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Adjunctive Intra-Aortic Balloon Pump During Venovenous Extracorporeal Membrane Oxygenation for Severe Respiratory Failure With Cardiac Dysfunction: A Retrospective Cohort Study

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Abstract

Background

Evidence regarding intra-aortic balloon pump (IABP) use during venovenous extracorporeal membrane oxygenation (VV-ECMO) is limited. We examined clinical outcomes and hemodynamic trajectories in patients with severe respiratory failure and cardiac dysfunction treated with or without adjunctive IABP.

Methods

In this single-center retrospective cohort study, five patients who received VV-ECMO + IABP were compared with 24 patients who received VV-ECMO only (16 original controls and eight additional controls with high-dose vasopressor exposure). The primary outcome was ventilator-free days through day 28 (VFD-28). Secondary outcomes included 7-day norepinephrine-equivalent and vasoactive-inotropic score (VIS) area under the curve (AUC), 72-hour lactate AUC, renal replacement therapy (RRT), mortality, and bleeding. The main sensitivity analysis compared the five VV-ECMO + IABP patients with all eight additional controls who had a norepinephrine-equivalent dose of at least 0.5 μg/kg/min on one or more days from D0 through D7. Additional sensitivity analyses used the 16 original controls and the four additional controls who met the high-dose threshold at D0.

Results

The VV-ECMO + IABP group had higher baseline SOFA scores, PEEP, and D0 norepinephrine-equivalent doses than the VV-ECMO-only group. Median VFD-28 was 0.0 days (interquartile range [IQR], 0.0–6.9) versus 0.7 days (IQR, 0.0–9.9; *P* = 0.740; rank-biserial correlation, − 0.10; 95% CI, − 0.54 to 0.41). Norepinephrine-equivalent AUC was 0.89 versus 0.76 (μg/kg/min)·day (*P* = 0.600), VIS AUC was 89.2 versus 68.8 score·days (*P* = 0.780), and verified 28-day mortality was 40.0% versus 37.5% (*P* = 1.000). RRT duration was 5.0 versus 1.0 days (*P* = 0.041), and recorded bleeding occurred in 80.0% versus 37.5% of patients (*P* = 0.144). In the 5-vs-8 sensitivity analysis, VFD-28 was 0.0 versus 0.0 days (*P* = 1.000), norepinephrine-equivalent AUC was 0.89 versus 2.01 (μg/kg/min)·day (*P* = 0.268), and verified 28-day mortality was 40.0% versus 37.5% (*P* = 1.000).

Conclusions

In this small retrospective cohort, adjunctive IABP during VV-ECMO was not associated with higher VFD-28, lower cumulative vasoactive exposure, or lower 28-day mortality. These findings require confirmation in studies with prospective cardiac phenotyping and larger samples.

Background

Severe hypoxemic respiratory failure, including acute respiratory distress syndrome (ARDS), is characterized by marked gas-exchange impairment and frequent extrapulmonary organ dysfunction. The 2024 global ARDS definition broadened diagnostic recognition beyond the Berlin criteria [1]. For patients with refractory hypoxemia or hypercapnic acidosis despite conventional management, VV-ECMO provides extracorporeal gas exchange and permits reduction of injurious ventilatory intensity [2, 3]. VV-ECMO, however, does not directly provide systemic circulatory support.

Contemporary VV-ECMO management includes lung-protective ventilation, prone positioning, and prevention of extrapulmonary organ injury. A randomized trial found that ultra-lung-protective ventilation during VV-ECMO reduced mechanical power but did not reduce plasma biomarkers of biotrauma over 48 hours, whereas a prospective propensity-matched study reported an association between early prone positioning during VV-ECMO and higher rates of successful ECMO weaning and lower 60-day mortality [4, 5]. Analyses of the timing and causes of death during VV-ECMO further indicate that outcomes are determined by both respiratory and nonrespiratory organ failure [6].

Cardiovascular dysfunction during severe ARDS may result from pulmonary vascular injury, hypoxemia, hypercapnia, positive-pressure ventilation, sepsis, or pre-existing cardiac disease. Acute cor pulmonale and right ventricular (RV) dysfunction occur despite lung-protective ventilation [7, 8]. RV injury during VV-ECMO has been associated with increased mortality, and recent expert consensus recommends standardized terminology, multimodal assessment, and serial echocardiographic evaluation [9, 10]. Serial echocardiographic studies have shown that RV dilatation or dysfunction may persist or progress during VV-ECMO, although the independent contribution of VV-ECMO exposure remains uncertain [11–15].

IABP provides diastolic augmentation and modest left ventricular (LV) afterload reduction but does not replace cardiac output. Published experience with IABP during VV-ECMO is limited to case-level evidence [16]. Randomized evidence does not support routine IABP use in myocardial-infarction-related cardiogenic shock [17], while observational studies of IABP combined with VA-ECMO have reported heterogeneous results in selected cardiogenic shock populations [18–20]. Because the hemodynamic effects of VA-ECMO differ from those of VV-ECMO, these findings cannot be directly applied to patients receiving VV-ECMO. We therefore compared clinical outcomes and hemodynamic trajectories between patients receiving VV-ECMO + IABP and those receiving VV-ECMO only.

