

Investigation of Neurological Outcomes in Critically Ill Patients Under Extracorporeal Membrane Oxygenation (ECMO) Treatment: Retrospective Observational Study

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ABSTRACT

Background: With the increasing use of extracorporeal membrane oxygenation (ECMO), neurological complications are being encountered more frequently during patient follow-up. These complications significantly contribute to morbidity and mortality, underscoring the need for further investigation into preventive strategies.

Aim: Neurological complications are common in patients receiving venoarterial ECMO (VA-ECMO); however, their incidence, spectrum, and prognostic significance remain incompletely characterized. This study aimed to identify risk factors, predictors of mortality, and neurological outcomes in comatose ECMO patients.

Study Design: Retrospective observational study.

Methods: We conducted a retrospective analysis of patients admitted to a cardiovascular surgery intensive care unit between January 2019 and January 2024 who received ECMO support.

Results: Among 189 patients, 31 (16.4%) developed neurological complications: 20 (64.5%) hypoxic-ischemic encephalopathy (HIE), 9 (29.0%) ischemic cerebrovascular events, and 2 (6.5%) hemorrhagic cerebrovascular events. Mortality was highest among patients with HIE (73.7%). Multivariable logistic regression identified two independent predictors of mortality: Glasgow Coma Scale (GCS) score <8 (OR = 77.25, 95% CI: 15.06–396.09, p < 0.001) and the presence of neurological complications (OR = 5.95, 95% CI: 1.35–26.25, p = 0.019).

Conclusions: Neurological complications, particularly hypoxic-ischemic encephalopathy, are frequent in VA-ECMO patients and are strongly associated with increased mortality. These findings highlight the importance of systematic neurological monitoring and early preventive strategies in this high-risk population.

Keywords: Extracorporeal membrane oxygenation, acute brain injury, neurological outcomes, sedation

ÖZET

Arka plan: Ekstrakorporel membran oksijenasyonunun (ECMO) kullanımının artmasıyla birlikte, hastaların takibi sırasında nörolojik komplikasyonlarla karşılaşmaktadır ve bu komplikasyonların önlenmesi için daha fazla araştırmaya ihtiyaç duyulmaktadır; bu komplikasyonlar bu hasta gruplarında mortaliteyi önemli ölçüde etkilemektedir.

Amaç: Venoarteriyel ekstrakorporel membran oksijenasyonu (VA-ECMO) alan hastalarda nörolojik komplikasyonlar sık görülmekle birlikte, bunların sıklığı, tipleri ve prognostik etkileri tam olarak tanımlanmamıştır. Bu çalışma, komada olan ECMO hastalarında mortalite belirleyicilerini, risk faktörlerini ve nörolojik sonuçları belirlemeyi amaçlamıştır.

Çalışma Tasarımı: Retrospektif gözlemsel çalışma

Yöntemler: Ocak 2019 ile Ocak 2024 tarihleri arasında kardiyovasküler cerrahi yoğun bakım ünitesine yatırılan ve ECMO desteği uygulanan hastaların retrospektif gözlemsel bir çalışmasını gerçekleştirdik.

Sonuçlar: 189 hastanın 20'sinde (%64,5) hipoksik-iskemik ensefalopati, 9'unda (%29,0) iskemik serebrovasküler hastalık ve 2'sinde (%6,5) hemorajik serebrovasküler hastalık gelişti. Ölüm oranları hipoksik-iskemik ensefalopati gelişen hastalarda en yüksekti (%73,7). Lojistik regresyon, mortalitenin iki bağımsız belirleyicisini tanımladı: Glasgow Koma Ölçeği (GCS) <8 (OR = 77.25, %95 CI: 15.06–396.09, p < 0.001) ve nörolojik komplikasyonların varlığı (OR = 5.95, %95 CI: 1.35–26.25, p = 0.019).

Nörolojik komplikasyonlar, özellikle hipoksik-iskemik ensefalopati, VA-ECMO hastalarında yaygındır ve artmış mortalite ile güçlü bir şekilde ilişkilidir. Bu bulgular, bu yüksek riskli popülasyonda sistematik nörolojik izleme ve önleyici stratejilere duyulan ihtiyacın altını çizmektedir.

