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ORIGINAL RESEARCH

The impact of mild and moderate to severe acute gastrointestinal injury on disease prognosis in ECMO-supported patients

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Abstract

Backgrounds: This study aims to evaluate the impact of mild and moderate-to-severe acute gastrointestinal injury (AGI) on clinical indicators and prognosis of patients undergoing extracorporeal membrane oxygenation (ECMO). **Methods**: From the existing case records, a retrospective analysis was conducted using the clinical data of 105 ECMO-supported patients treated at our hospital between January 2023 and October 2024. Based on AGI grades, patients were categorized into two groups: Group A (mild injury, Grade I + II, n = 55) and Group B (moderate-to-severe injury, Grade III + IV, n = 50). Both groups received ECMO support, a standardized gastrointestinal nutrition strategy, and identical treatments aimed at symptom relief. **Results**: Comparisons between the two groups revealed that inflammatory markers at timepoints T0–T4 were significantly lower in Group A than in Group B ($p < 0.05$), with less fluctuation in Group A ($p > 0.05$) but greater fluctuation in Group B ($p < 0.05$). Between T0–T4, systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP) were significantly higher in Group A, while central venous pressure (CVP) was lower in Group A compared to Group B ($p < 0.05$). Additionally, Group A exhibited smaller changes in hemodynamic indicators ($p > 0.05$), while Group B showed greater fluctuations ($p < 0.05$). Regarding outcomes, Group A had higher ECMO weaning success and discharge rates, while lower mortality compared to Group B ($p < 0.05$). Furthermore, complications, rehospitalization and disease recovery rate in Group A were significantly better than in Group B. **Conclusions**: Mild AGI (Grade I + II) has a lesser impact on inflammatory markers and hemodynamics in ECMO-supported patients and is associated with a favorable prognosis.

Keywords

Acute gastrointestinal injury; Extracorporeal membrane oxygenation support; Inflammation; Mortality

1. Introduction

Extracorporeal membrane oxygenation (ECMO) is a critical life support technique commonly used to treat acute respiratory distress syndrome (ARDS) and cardiogenic shock [1, 2]. The mechanism of ECMO involves an external mechanical device that extracts blood from the patient, facilitates oxygenation and CO_2 removal, and then returns the oxygenated blood to the body [3]. Due to the severity of underlying conditions and the invasiveness of the procedure, ECMO patients are at increased risk for complications, with infections being the most common [4]. The gastrointestinal tract plays a vital role in nutrient and water absorption while also serving as a critical barrier against harmful substances and pathogens. The intestinal mucosal protective barrier functions through a synergistic combination of mechanical, chemical, immune and biological components [5, 6].

Acute gastrointestinal injury (AGI) refers to the rapid damage to the gastrointestinal tract, leading to impaired function. This condition may manifest as inflammation, hemorrhage, necrosis or perforation, and is often associated with a systemic inflammatory response that can significantly affect the patient's overall physiological status. AGI typically develops acutely, with symptoms appearing within hours to a day. It can result from multiple factors, including ischemia, infection, trauma, or the use of certain medications, such as nonsteroidal anti-inflammatory drugs and antibiotics. Clinically, AGI is characterized by symptoms such as abdominal pain, nausea, vomiting, diarrhea, bleeding and abdominal distension [7, 8].

AGI is a common complication in critically ill patients, with approximately 60% of such patients experiencing gastrointestinal dysfunction. ECMO patients are particularly vulnerable to

early gastrointestinal injury [9, 10]. Among ECMO-supported patients, the probability of early AGI onset is higher due to the multiple complications often associated with ECMO, which can lead to gastrointestinal dysfunction and, in severe cases, organ failure [11, 12]. Therefore, investigating the impact of AGI in ECMO patients is of significant clinical importance. This study aims to explore the incidence of AGI in ECMO-supported patients and its effect on patient prognosis. We hypothesize that the presence and severity of AGI are closely associated with treatment outcomes, recovery speed, and survival rates in ECMO patients. By comparing the disease prognosis of patients with different AGI grades, high-risk groups can be identified, providing a basis for clinicians to formulate personalized treatment plans and evaluate AGI as a prognostic indicator. Additionally, this study will offer practical guidance to clinicians, aiding them in making more informed decisions when managing acute gastrointestinal injuries of varying severity, ultimately optimizing clinical pathways and improving patient survival rates.

Interleukin-1β (IL-1β) is a key pro-inflammatory cytokine whose elevation in AGI patients often correlates with the severity of the inflammatory response, indicating a higher risk of complications and poor prognosis. Similarly, IL-6, another important pro-inflammatory cytokine, is associated with the onset of systemic inflammatory response syndrome (SIRS) and with worsening conditions, prolonged hospital stays and increased mortality [13, 14]. IL-10, an important anti-inflammatory cytokine, reflects the body's ability to regulate immune responses. Elevated IL-10 levels in AGI patients are often linked to a better prognosis [15]. Monitoring the levels of these inflammatory markers is essential for assessing the patient's condition, adjusting treatment plans and predicting outcomes.

Based on these, clinical research on the early occurrence of AGI in ECMO patients is crucial. This study aims to explore the incidence of early AGI and provide evidence to inform the diagnosis and treatment of AGI in clinical ECMO-supported patients.