Methods

Study design, setting, and ethics

This single-center retrospective observational cohort study was conducted in the Department of Critical Care Medicine at Dongguan People's Hospital and included admissions from February 2022 through September 2025. The study was reported in accordance with the Strengthening the Reporting of

Observational Studies in Epidemiology statement (STROBE) [21]. The protocol was approved by the Medical Ethics Committee of Dongguan People's Hospital (approval no. IRBIITKY-2026-268-01).

Study population and analytic cohorts

The study cohort was assembled by screening 78 adults treated with VV-ECMO for severe hypoxemic respiratory failure. Eligible patients were aged 18 years or older, received VV-ECMO, and had clinical or echocardiographic evidence of cardiac dysfunction. Exclusion criteria were venoarterial or venoarterial-venous ECMO, ECMO support for less than 24 hours, VV-ECMO initiated after IABP removal, and absence of cardiac dysfunction.

IABP exposure was defined as IABP use in temporal association with VV-ECMO support for cardiac dysfunction or persistent circulatory failure. Treatment selection and timing were determined by the treating clinicians. IABP was therefore analyzed as a cohort-level exposure rather than as a time-varying intervention.

The primary analysis compared five patients receiving VV-ECMO + IABP with 24 patients receiving VV-ECMO only. The VV-ECMO-only group comprised 16 original controls and eight additional controls who had a norepinephrine-equivalent dose of at least 0.5 μg/kg/min on one or more days from D0 through D7. The main sensitivity analysis compared the five VV-ECMO + IABP patients with all eight additional high-dose controls. Two additional sensitivity analyses compared the VV-ECMO + IABP group with the 16 original controls and with the four additional controls who met the high-dose threshold at D0. Because four of the eight additional controls first met the threshold after D0, the 5-vs-8 analysis was considered post-baseline severity-enriched.

Data sources, time origin, and quality control

Demographic characteristics, disease etiology, severity scores, ventilatory settings, echocardiographic findings, cardiac biomarkers, lactate, daily norepinephrine-equivalent dose, daily VIS, organ-support duration, mortality, and bleeding events were abstracted from the medical records. D0 for longitudinal hemodynamic variables was the day of VV-ECMO initiation. Lactate was evaluated at 0, 6, 12, 24, 48, and 72 hours. Norepinephrine-equivalent doses were calculated using a published dose-equivalence approach [24].

Outcomes

The primary outcome was VFD-28 from VV-ECMO initiation, defined using the source-recorded duration of mechanical ventilation and vital status through day 28 [22, 23]. Secondary outcomes were the trapezoidal AUC from D0 through D7 for daily norepinephrine-equivalent dose and VIS; lactate AUC from 0 through 72 hours; percentage lactate decline from 0 to 72 hours; VV-ECMO duration; RRT use and RRT

days within 14 days; ICU and hospital length of stay; ICU, hospital, and verified 28-day mortality; and any recorded bleeding event.

Statistical analysis

Continuous variables are presented as median (IQR), and categorical variables as number (percentage). Baseline differences were described using standardized mean differences (SMDs); baseline hypothesis tests were not performed. Continuous outcomes were compared using two-sided exact conditional rank-permutation tests. Effect estimates are reported as rank-biserial correlations (r_rb) with 95% CIs obtained from 3,000 group-stratified nonparametric bootstrap samples. Binary outcomes were compared using Fisher's exact test and are reported with absolute RDs and Newcombe–Wilson 95% CIs.

The primary outcome was evaluated using a two-sided α of 0.05. Secondary and sensitivity analyses were exploratory and are reported with unadjusted two-sided P values; no multiplicity correction was applied. Longitudinal plots present medians and IQRs without pointwise hypothesis testing. Analyses were performed using Python version 3.9.12.

Results

Study Cohort

During the study period, 29 patients were included in the primary analysis: five in the VV-ECMO + IABP group and 24 in the VV-ECMO-only group (Fig. 1). The VV-ECMO-only group included 16 original controls and eight additional high-dose controls. All eight additional controls met the norepinephrine-equivalent threshold during D0–D7, and four met the threshold at D0.

Baseline characteristics

Baseline characteristics are shown in Table 1.

Table 1
Baseline characteristics between VV-ECMO + IABP and VV-ECMO only group

Characteristic	VV-ECMO + IABP (n = 5)	VV-ECMO only (n = 24)	SMD
Age, years	52.0 [46.0, 62.0]	59.0 [44.2, 70.5]	-0.37
BMI, kg/m²	22.4 [22.1, 24.1]	25.4 [23.4, 26.1]	-0.84
PaO_2/FiO_2, mmHg	71.0 [66.0, 90.0]	97.0 [61.8, 182.5]	-0.47
PEEP, cmH_2O	13.0 [12.0, 14.0]	10.0 [7.5, 10.5]	1.29
SOFA score	18.0 [14.0, 18.0]	13.0 [11.8, 15.0]	0.95
APACHE II score	28.0 [26.0, 29.0]	23.0 [19.0, 27.0]	0.59
LVEF, %	50.0 [47.0, 53.0]	52.0 [43.5, 60.0]	-0.10
Norepinephrine-equivalent dose at D0, μg/kg/min	1.01 [0.20, 1.34]	0.34 [0.14, 0.68]	0.78
Lactate at 0 h, mmol/L	5.0 [3.3, 7.0]	2.7 [1.8, 4.4]	0.20
Female sex	3 (60.0%)	8 (33.3%)	—

Values are median [IQR] unless otherwise indicated. SMDs are based on available cases; baseline P values were not calculated. Abbreviations: APACHE II, Acute Physiology and Chronic Health Evaluation II; IABP, intra-aortic balloon pump; LVEF, left ventricular ejection fraction; PEEP, positive end-expiratory pressure; SOFA, Sequential Organ Failure Assessment.

