activated partial thromboplastin time (target: 60–80 seconds or 1.5–3 times baseline). Invasive hemodynamic monitoring and bedside echocardiography evaluated cardiac function. ECMO weaning was initiated when hemodynamics and homeostasis stabilized, defined as no re-initiation of support for 48 hours.

Adsorptive ECMO therapy: An HA380 HP column (Jafron Biomedical Co., China), packed with neutral macroporous resin adsorbent, was connected in parallel to the ECMO circuit within 2 hours of ECMO initiation for CS patients with SIRS or Sequential Organ Failure Assessment (SOFA) scored ≥ 10. The column specifically adsorbed lipophilic and hydrophobic medium-to-large molecules (60 kDa), including IL-6, TNF-α, and protein-bound toxins, exhibiting a dynamic binding capacity of 280 mg/g resin for IL-6, with 60% cytokine removal achieved within 30 minutes of treatment. After heparinization and priming, the column was connected in parallel to the ECMO circuit: Blood from the oxygenator split into two pathways, with a blood flow rate of 1.5-2.0 L/min through the adsorptive branch to remove inflammatory factors (approximately 30% of total ECMO flow) before recombining and returning to the patient *via* the blood pump.

A 2-1-1 protocol used in HP column management included: 2 columns of HA380 initiation on the first day (the first column connected within 2 hours of ECMO and the second after 12 hours of ECMO), followed by 1 column each day, with each column used for 4-6 hours, totaling 24-30 hours of treatment. Intermittent column replacement every 12 hours has been shown to optimize cytokine removal while minimizing hemocompatibility issues[13]. Anticoagulation was consistent with the conventional ECMO group to maintain target anticoagulation intensity.

Data collection

Baseline data included: Demographics; primary etiology, laboratory indices (complete blood count, liver/kidney function, coagulation parameters); hemodynamic parameters [left ventricular ejection fraction, SOFA score, Acute Physiology and Chronic Health Evaluation II score]; and baseline SIRS incidence. Outcome measures included: ECMO support duration; successful weaning; continuous renal replacement therapy utilization; post-weaning 24-hour SOFA score; vasoactive-inotropic score [VIS, calculated as dopamine + dobutamine + 100 × (norepinephrine + epinephrine) in μg/kg/min]; SIRS incidence [≥ 2 criteria: (1) Temperature > 38 or < 36°C; (2) Heart rate > 90 bpm; (3) Respiratory rate > 20 breaths/min or $PaCO_2$ < 32 mmHg; (4) White blood cell count > 12 × 10^9/L or < 4 ×10^9/L or immature granulocytes > 10%; and (5) In-hospital survival, and 30-day and 90-day survival after discharge (*via* medical records or telephone follow-up)].

Inflammatory factor monitoring

Peripheral venous blood samples were collected at six time points: T0 (within 2 hours of ECMO initiation); T6 (6 hours); T12 (12 hours); D1 (24 hours); D2 (48 hours); and D3 (72 hours) for measurement of IL-6, TNF-α, C-reactive protein (CRP), and lactate.

Table 1 Baseline characteristics, *n* (%)

	ECMO (*n* = 42)	ECMO+HP (*n* = 39)	*P* value
Age, year	56.6 ± 14.3	59.2 ± 12.0	0.231
Male	34 (81.0)	24 (61.5)	0.091
Body mass index, kg/m²	25.7 ± 3.3	24.8 ± 2.6	0.102
Etiology of cardiogenic shock			
Acute myocardial infarction	30 (71.4)	27 (69.2)	0.807
Myocarditis	2 (4.8)	1 (2.6)	
Postcardiac surgery	10 (23.8)	11 (28.2)	
Hypertension	24 (57.1)	21 (53.8)	0.765
Diabetes	14 (33.3)	10 (25.6)	0.449
White blood cell, 10^9/L	10.0 (8.3, 15.0)	9.7 (6.8, 14.4)	0.33
Blood platelet, /L	186.0 (145.0, 249.0)	151.0 (83.0, 209.0)	0.126
APTT, seconds	38.7 (28.6, 54.4)	37.5 (27.4, 52.1)	0.606
Procalcitonin, ng/mL	2.3 (0.6, 11.2)	2.2 (0.3, 15.7)	0.712
Alanine aminotransferase, U/L	62.5 (25.5, 127.0)	63.0 (29.0, 313.0)	0.292
Total bilirubin, μmol/L	14.7 (9.1, 29.7)	14.1 (8.8, 26.7)	0.484
Serum creatinine, μmol/L	94.0 (71.5, 123.5)	95.0 (72.0, 163.0)	0.577
Left ventricular ejection fractions	31.5 (19.5, 39.0)	36.0 (29.0, 41.0)	0.097
SOFA score	10.0 (8.0, 12.0)	10.0 (9.0, 12.0)	0.181
APACHEII score	14 (12, 17)	17 (14, 19)	0.073
Vasoactive-inotropic score	19.3 ± 1.9	19.1 ± 2.0	0.782
SIRS	11 (26.2)	13 (33.3)	0.482