Anahtar Kelimeler: Ekstrakorporel membran oksijenasyonu, akut beyin hasarı, nörolojik sonuçlar, sedasyon

Extracorporeal membrane oxygenation (ECMO) was first successfully applied in humans by Robert Bartlett in the early 1970s. Over the past five decades, substantial technological advancements have improved survival rates and broadened the clinical indications for ECMO. Consequently, its utilization has increased markedly, and prolonged support is now more frequently implemented (1).

Despite these advances, venoarterial ECMO (VA-ECMO) remains associated with a high incidence of neurological, hemorrhagic, and thrombotic complications. In-hospital mortality rates have been reported to range between 50% and 70% (2). Neurological complications are particularly significant, with an incidence of approximately 13.3% in adult VA-ECMO patients; among these, 5.9–7.8% represent ischemic and/or hemorrhagic stroke (3). Clinically, these complications may manifest as altered consciousness, seizures, coma, or loss of brainstem reflexes. In many cases, intracranial hemorrhage or infarction, rather than primary cardiac or pulmonary pathology, constitutes the ultimate cause of death.

Neurological injury in ECMO patients is multifactorial. Contributing factors include pre-ECMO conditions such as hypotension, hypoxia, acidosis, electrolyte imbalance, and coagulopathy; reperfusion injury during cannulation; embolic events originating from the arterial cannula; and ECMO-induced coagulation disturbances (4). The underlying mechanisms remain incompletely understood but likely involve a combination of these processes.

Because neurological injury may remain clinically silent until advanced stages, cranial computed tomography (CT) plays a crucial role in detecting intracranial abnormalities, including non-hemorrhagic infarctions, diffuse cerebral edema, and cerebral atrophy (5). Neuromonitoring may facilitate early detection of brain injury; however, its application is often limited by the critical condition and frequent comatose state of ECMO patients (6).

Acute brain injury (ABI), including ischemic stroke, intracranial hemorrhage, and hypoxic–ischemic brain injury, has been reported in up to 20% of adult VA-ECMO patients according to the Extracorporeal Life Support Organization (ELSO) registry (7). Given the increasing use of ECMO and the growing recognition of ABI, identifying modifiable risk factors—such as hypoxia, reduced pulse pressure, and hypercarbia—is essential.

Persistent coma in ECMO patients may reflect ABI, metabolic disturbances, or the effects of sedative agents, complicating clinical interpretation (8). Therefore, this study aimed to evaluate neurological complications in critically ill ECMO patients, determine the frequency

and types of ABI, assess their impact on mortality, and investigate the predictive roles of blood gas parameters, GCS, ECMO duration, and sedation practices.

Materials and Methods

Study Design and Participants

This retrospective observational study was conducted by reviewing medical records of patients admitted to the cardiovascular surgery intensive care unit between January 1, 2019, and January 1, 2024. Patients aged ≥ 18 years who received ECMO support during hospitalization were eligible for inclusion.

Study Procedures

Neurological status was assessed using the Glasgow Coma Scale (GCS) over a 24-hour period during ECMO support when patients were not receiving continuous sedative infusions (except dexmedetomidine) or neuromuscular blocking agents.

“Off-sedation coma” was defined as a GCS score < 8 persisting during the first 24 hours following discontinuation of sedation. Patients with GCS ≥ 8 were classified as “off-sedation non-coma.”

Neurological examinations were performed daily following sedation cessation and included GCS scoring and pupillary reflex assessment. Nursing staff monitored pupil size and reactivity every 4 hours. Clinical events such as seizures, delirium, confusion, and withdrawal syndrome were documented. In cases of mydriasis or anisocoria, cranial CT was performed to evaluate for acute intracranial pathology.

During ECMO support, new-onset neurological complications were assessed radiologically using cranial CT and categorized as ischemic stroke, hemorrhagic stroke, cerebral edema, or hypoxic–ischemic encephalopathy. Seizures were recorded as clinical outcomes.