Median age was 52.0 years (IQR, 46.0–62.0) in the VV-ECMO + IABP group and 59.0 years (IQR, 44.2–70.5) in the VV-ECMO-only group (SMD, − 0.37). The VV-ECMO + IABP group had higher PEEP (13.0 [IQR, 12.0–14.0] vs 10.0 [IQR, 7.5–10.5] cmH2O; SMD, 1.29), SOFA scores (18.0 [IQR, 14.0–18.0] vs 13.0 [IQR, 11.8–15.0]; SMD, 0.95), APACHE II scores (28.0 [IQR, 26.0–29.0] vs 23.0 [IQR, 19.0–27.0]; SMD, 0.59), and D0 norepinephrine-equivalent doses (1.01 [IQR, 0.20–1.34] vs 0.34 [IQR, 0.14–0.68] μg/kg/min; SMD, 0.78). Median norepinephrine-equivalent dose decreased from 1.01 μg/kg/min at D0 to 0.03 μg/kg/min at D7 in the VV-ECMO + IABP group and from 0.34 to 0.02 μg/kg/min in the VV-ECMO-only group (Fig. 2). Median VIS decreased from 100.67 to 0 and from 33.76 to 0.78, respectively (Fig. 3). Median lactate decreased from 4.95 mmol/L at 0 hours to 2.70 mmol/L at 72 hours in the VV-ECMO + IABP group and from 2.70 to 1.30 mmol/L in the VV-ECMO-only group (Fig. 4).

Outcomes

Median VFD-28 was 0.0 days (IQR, 0.0–6.9) in the VV-ECMO + IABP group and 0.7 days (IQR, 0.0–9.9) in the VV-ECMO-only group (*P* = 0.740; r_rb = − 0.10; 95% CI, − 0.54 to 0.41) (Table 2).

Table 2
Primary and secondary outcomes between VV-ECMO + IABP and VV-ECMO only group

Outcome	VV-ECMO + IABP	VV-ECMO only	*P* value	Effect estimate (95% CI)
Ventilator-free days through day 28	0.0 [0.0, 6.9]	0.7 [0.0, 9.9]	0.740	r_rb -0.10 (-0.54 to 0.41)
Norepinephrine-equivalent AUC D0-D7	0.89 [0.55, 1.84]	0.76 [0.35, 2.46]	0.600	r_rb -0.17 (-0.63 to 0.34)
VIS AUC D0-D7	89.2 [39.7, 184.6] (n = 5)	68.8 [50.8, 198.6]	0.780	r_rb -0.10 (-0.68 to 0.50)
Lactate AUC 0–72 h, mmol·h/L	267.5 [181.4, 318.3] (n = 4)	136.5 [103.3, 221.7] (n = 15)	0.469	r_rb 0.27 (-0.53 to 0.87)
Lactate decline 0–72 h, %	53.1 [45.2, 57.0] (n = 4)	55.5 [19.3, 72.0] (n = 18)	0.764	r_rb -0.11 (-0.58 to 0.33)
VV-ECMO duration, days	7.5 [6.2, 20.0] (n = 5)	10.2 [6.1, 17.4] (n = 24)	0.933	r_rb -0.03 (-0.67 to 0.63)
RRT days within 14 days	5.0 [4.0, 12.0] (n = 5)	1.0 [0.0, 4.8] (n = 24)	0.041	r_rb 0.57 (0.19 to 0.88)
ICU length of stay, days	24.0 [22.0, 45.0] (n = 5)	24.0 [14.8, 39.5] (n = 24)	0.705	r_rb 0.12 (-0.40 to 0.63)
Hospital length of stay, days	25.0 [23.0, 59.0] (n = 5)	30.0 [18.2, 57.0] (n = 24)	0.790	r_rb 0.08 (-0.43 to 0.59)
Use of RRT	5 (100.0%) (n = 5)	16 (66.7%) (n = 24)	0.283	RD 0.33 (-0.13 to 0.53)
ICU mortality	0 (0.0%) (n = 5)	5 (20.8%) (n = 24)	0.553	RD -0.21 (-0.40 to 0.24)
Hospital mortality	0 (0.0%) (n = 5)	5 (20.8%) (n = 24)	0.553	RD -0.21 (-0.40 to 0.24)
Verified 28-day mortality	2 (40.0%) (n = 5)	9 (37.5%) (n = 24)	1.000	RD 0.03 (-0.32 to 0.43)
Any recorded bleeding event	4 (80.0%) (n = 5)	9 (37.5%) (n = 24)	0.144	RD 0.43 (-0.04 to 0.66)

Continuous outcomes were compared using two-sided exact conditional rank-permutation tests and are shown with rank-biserial correlation (r_rb) and 95% bootstrap CI. Binary outcomes were compared using Fisher's exact test and are shown with absolute risk difference (RD) and Newcombe–Wilson 95% CI. P values are unadjusted; secondary outcomes are exploratory. AUC analyses required complete scheduled measurements. Abbreviations: AUC, area under the curve; CI, confidence interval; ICU, intensive care unit; RRT, renal replacement therapy; VIS, vasoactive-inotropic score; VFD-28, ventilator-free days through day 28.