ECMO: Extracorporeal membrane oxygenation; HP: Hemoperfusion; APTT: Activated partial thromboplastin time; SOFA: Sequential organ failure assessment; APACHE II: Acute Physiology and Chronic Health Evaluation II; SIRS: Systemic inflammatory response syndrome.

Statistical analysis

Continuous data were expressed as mean ± SD while categorical data were displayed with number and percentage (%). For comparison between conventional and adsorptive ECMO groups, Student's independent *t* test was used; if normality was not assumed, the Mann–Whitney *U* test was used instead. A χ^2 test was used to test the difference in the distribution of categorical data between the two groups. Two-way mixed-design analysis of variance and Fisher's least significant difference as *post hoc* comparisons were used to compare the differences between groups and among time points. A two-tailed $P < 0.05$ was recognized as statistically significant of each test. All analyses were performed using IBM SPSS version 25.0 (IBM Corporation, Somers, NY, United States).

RESULTS

Patients' demographic and clinical characteristics

A total of 81 patients were enrolled, including 42 in the conventional ECMO group and 39 in the adsorptive ECMO group. Baseline characteristics, including demographics, etiology of CS, laboratory parameters, severity scores, and baseline SIRS incidence, were comparable between the two groups ($P > 0.05$, Table 1).

Hemodynamics and blood biochemical data

Hemodynamic parameters and the estimations of group differences are displayed in Table 2. There were no differences in MAP among all time points between the two groups. CRP levels in the adsorptive ECMO group were significantly lower at T6, T12, and D1–D3 time points, with a sustained downward trend, whereas CRP continued to rise in the conventional group. IL-6 Levels in the adsorptive group were significantly reduced from T6 onward, achieving a 94.4% reduction at D3 compared to baseline, in contrast to the fluctuating elevation observed in the conventional group. Lactate levels in the adsorptive group decreased significantly at T6, maintaining low levels throughout D1–D3, while the conventional group

Table 2 Inflammatory factors and clinical indicator in two groups

	T0	T6	T12	D1	D2	D3	*P* value
C-reactive protein, mg/L							
ECMO	36.0 (10.4, 107.7)	62.7 (37.4, 124.9)	74.5 (48.7, 123.1)	98.5 (59.0, 152.6)	132.8 (75.2, 181.4)	130.8 (84.9, 193.3)	< 0.001
ECMO+HP	34.6 (22.4, 113.3)	128.3 (63.6, 178.3)[a]	65.3 (37.3, 91.0)[a]	43.2 (25.9, 64.6)[a]	31.5 (21.2, 53.9)[a]	18.3 (9.7, 25.6)[a]	< 0.001
Interleukin-6, pg/mL							
ECMO	203.5 (118.7, 420.0)	310.6 (221.0, 462.9)	341.0 (133.9, 549.5)	247.3 (175.2, 460.6)	391.9 (234.8, 494.2)	339.9 (202.0, 485.4)	0.109
ECMO+HP	214.1 (160.9, 388.0)	146.3 (108.9, 225.4)[a]	101.0 (69.2, 123.4)[a]	57.0 (45.4, 78.5)[a]	32.7 (24.3, 47.5)[a]	11.9 (9.3, 15.6)[a]	< 0.001
Lactic acid, mmol/L							
ECMO	14.6 (11.0, 17.4)	17.9 (14.0, 20.7)	17.4 (14.2, 21.0)	17.9 (13.2, 20.4)	18.0 (14.4, 20.6)	16.2 (12.7, 20.7)	0.115
ECMO+HP	16.1 (12.7, 18.4)	5.5 (2.7, 11.4)[a,b]	2.9 (1.9, 4.5)[a,b]	1.8 (1.5, 2.4)[a,b]	1.4 (1.2, 2.0)[a,b]	1.3 (0.9, 2.5)[a,b]	< 0.001
Tumor necrosis factor-α, pg/mL							
ECMO	416.6 ± 81.8	423.0 ± 68.4	420.6 ± 75.4	414.1 ± 70.0	435.5 ± 70.0	420.1 ± 69.4	0.834
ECMO+HP	412.0 ± 90.7	307.1 ± 95.1[a]	249.6 ± 84.1[a,b]	216.0 ± 79.0[a,b]	178.2 ± 66.4[a,b]	123.3 ± 58.5[a,b]	< 0.001
Mean arterial pressure, mmHg							
ECMO	60.2 ± 9.4	61.3 ± 7.5	61.5 ± 6.3	63.2 ± 6.6	64.8 ± 8.0	62.4 ± 6.8	0.312
ECMO+HP	58.9 ± 8.5	59.5 ± 7.4	60.0 ± 5.4	61.3 ± 6.9	62.0 ± 6.0	60.8 ± 6.4	0.187