Collected variables included GCS, worst arterial blood gas parameters within 24 hours prior to ECMO initiation (PaO₂, PaCO₂, pH, bicarbonate, lactate), ECMO duration, duration of sedation, neurological outcomes (assessed using the modified Rankin Scale), and in-hospital mortality.

Inclusion and Exclusion Criteria

Patients aged ≥ 18 years who received ECMO support during the study period were included. Patients with incomplete neurological or outcome data were excluded.

Sample Size

Based on institutional data indicating approximately 50 ECMO cases annually, a minimum sample size of 135 patients was estimated for a 5-year retrospective analysis ($\alpha = 0.05$, power = 95%, standard deviation = 1).

Statistical Analysis

Continuous variables were expressed as mean \pm standard deviation (SD) for normally distributed data and median (interquartile range [IQR]) for non-normally distributed data. Categorical variables were presented as frequencies and percentages.

Group comparisons were performed using the Mann-Whitney U test for non-parametric variables, with effect sizes reported as rank-biserial correlation coefficients.

Binary logistic regression analysis was performed to identify independent predictors of mortality and neurological complications. All categorical variables were entered into the model using binary coding, with clinically relevant reference categories (e.g., GCS ≥ 8) defined a priori. The selection of variables for the multivariable

model was based on clinical relevance and univariate analysis results.

Model performance was evaluated using the Nagelkerke R^2 and the area under the receiver operating characteristic curve (ROC-AUC). Odds ratios (ORs) with 95% confidence intervals (CIs) were reported. Multiple linear regression analysis was conducted to evaluate predictors of ECMO duration. Statistical significance was defined as $p < 0.05$. All analyses were performed using SPSS Statistics version 26, and only complete cases were included.

Results

Participant Characteristics

A total of 189 patients were included in the analysis. Neurological complications developed in 31 patients (16.4%) during ECMO support. Among these, 20 patients (64.5%) were diagnosed with hypoxic-ischemic encephalopathy (HIE), 9 (29.0%) with ischemic cerebrovascular events, and 2 (6.5%) with hemorrhagic cerebrovascular events (Figure 1).

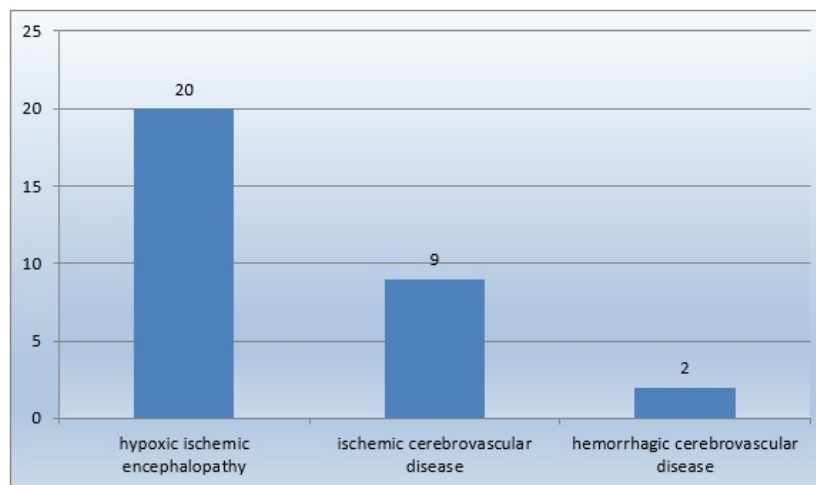


Figure 1: Different types of ABI in patients on ECMO

Patients who developed neurological complications were younger (mean 56.1 ± 13.3 years) compared to those without complications (mean 60.2 ± 11.8 years), although this difference did not reach statistical significance ($p = 0.104$).

A significant difference was observed in intensive care unit length of stay. Patients with neurological complications had a markedly shorter duration of hospitalization

(median 4.0 days, IQR 2.0–8.0) compared to those without complications (median 16.0 days, IQR 9.0–25.0; $p < 0.001$), corresponding to a large effect size ($r = 0.585$) (Table 1).

APACHE II scores were slightly higher in the complication group (median 29.0 [26.0–31.0]) compared to the non-complication group (median 28.0 [25.0–29.0]), with this difference reaching statistical significance ($p = 0.002$; $r = 0.347$).