Norepinephrine-equivalent AUC from D0 through D7 was 0.89 (IQR, 0.55–1.84) versus 0.76 (IQR, 0.35–2.46) (μg/kg/min)·day (*P* = 0.600; r_rb = − 0.17; 95% CI, − 0.63 to 0.34), and VIS AUC was 89.2 (IQR, 39.7–184.6) versus 68.8 (IQR, 50.8–198.6) score·days (*P* = 0.780; r_rb = − 0.10; 95% CI, − 0.68 to 0.50). Lactate AUC from 0 through 72 hours was 267.5 (IQR, 181.4–318.3) versus 136.5 (IQR, 103.3–221.7) mmol·h/L (*P* = 0.469), and the 72-hour percentage decline was 53.1% (IQR, 45.2%–57.0%) versus 55.5% (IQR, 19.3%–72.0%; *P* = 0.764).

VV-ECMO duration was 7.5 days (IQR, 6.2–20.0) in the VV-ECMO + IABP group and 10.2 days (IQR, 6.1–17.4) in the VV-ECMO-only group (*P* = 0.933). RRT was used in 5/5 (100.0%) and 16/24 (66.7%) patients, respectively (*P* = 0.283; RD, 33.3 percentage points; 95% CI, − 12.8 to 53.3). Median RRT duration within 14 days was 5.0 days (IQR, 4.0–12.0) versus 1.0 day (IQR, 0.0–4.8; *P* = 0.041; r_rb = 0.58; 95% CI, 0.19–0.88).

Verified 28-day mortality was 2/5 (40.0%) in the VV-ECMO + IABP group and 9/24 (37.5%) in the VV-ECMO-only group (*P* = 1.000; RD, 2.5 percentage points; 95% CI, − 32.0 to 42.9). Recorded bleeding occurred in 4/5 (80.0%) and 9/24 (37.5%) patients, respectively (*P* = 0.144; RD, 42.5 percentage points; 95% CI, − 4.3 to 65.6). ICU and hospital mortality were each 0/5 versus 5/24 (20.8%; *P* = 0.553). ICU and hospital lengths of stay are reported in Table 2.

Sensitivity analyses

In the main sensitivity analysis comparing the five VV-ECMO + IABP patients with all eight additional high-dose controls, median VFD-28 was 0.0 days (IQR, 0.0–6.9) versus 0.0 days (IQR, 0.0–8.6; *P* = 1.000; r_rb = − 0.03; 95% CI, − 0.60 to 0.55). Norepinephrine-equivalent AUC was 0.89 (IQR, 0.55–1.84; n = 5) versus 2.01 (IQR, 0.93–2.62; n = 7) (μg/kg/min)·day (*P* = 0.268; r_rb = − 0.43; 95% CI, − 0.94 to 0.26). Verified 28-day mortality was 40.0% versus 37.5% (*P* = 1.000; RD, 2.5 percentage points; 95% CI, − 40.1 to 46.4), and median RRT duration was 5.0 days (IQR, 4.0–12.0) versus 2.0 days (IQR, 0.8–7.2; *P* = 0.266). VIS AUC was not estimated because only one high-dose control had complete, internally consistent VIS data.

In the additional analysis comparing the VV-ECMO + IABP group with the 16 original controls (Tables S3 and S4), median VFD-28 was 0.0 days (IQR, 0.0–6.9) versus 1.4 days (IQR, 0.0–9.9; *P* = 0.662), norepinephrine-equivalent AUC was 0.89 (IQR, 0.55–1.84) versus 0.65 (IQR, 0.33–2.23) (μg/kg/min)·day (*P* = 0.905), verified 28-day mortality was 40.0% versus 37.5% (*P* = 1.000), and median RRT duration was 5.0 versus 0.5 days (*P* = 0.026).

In the analysis restricted to the four additional controls who met the high-dose threshold at D0 (Tables S1 and S2), median VFD-28 was 0.0 days (IQR, 0.0–6.9) versus 6.6 days (IQR, 0.0–14.6; *P* = 0.683), norepinephrine-equivalent AUC was 0.89 (IQR, 0.55–1.84) versus 0.93 (IQR, 0.32–1.89) (μg/kg/min)·day (*P* = 1.000), verified 28-day mortality was 40.0% versus 0.0% (*P* = 0.444), and median RRT duration was 5.0 versus 2.0 days (*P* = 0.190).

Discussion

In this single-center cohort, adjunctive IABP during VV-ECMO was not associated with higher VFD-28, lower norepinephrine-equivalent or VIS AUC, or lower verified 28-day mortality. Patients receiving VV-ECMO + IABP had higher baseline SOFA scores, PEEP, and D0 vasoactive requirements than VV-ECMO-only controls. RRT duration was longer and bleeding was more frequent in the VV-ECMO + IABP group, although the estimates were imprecise and the secondary analyses were unadjusted.