[a]$P < 0.05$ *vs* ECMO group (two-way analysis of variance with Fisher's least significant difference *post hoc*).
[b]$P < 0.05$ *vs* T0 within group (Wilcoxon signed-rank test).
ECMO: Extracorporeal membrane oxygenation; HP: Hemoperfusion.

exhibited persistent elevation above normal ranges. TNF-α levels in the adsorptive group were significantly lower from T6 onward, with a 70.1% reduction at D3.

Complications and prognosis

Clinical outcomes showed that the adsorptive ECMO group had a significantly shorter ECMO support duration [114.0 (75.0-139.0) hours *vs* 135.0(73.0-199.3) hours, $P = 0.046$], higher successful weaning rate (66.7% *vs* 42.9%, $P = 0.013$), lower post-weaning SOFA score [7 (6-8) *vs* 9 (8–10), $P < 0.001$], and reduced VIS (8.4 ± 1.3 *vs* 9.8 ± 1.6, $P < 0.001$). A trend downward to SIRS incidence (10.3% *vs* 26.2%, $P = 0.065$) was observed in the adsorptive group. No significant group differences were noted in complications (hemorrhage rate: 30.8% *vs* 31.0%; infection rate: 43.6% *vs* 56.4%), and in-hospital, 30-day, and 90-day survival (Table 3).

DISCUSSION

Adsorptive ECMO efficiently cleared IL-6 (94.4% reduction at 72 hours), TNF-α (70.1%), and lactate (65.8% at 6 hours), leading to shorter ECMO duration (114 vs. 135 hours, $P = 0.032$), higher weaning success (66.7% *vs* 42.9%, $P = 0.013$), and improved hemodynamics (lower SOFA/VIS scores). Despite these improvements, in-hospital survival did not differ between groups (64.1% *vs* 52.4%, $P = 0.285$).

As an extracorporeal purification technique for targeted removal of medium-molecule inflammatory mediators, HP blocks the cascade amplification of the cytokine storm, forming a synergistic effect with ECMO in improving tissue perfusion[14]. In recent years, HP has been increasingly studied in critical care settings such as sepsis and acute respiratory distress syndrome (ARDS)[15-19]. In this study, the etiological distribution of CS caused by acute myocardial infarction, fulminant myocarditis, or cardiac surgery was balanced between the two groups (Table 1). Although cardiac surgery patients may have higher initial inflammatory burden due to cardiopulmonary bypass stress, the adsorptive ECMO group demonstrated consistent IL-6 clearance across all three etiologies, suggesting the broad applicability of the anti-inflammatory effect of HP independent of specific causes. However, evidence on cytokine clearance by ECMO combined with HP in CS patients remains limited.

Table 3 Complications and prognosis in two groups, *n* (%)

	ECMO (*n* = 42)	ECMO+HP (*n* = 39)	*P* value
Hemorrhage	13 (31.0)	12 (30.8)	0.986
Infection	22 (56.4)	17 (43.6)	0.429
Central nervous system dysfunction	5 (11.9)	4 (10.3)	0.723
Use of intra-aortic balloon pumping	34 (81.0)	31 (79.5)	0.774
Use of continuous renal replacement therapy	17 (40.5)	21 (53.8)	0.228
ECMO operation time, hour	135.0 (73.0, 199.3)	114.0 (75.0, 139.0)	0.046
Successful weaning of ECMO	18 (42.9)	26 (66.7)	0.013
SOFA after weaning	9 (8, 10)	7 (6, 8)	< 0.001
SIRS after weaning	11 (26.2)	4 (10.3)	0.077
Vasoactive-inotropic score	9.8 1.6	8.4 1.3	< 0.001
Duration of hospitalization, d	23 (13, 45.3)	22 (10, 41)	0.762
In-hospital survival	22 (52.4)	25 (64.1)	0.285
30-day survival	15 (38.5)	18 (46.2)	0.492
90-day survival	13 (33.3)	16 (41.0)	0.482

ECMO: Extracorporeal membrane oxygenation; HP: Hemoperfusion; SOFA: Sequential Organ Failure Assessment score; SIRS: Systemic inflammatory response syndrome.