No statistically significant differences were observed between groups in terms of physiological parameters, including pH (7.38 vs 7.31; $p = 0.361$), arterial oxygen tension (125.0 vs 146.0 mmHg; $p = 0.408$), or lactate levels (3.0 vs 6.0 mmol/L; $p = 0.331$).

Comorbidity burden, renal function, hepatic function, sepsis incidence, GCS scores, sedation practices, and history of cardiac arrest were comparable between groups (all $p > 0.05$).

Patients without neurological complications had a longer median ECMO duration (5.0 days, IQR 3.0–6.0) compared to those with complications (3.0 days, IQR 2.0–6.0), although this difference was not statistically significant ($p = 0.091$; $r = 0.190$). This finding may reflect early termination of ECMO due to neurological deterioration in affected patients.

Overall, significant differences between groups were observed for APACHE II scores and hospital length of stay, while other variables did not demonstrate statistically significant variation.

Table 1. Descriptive statistics and group comparisons by complication status

Variable	Neurological Complications (n=31)	No Complications (n=158)	p-value	Effect Size (r)
<i>Mean ± SD or Median (IQR)</i>				
Age (years)	56.1 ± 13.3	60.2 ± 11.8	0.104	0.185
Sex (n, %)			0.504	
Male	21, 67.7%	97, 61.4%		
Female	10, 32.3%	61, 38.6%		
APACHE II	29.0 (26.0–31.0)	28.0 (25.0–29.0)	0.002*	0.347
Mortality			<0.001***	
Yes	20, 64.5%	17, 10.8%		
No	11, 35.5%	141, 89.2%		
ICU Hospital stay (days)	4.0 (2.0–8.0)	16.0 (9.0–25.0)	<0.001***	0.585
Hepatic function			0.997	
Increased	20, 64.5%	102, 64.6%		
Normal	11, 35.5%	56, 35.4%		
Renal function	58.0 (40.0–80.0)	58.0 (42.0–80.0)	0.726	0.040
Comorbidity count	1.0 (1.0–1.0)	1.0 (1.0–2.0)	0.122	0.139
Sepsis			0.191	
Yes	14, 45.2%	52, 32.9%		
No	17, 54.8%	106, 67.1%		
GCS			0.065	
≥8	10, 32.3%	28, 17.7%		
<8	21, 67.7%	130, 82.3%		
Sedation			0.406	
Yes	19, 61.3%	84, 53.2%		
No	12, 38.7%	74, 46.8%		
Arrest			0.574	
Yes	1, 3.2%	9, 5.7%		
No	30, 96.8%	149, 94.3%		
ECMO duration (days)	3.0 (2.0–6.0)	5.0 (3.0–6.0)	0.091	0.190
pH	7.31 (7.24–7.39)	7.38 (7.25–7.42)	0.361	0.104
PO₂ (mmHg)	146.0 (100.0–190.0)	125.0 (95.0–170.0)	0.408	0.094
PCO₂ (mmHg)	34.0 (31.0–38.0)	35.0 (30.0–40.0)	0.616	0.057
HCO₃ (mmol/L)	19.0 (16.0–22.0)	20.0 (16.0–23.0)	0.618	0.057
Lactate (mmol/L)	6.0 (3.0–8.0)	3.0 (2.0–7.0)	0.331	0.109

* $p < 0.05$; *** $p < 0.001$. Bold p-values indicate statistical significance. Percentages are calculated within each column (column percentages).

Binary Logistic Regression Analysis Results

Mortality Predictors in ECMO Patients

A binary logistic regression analysis was performed to identify factors associated with mortality in patients receiving ECMO support. The model included nine predictor variables, encompassing demographic characteristics, clinical parameters, and complication profiles. The overall model was statistically significant ($\chi^2(9) = 78.0, p < 0.001$) and demonstrated good model fit, with a Nagelkerke R^2 of 0.611 and an Akaike Information Criterion (AIC) of 98.0.