Direct clinical evidence for adding IABP to VV-ECMO remains limited. The principal published report described IABP use during VV-ECMO in individual patients with ARDS and cardiac dysfunction but did not provide a contemporaneous comparator group [16]. Our study extends this evidence by including a VV-ECMO-only comparator; however, the absence of differences in VFD-28, cumulative vasoactive exposure, and mortality should not be interpreted as evidence of equivalence because only five patients received IABP and the confidence intervals were wide.

Evidence from cardiogenic shock and VA-ECMO cannot be directly extrapolated to VV-ECMO. The IABP-SHOCK II trial found no mortality benefit from routine IABP use in myocardial-infarction-related cardiogenic shock, whereas observational VA-ECMO studies and meta-analyses have reported variable associations between concomitant IABP and survival [17–20]. In VA-ECMO, IABP may reduce the LV afterload imposed by retrograde arterial flow. That mechanism is absent during VV-ECMO, in which IABP can augment diastolic pressure and reduce LV afterload but cannot provide direct forward flow. The lack of a clear reduction in vasoactive exposure in our cohort is therefore consistent with the limited circulatory support provided by IABP when vasoplegia, RV failure, or biventricular dysfunction predominates.

The cardiac phenotype is likely to be important when circulatory support is considered during VV-ECMO. RV injury is associated with mortality during VV-ECMO, and recent serial echocardiographic studies have documented persistent or progressive RV abnormalities during support [9, 10, 12–15]. IABP does not directly unload the RV or reduce pulmonary vascular resistance.

The renal and bleeding findings were also evaluated in relation to previous VV-ECMO cohorts. RRT during VV-ECMO is associated with severe multiorgan dysfunction and poor long-term outcomes [25]. Accordingly, the longer RRT duration observed in the VV-ECMO + IABP group may reflect greater baseline severity rather than an effect of IABP. Bleeding occurred in 80.0% of the VV-ECMO + IABP group and 37.5% of the VV-ECMO-only group. In the international PROTECMO cohort, 52.5% of 652 patients had at least one bleeding event, and ELSO registry analyses have associated hemorrhagic and thrombotic complications with adverse outcomes during VV-ECMO [26, 27]. Because bleeding events were identified retrospectively and were not prospectively adjudicated, device-attributable bleeding could not be distinguished from bleeding related to ECMO, anticoagulation, cannulation, or critical illness.

The sensitivity analyses did not identify a consistent association between IABP use and VFD-28 or 28-day mortality. In the comparison with all eight additional high-dose controls, cumulative norepinephrine-

equivalent exposure was numerically lower in the VV-ECMO + IABP group, but the confidence interval was wide and one control lacked complete data. Moreover, four of these controls first met the high-dose threshold after D0; selection on post-baseline vasopressor exposure may introduce survivor and time-dependent selection bias. The analyses restricted to the 16 original controls and the four baseline high-dose controls yielded similarly imprecise estimates. These comparisons should therefore be regarded as sensitivity analyses rather than independent confirmation of a treatment effect.

This study has several limitations. The retrospective single-center design and the inclusion of only five patients in the VV-ECMO + IABP group limited statistical precision and precluded reliable multivariable or propensity-score adjustment. The indication and timing of IABP were not standardized, and residual confounding by cardiac phenotype and illness severity is likely. Serial echocardiographic measures, cardiac output, RV–pulmonary arterial coupling, and several biomarkers were incomplete, preventing phenotype-specific analyses. VFD-28 was derived from source-recorded ventilation duration and vital status rather than from daily prospective assessment. Bleeding events were not adjudicated using a standardized severity definition. Finally, secondary and sensitivity analyses were exploratory and were not adjusted for multiple comparisons.

Conclusions

In this small retrospective cohort, adjunctive IABP during VV-ECMO was not associated with higher VFD-28, lower cumulative vasoactive exposure, or lower 28-day mortality. The marked baseline differences and wide confidence intervals preclude conclusions regarding benefit or harm. Prospective studies incorporating standardized cardiac phenotyping and predefined criteria for escalation of circulatory support are required.

Declarations

Ethics approval and consent to participate

The study was approved by the Medical Ethics Committee of Dongguan People's Hospital (approval no. IRBIITKY-2026-268-01).

Clinical trial number

Not applicable.

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests

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Author Contribution

W.J. designed the study, collected and analysed the data, and drafted the manuscript. G.Y. and S.H.Q. assisted with data collection. W. J. and H.H.B. performed data analysis. H.H.B. assisted with manuscript preparation. S.L.H. and Z. Z.H. critically revised the manuscript. All authors read and approved the final manuscript.

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Not applicable

Data Availability

The datasets generated and analysed during the current study are not publicly available owing to patient privacy and ethical restrictions. Anonymized data may be available from the corresponding author upon reasonable request and after formal approval by the Ethics Committee of The Tenth Affiliated Hospital of Southern Medical University (Dongguan People's Hospital).