ECMO combined with HP has shown potential to reduce inflammatory burden and improve organ function in critical conditions like septic shock and ARDS[20,21], which aligns with our findings. The adsorptive ECMO group exhibited significantly lower CRP, IL-6, and TNF-α levels from 6 to 72 hours, with faster lactate clearance[22]. Notably, the sterile CS caused by acute myocardial injury in this study shared inflammatory mechanisms with infectious CS, implying the universal applicability of HP across different CS pathologies[14]. Despite the lack of significant difference in in-hospital survival, the potent IL-6 clearance (94.4% reduction at 72 hours) and improved ECMO weaning success rate (66.7% *vs* 42.9%) provide a new therapeutic direction for cardiac-related CS. Kogelmann *et al*[22] also reported that venovenous ECMO combined with HP improved lung function by reducing hyperlactatemia in sepsis-associated ARDS patients, consistent with our finding of a 65.8% lactate reduction within 6 hours in the adsorptive ECMO group, indicating improved microcirculatory perfusion[23]. Additionally, the rapid IL-6 clearance observed here corroborates Bruenger *et al*'s experience[24] in cardiogenic septic shock, jointly validating the targeted regulation of core proinflammatory cytokines by this combined therapy.

The improvement in clinical parameters supports the positive impact of inflammation control on hemodynamics. The adsorptive ECMO group showed shorter ECMO duration, lower post-weaning SOFA scores, and reduced VIS, consistent with the results of Na *et al*[25] that "a 10-point reduction in VIS is associated with a 41% mortality decrease," suggesting that inflammation mitigation may reduce vasopressor dependence by improving myocardial contractility and microcirculation.

The development of SIRS is determined by the balance between pro- and anti-inflammatory cytokines, with prior studies linking its severity to mortality[26]. Although the adsorptive ECMO group trended toward lower post-weaning SIRS incidence (10.3% *vs* 26.2%), this did not reach statistical significance, likely due to the small sample size, and it requires validation in larger studies. Similar baseline coagulation function and comparable bleeding complication rates (30.8% *vs* 31.0%) confirmed the compatibility and safety of HP with ECMO anticoagulation protocols[27]. However, this study found no direct evidence of clinical benefit from ECMO combined with HP. Despite improved weaning success, there were no differences in in-hospital, 30-day, or 90-day survival, possibly due to the retrospective design, small sample size, or the inherent high lethality of CS. Similarly, studies on extracorporeal cardiopulmonary resuscitation combined with cytokine adsorption have not shown survival benefits, highlighting the need for cautious interpretation of single inflammation-control strategies in complex critical illnesses[28].

This study had several limitations. First, its single-center retrospective design may have introduced selection bias. Second, the small sample size may have affected the statistical power for secondary endpoints (*e.g.*, SIRS incidence). Third, only proinflammatory cytokines were monitored, without assessment of anti-inflammatory cytokines (*e.g.*, IL-10) or coagulation indices (*e.g.*, D-dimer), limiting the comprehensive evaluation of the impact of the adsorptive cartridge on the coagulation–inflammation balance.

CONCLUSION

In conclusion, adsorptive ECMO efficiently clears proinflammatory cytokines (IL-6 and TNF-α), significantly shortens ECMO duration, and improves weaning success with good safety, but has no significant impact on hospitalization duration or short-/Long-term survival. Prospective, multicenter randomized controlled trials are needed to determine the clinical value and optimal population for HP in CS treatment.

FOOTNOTES

Author contributions: Hao JY contributed to study design, data collection, initial manuscript writing; Wang SF and Yang Q contributed to research methodology design, technical support; Wang W and Zhao ZX contributed to data analysis and statistical processing; Guo S and Zhou Y contributed to data validation, figure and table preparation; Dong F contributed to study supervision and project coordination; Lin WH contributed to research guidance, funding acquisition and manuscript review.

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Institutional review board statement: This study was approved by the Ethics Committee of TEDA International Cardiovascular Hospital (Ethics Approval No.: 2022K034). It was conducted in accordance with the principles of the Declaration of Helsinki, and written informed consent was obtained from all patients or their legal guardians.

Informed consent statement: Due to the anonymization of all patient data, no additional consent for publication is required.

Conflict-of-interest statement: All authors declare no financial or non-financial competing interests.

Data sharing statement: The data generated in this study are stored in the TEDA International Cardiovascular Hospital ECMO Registry (Registration No.: 2022-034). Anonymized data may be requested from the corresponding author upon reasonable request.

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