The analysis identified two independent predictors of mortality. Patients with a GCS score <8 had markedly

increased odds of mortality compared to those with $GCS \geq 8$ (OR = 77.25, 95% CI: 15.06–396.09, $p < 0.001$), indicating that severely impaired neurological status is a strong prognostic factor. Additionally, the presence of neurological complications was significantly associated with an increased risk of mortality (OR = 5.95, 95% CI: 1.35–26.25, $p = 0.019$) (Table 2).

Other variables, including age, renal dysfunction, ECMO duration, need for haemodialysis, sex, sedation exposure, and history of cardiac arrest, were not significantly associated with mortality in this model (all $p > 0.05$).

The model demonstrated excellent discriminative performance, with a sensitivity of 75.0%, specificity of 96.9%, and an area under the receiver operating characteristic curve (ROC-AUC) of 0.910.

Table 2. Binomial Logistic Regression Predicting Mortality (vs. Survival) in ECMO Patients

Predictor	B (Estimate)	SE	Z	p-value	Odds Ratio	95% CI for OR
Intercept	0.010	2.653	0.004	0.997	1.01	[0.006, 183.13]
Age	-0.011	0.025	-0.418	0.676	0.99	[0.942, 1.040]
Renal function	0.009	0.012	0.712	0.476	1.01	[0.985, 1.033]
ECMO duration	-0.112	0.080	-1.402	0.161	0.89	[0.764, 1.046]
GCS <8 (vs. ≥ 8)	4.347	0.834	+5.215	<0.001	77.25	[15.06, 396.09]
Haemodialysis (Yes)	1.428	0.933	1.530	0.126	4.17	[0.669, 25.960]
Gender (M vs. F)	-0.495	0.658	-0.752	0.452	0.61	[0.168, 2.215]
Neurological complications	1.783	0.758	2.354	0.019	5.95	[1.348, 26.253]
Sedation (Yes)	-0.344	0.820	-0.419	0.675	0.71	[0.142, 3.539]
Cardiac arrest (Yes)	0.108	1.759	0.062	0.951	1.11	[0.035, 34.995]

Model fit: $\chi^2(9) = 78.0, p < 0.001; AIC = 98.0$
Pseudo R^2 : Nagelkerke $R^2 = 0.611; Cox \& Snell R^2 = 0.508$
Prediction performance: Sensitivity = 75.0%, Specificity = 96.9%, AUC = 0.910
 Note: Bold values indicate statistical significance at $p < 0.05$. OR = Odds Ratio; CI = Confidence Interval; B = regression coefficient; SE = standard error; Z = Wald statistic.

Factors Influencing ECMO Duration

Multiple linear regression analysis identified several significant predictors of ECMO duration. Total hospital length of stay emerged as the strongest positive predictor, with each additional hospital day associated with a 0.15-day increase in ECMO support duration ($p < 0.001$). In contrast, higher APACHE II scores were significantly associated with shorter ECMO duration ($p = 0.007$), reflecting an inverse relationship between illness severity and treatment duration. This finding likely indicates that

patients with higher APACHE II scores experienced more severe disease and earlier mortality, resulting in reduced ECMO support time.

Clinical complications were also significantly associated with shorter ECMO duration. Patients who developed neurological complications required approximately 1.8 fewer days of ECMO support ($p = 0.017$), while those with sepsis or shock required 3.3 fewer days ($p < 0.001$). Mortality was similarly associated with shorter ECMO duration compared to survival ($p = 0.043$). A trend toward

reduced ECMO duration was observed in patients with cardiac arrest; however, this did not reach statistical significance ($p = 0.062$).

In contrast, standard biochemical markers—including arterial blood gas parameters (PaO_2 : $p = 0.712$; PaCO_2 : $p = 0.725$), lactate levels ($p = 0.952$), and renal function

($p = 0.171$)—were not significant predictors of ECMO duration. Overall, the model explained 45.2% of the variance in ECMO support duration, suggesting that clinical factors, rather than laboratory parameters, play a more substantial role in determining ECMO weaning decisions in this patient population.