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Figures

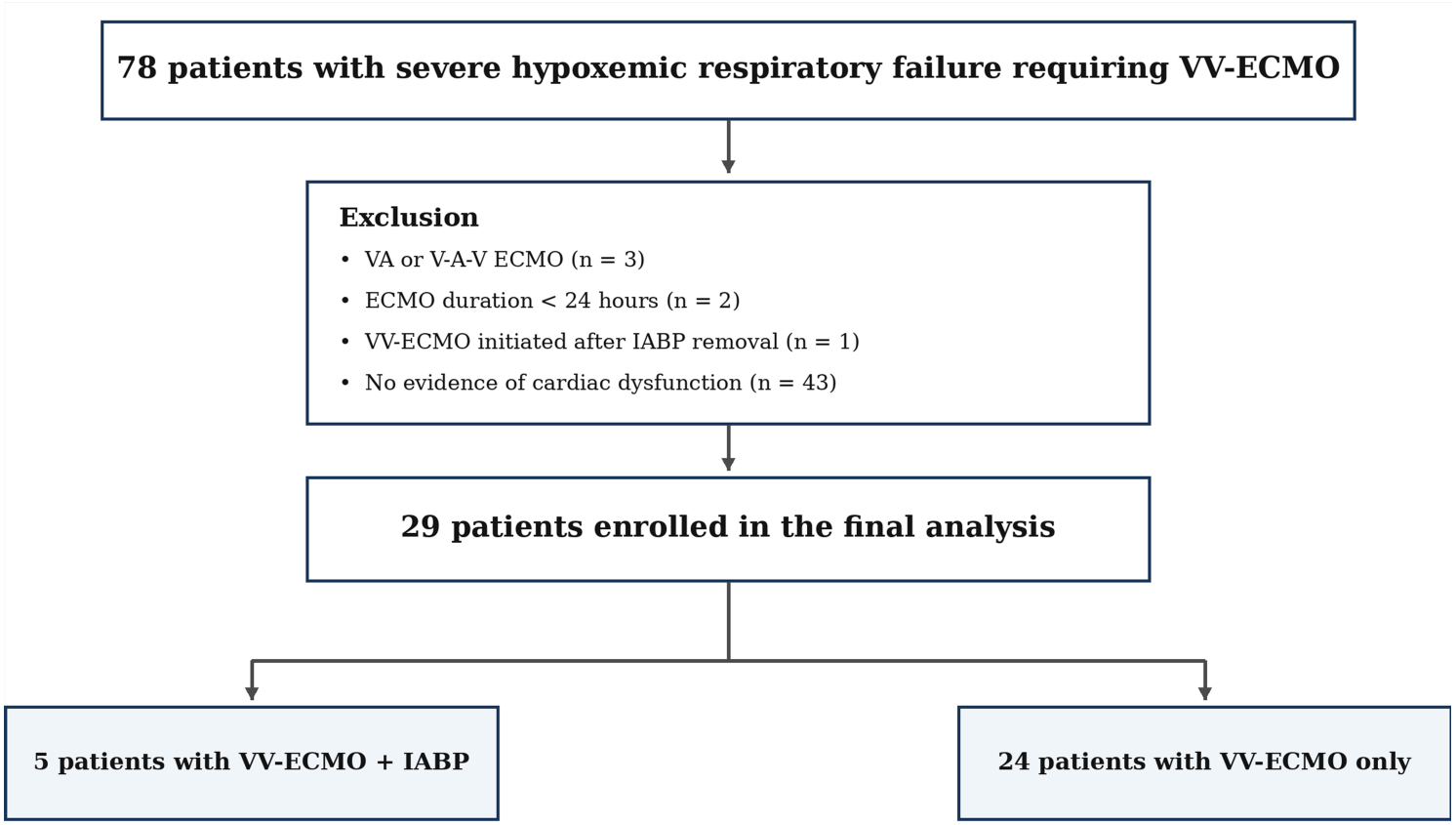


Figure 1

Flow chart of study cohort.

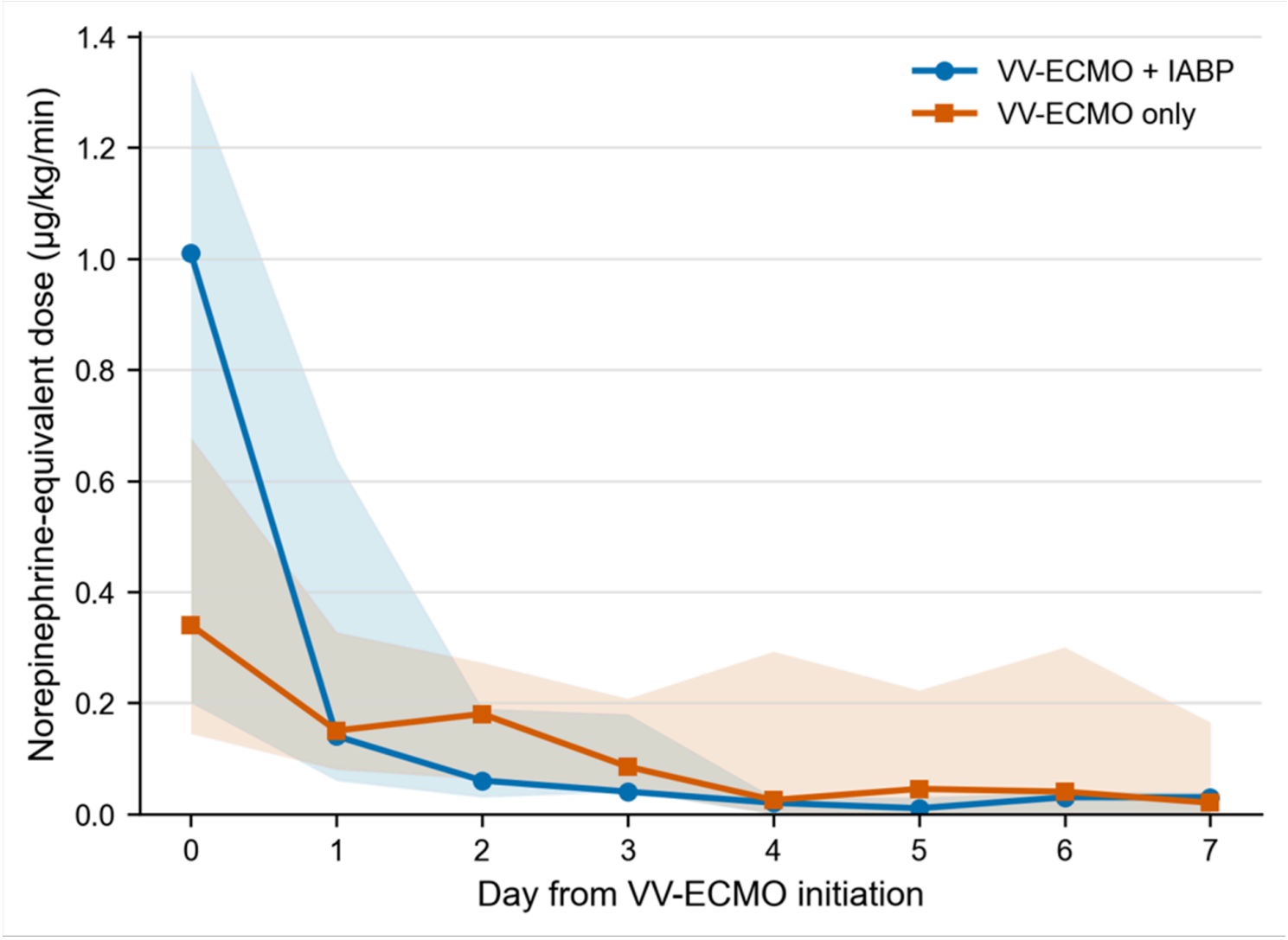


Figure 2

Thenorepinephrine-equivalent dose trajectory.