Table 3. Multiple Linear Regression Predicting ECMO Duration

Predictor	B (Estimate)	SE	95% CI	T	p-value
Intercept	-14.509	29.807	[-73.21, 44.19]	-0.487	0.627
Total hospital stay (Day)	0.145	0.027	[0.092, 0.198]	5.418	<0.001
APACHE II score	-0.283	0.103	[-0.486, -0.080]	-2.748	0.007
Neurological complication (Yes)	-1.793	0.747	[-3.265, -0.321]	-2.399	0.017
Sepsis/Shock (Yes)	-3.252	0.611	[-4.457, -2.047]	-5.319	<0.001
Mortality	-2.108	1.032	[-4.144, -0.072]	-2.042	0.043
Cardiac Arrest (Yes)	-2.157	1.150	[-4.424, 0.110]	-1.877	0.062
Renal dysfunction	0.013	0.009	[-0.005, 0.031]	1.375	0.171
pH	5.472	4.108	[-2.629, 13.573]	1.332	0.185
PO_2	0.001	0.003	[-0.005, 0.007]	0.370	0.712
PCO_2	0.014	0.039	[-0.063, 0.091]	0.352	0.725
HCO_3	-0.062	0.113	[-0.285, 0.161]	-0.546	0.586
Lactate	0.005	0.077	[-0.147, 0.157]	0.060	0.952
Sedation (Yes)	-0.180	0.514	[-1.195, 0.835]	-0.351	0.726

Model summary: $R^2 = 0.452$, Adjusted $R^2 = 0.411$, $F(13, 173) = 10.97$, $p < 0.001$. Bold indicates statistical significance at $p < 0.05$.

Discussion

This study represents a relatively large single-center analysis of neurological complications in patients undergoing VA-ECMO. Among 189 patients, 31 (16.4%) developed neurological complications, most commonly hypoxic-ischemic encephalopathy (HIE), followed by ischemic cerebrovascular disease and hemorrhagic cerebrovascular disease. Our analysis identified two strong independent predictors of mortality: a GCS score <8 and the presence of neurological complications. These findings highlight the prognostic importance of neurological status in ECMO patients and reinforce previous evidence that poor neurological function is a powerful determinant of outcome (8).

Neurological complications and mortality

The overall mortality in our cohort was 19.6% (37/189). Among patients with neurological complications, mortality was markedly higher at 64.5% (20/31), compared to 10.8% (17/158) in patients without neurological complications. Mortality was particularly high in patients with HIE (73.7%). These findings are consistent with prior studies demonstrating that ECMO, although life-saving, carries a substantial risk of acute brain injury, including ischemic and hemorrhagic stroke as well as hypoxic-ischemic injury (9–10). Similarly, Feng et al. reported that a GCS score <8 was significantly associated with increased mortality and poorer neurological outcomes in ECMO patients (8).

Pathophysiology of acute brain injury in ECMO

Multiple mechanisms contribute to acute brain injury (ABI) during ECMO support. Hypoxic–ischemic injury may occur due to pre-ECMO cardiac arrest, hypoperfusion, or prolonged hypoxia prior to cannulation (10,15). In our study, patients with HIE frequently had cardiac arrest before ECMO initiation, with cardiopulmonary resuscitation (CPR) durations of 30–45 minutes. This finding supports hypoxia as a key predisposing factor. Consistent with our findings, Iacobelli et al. reported that 22% of patients with pre-ECMO cardiac arrest developed cerebral infarction (15).

Ischemic stroke during ECMO may also result from cerebral hypoperfusion, embolic phenomena, or reperfusion injury. ECMO induces a prothrombotic state through contact activation of the coagulation cascade, while anticoagulation therapy simultaneously increases bleeding risk (11,12). This complex hemostatic imbalance predisposes patients to both ischemic and hemorrhagic complications. Sepsis, renal failure, and thrombocytopenia further increase the risk of intracranial hemorrhage. In our cohort, the patient with hemorrhagic cerebrovascular disease (HCD) had a platelet count $<20,000/\mu\text{L}$, highlighting the role of severe thrombocytopenia. The mandatory use of anticoagulation during ECMO may further exacerbate this vulnerability (13).