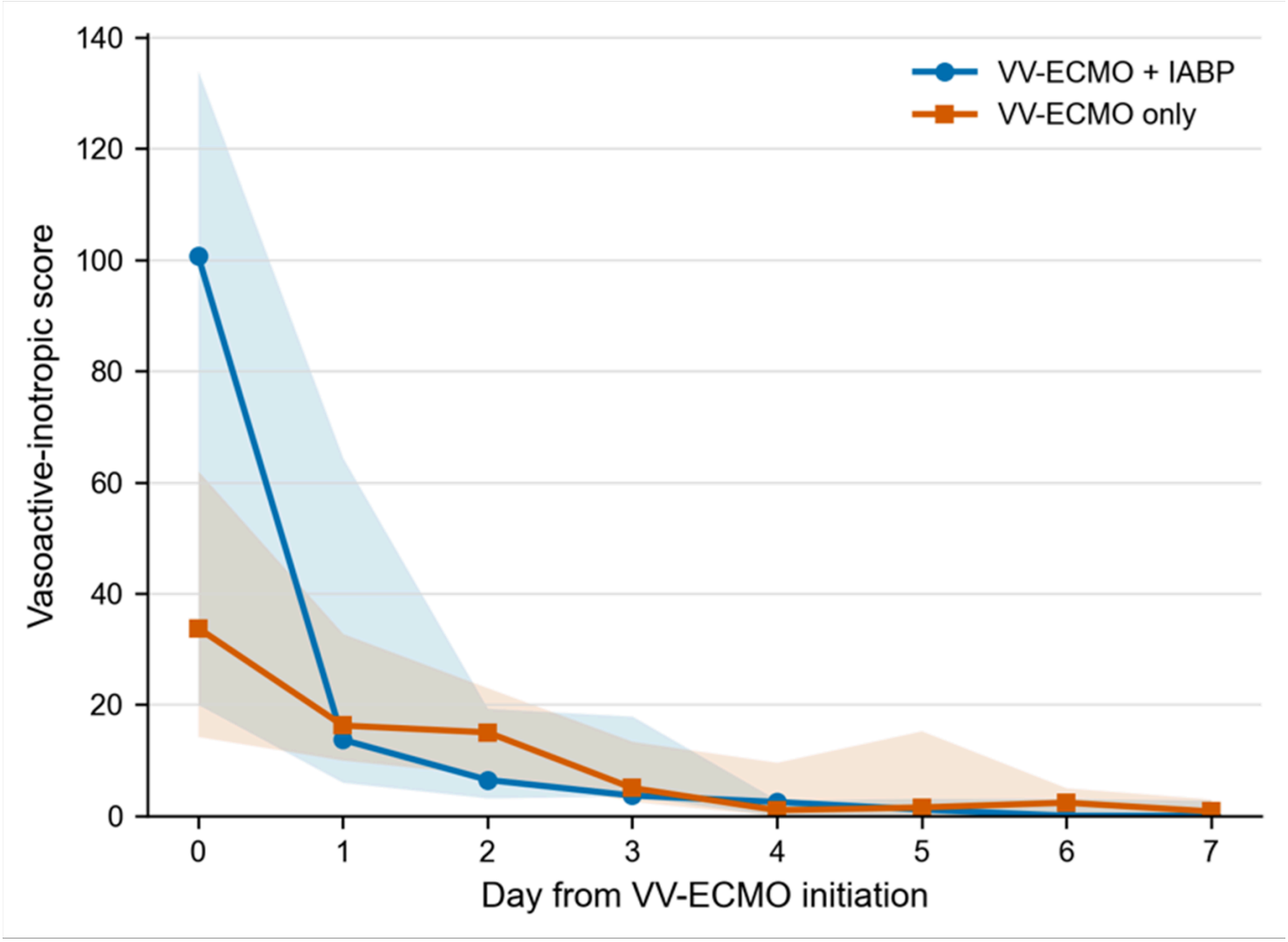


Figure 3

Seven-day vasoactive-inotropic score trajectory.

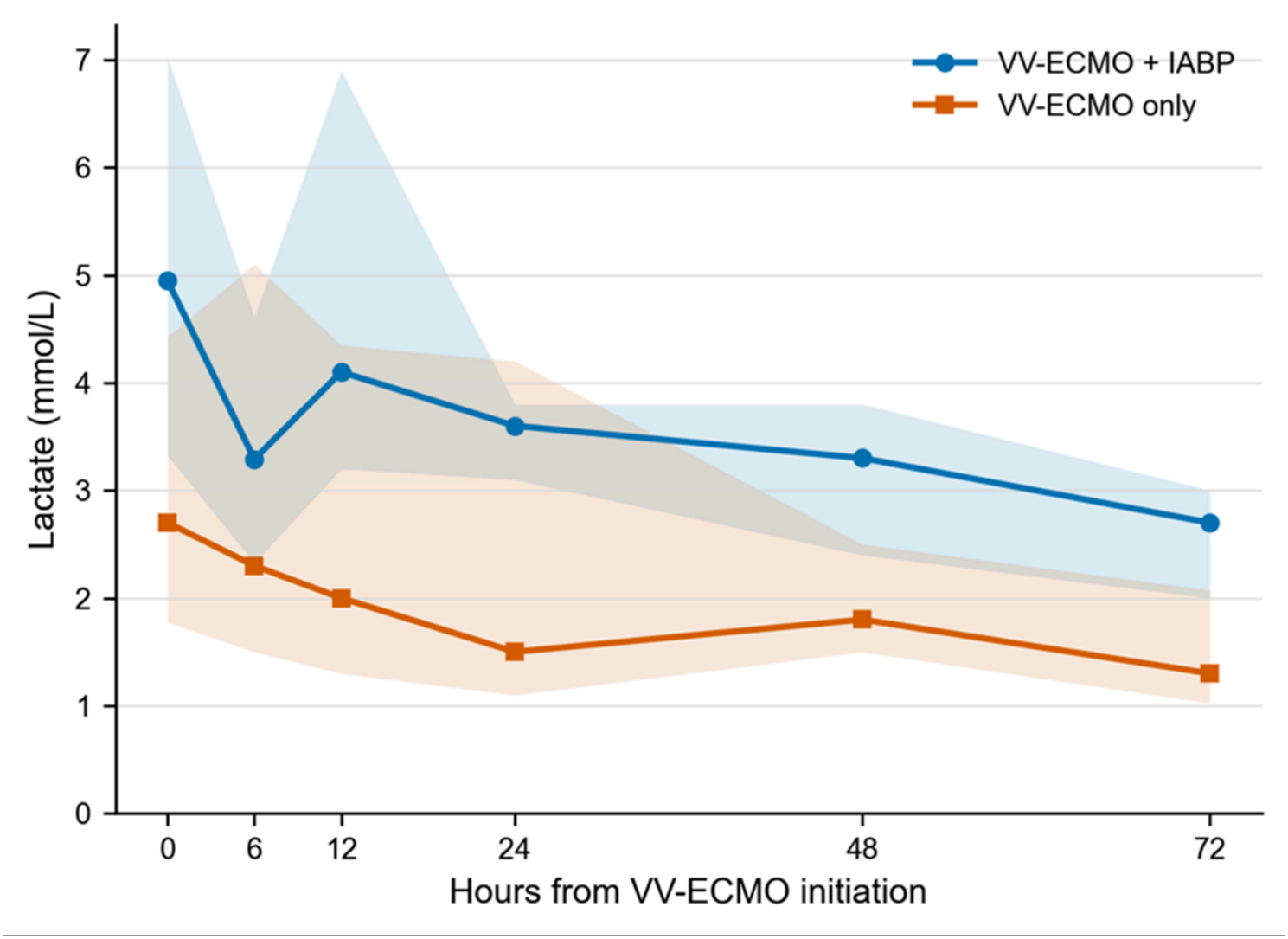


Figure 4

Lactate trajectory during the first 72 hours.

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