Hemorrhagic complications

Cavayas et al. demonstrated that intracranial hemorrhage is associated with poor prognosis in ECMO patients, with risk factors including sepsis, renal failure, and thrombocytopenia (13). Our findings are consistent with these observations, as both anticoagulation exposure and severe thrombocytopenia were present in the patient who developed HCD. Given these risks, proactive platelet management strategies and early neuroimaging surveillance may help mitigate secondary neurological injury (13).

Hemodynamic instability and autoregulation

Another important mechanism underlying ABI is impaired cerebral autoregulation in ECMO patients. Hemodynamic instability, vasopressor requirements, and deep sedation may reduce mean arterial pressure, leading to cerebral hypoperfusion (11,14). Additionally, the direct connection of the ECMO circuit to the arterial system bypasses the

pulmonary filter, potentially allowing embolic material to reach the cerebral circulation (11). Our findings of early neurological deterioration and shortened ECMO duration in patients with complications likely reflect these mechanisms.

Hyperoxia and reperfusion injury

Hyperoxia during ECMO initiation has also been implicated in ischemic stroke development due to oxidative stress and reperfusion injury (11,16,17). In our study, mild hyperoxia was observed in patients with ischemic stroke; however, this association did not reach statistical significance. Previous studies by Munshi et al. and Jentzer et al. have reported inconsistent associations between hyperoxia and mortality in ECMO patients (16,17).

Lactate as a prognostic marker

Elevated lactate levels have been proposed as predictors of ischemic stroke during ECMO. Omar et al. and Sutter et al. demonstrated that pre-ECMO lactate levels >10 mmol/L were independently associated with ischemic cerebrovascular events (18,19). In our cohort, patients with HIE exhibited higher lactate levels prior to ECMO initiation, supporting lactate as a potential marker of impaired cerebral perfusion and subsequent brain injury.

Length of stay and disease severity

Interestingly, patients with neurological complications had significantly shorter ICU stays (median 4 days) compared to those without complications (median 16 days). This finding contrasts with prior studies reporting prolonged hospitalization in patients with ischemic or hemorrhagic stroke (20). In our cohort, the shorter length of stay likely reflects early mortality due to severe neurological deterioration. Additionally, patients with complications had significantly higher APACHE II scores, supporting previous evidence that disease severity is a strong predictor of poor outcomes and mortality (21).

Clinical implications

Our findings reinforce that neurological complications are not only frequent but also highly lethal in ECMO patients. A GCS score <8 and the presence of ABI are early and clinically accessible predictors of mortality. Therefore, early neurological assessment, continuous neuromonitoring, and targeted management of modifiable risk factors (e.g.,

thrombocytopenia, sepsis, and hyperoxia), along with individualized anticoagulation strategies, are essential to improve patient outcomes.

Conclusion

Extracorporeal membrane oxygenation (ECMO) has become an essential life-support modality for patients with severe cardiac and pulmonary failure; however, its use is associated with substantial morbidity and mortality. Neurological complications—including ischemic stroke, intracranial hemorrhage, and hypoxic–ischemic brain injury—remain among the most severe adverse events and are strongly associated with increased mortality.

In this study, neurological complications occurred in 16.4% of ECMO patients, with hypoxic–ischemic encephalopathy representing the most common and most fatal subtype. A GCS score <8 and the presence of neurological complications emerged as independent predictors of mortality. These findings emphasize the prognostic value of neurological assessment and underscore the need for routine, systematic monitoring in this high-risk population.

With the continued expansion of ECMO utilization, further research is urgently needed to develop strategies for early detection, prevention, and management of neurological complications. Such efforts are critical to improving survival and long-term neurological outcomes in critically ill patients requiring ECMO support.

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Declarations

Funding

None.

Conflict of Interest

The authors declare no competing interest.

Ethics Approval

This study was approved by the Ethics Committee of Istanbul Medipol University (Date: 09.01.2025, Decision No: 65, Document No: E-10840098-202.3.02-635).

Availability of Data and Material

Available from the corresponding author upon reasonable request.

Author Contributions

AGKK: Conceptualization, Methodology, Writing original draft. ZY: Conceptualization, Methodology, Data curation. CIA: Writing (review & editing).

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