2. Materials and methods

2.1 General data and collection

According to the sample size estimation formula for two-sample mean comparison based on group design: $n = 2 \times [(\mu_\alpha + \mu_\beta)^2\sigma^2]/\delta^2$ [16]. The test level $\alpha = 0.05$, the probability of committing a Type II error β is set at 0.1, and the test power $(1 - \beta)$ is 0.9. From tables, $\mu_{0.05} = 1.96$, $\mu_{0.1} = 1.282$ [17]. The estimated value of the mean difference δ based on preliminary clinical observations (12 cases) is 5, and the estimated sample standard deviation σ is also based on preliminary clinical observations (12 cases) as 7. Substituting into the formula, the calculated sample size is n1 = n2 = 41.2 cases. After considering a 20% loss to follow-up rate, and matching the treatment and control groups at a 1:1.1 ratio, Group B requires 50 patients, and Group A requires 55 patients. Finally, the study included 55 patients in Group A and 50 patients in Group B, who were assigned to their respective study groups according to the recorded treatment methods.

From existing case records, clinical data were retrospectively collected from 105 patients who received ECMO support at our hospital between 01 January 2023 and 01 October 2024. Patients were divided into two groups on the first day of ECMO support (T1) according to recorded AGI grades. Group A included 55 patients with mild injury (Grade I + II), while Group B included 50 patients with moderate to severe injury (Grade III + IV). The study was approved by the hospital's ethics committee (Approval no. KYSB20240181).

Inclusion Criteria: Patients were included in the study if they met the diagnostic criteria for ECMO [18]. The indications for ECMO included acute respiratory failure, defined by severe hypoxemia (arterial oxygen saturation (SaO_2) $<$80% or partial pressure of arterial oxygen (PaO_2) $<$60 mmHg despite adequate oxygen therapy) or respiratory (Partial Pressure of Carbon Dioxide in Arterial Blood ($PaCO_2$) $>$50 mmHg with pH $<$7.25, uncorrected by conventional treatment). Additionally, patients with acute heart failure were eligible, characterized by cardiogenic shock presenting as persistent hypotension despite fluid resuscitation and drug support or severe ventricular dysfunction with worsening symptoms of heart disease. ECMO was also indicated in cases requiring cardiopulmonary support, such as severe pneumonia, pulmonary embolism, ARDS or post-cardiac surgery. Diagnostic evaluations included arterial blood gas analysis (ABG) to monitor oxygenation and carbon dioxide elimination, imaging studies such as chest X-rays or Computed Tomography (CT) scans to assess lung pathology, and cardiac function assessments using techniques such as transesophageal echocardiography. Comprehensive evaluations comprised assessments of vital signs, hemodynamic indicators (*e.g.*, central venous pressure (CVP) and arterial blood pressure), and the oxygenation index (OI; calculated as OI = (PaO_2/Fraction of Inspired Oxygen (FiO_2)) $\times$ 100) to assess oxygenation capacity. The decision to initiate ECMO requires a comprehensive assessment that typically involves a multidisciplinary approach from critical care, cardiology, and pulmonology teams based on the patient's overall health, prognosis, and the potential benefits of ECMO support. The patient's prognosis and quality of life are carefully analyzed as part of this assessment, and the wishes of the patient and their family were prioritized, with transparent communication to ensure informed decision-making. The patients included in this study were required to meet specific criteria: they had to be at least 18 years old and receive ECMO support within two hours of hospital admission.

Exclusion Criteria: Cases were excluded if they met the following: (1) ECMO support duration $<$72 hours; (2) Contraindications to anticoagulant therapy (patients with contraindications to anticoagulation may face higher bleeding risks and clinical management difficulties during ECMO); (3) Concurrent inflammatory bowel disease, intestinal obstruction, gastrointestinal tumors, chronic gastrointestinal insufficiency (a digestive system disease characterized by long-term insufficient function of the gastrointestinal tract, leading to a decreased ability to digest and absorb food); (4) Pregnant and perinatal women; (5) Recent gastrointestinal surgery (within 3 months).

2.2 Methods

This retrospective study analyzed recorded intervention methods from existing case records. Both patient groups received ECMO support based on guidelines from the Extracorporeal Life Support Organization (ELSO) [19] and a standardized gastrointestinal nutrition support strategy. Other symptomatic treatments were identical across groups.

2.2.1 Diagnostic criteria for AGI

The diagnosis of AGI involves a comprehensive evaluation of the patient's medical history, clinical presentation, laboratory findings and imaging results [20]. Specifically, the diagnostic criteria include:

(1) Clinical presentation

Abdominal pain: Typically persistent or intermittent, which may be localized to a specific area of the abdomen or affect the entire abdomen. Nausea and vomiting: May or may not be associated with food intake. Abdominal bloating: Often caused by the accumulation of intestinal gases or the retention of intestinal contents. Other symptoms: Such as diarrhea, constipation or the presence of black or bright red blood in the stool.

(2) Laboratory tests

Complete blood count: May reveal leukocytosis, indicative of infection or inflammation, and anemia, especially if associated with bleeding. Liver and kidney function tests: Abnormal results may indicate complications such as acute liver injury or renal failure. Electrolyte imbalance: Conditions such as vomiting and diarrhea may lead to imbalances in electrolytes, including sodium, potassium and chloride. Severe imbalances can cause fluctuations in vital signs. Blood gas analysis: If gastrointestinal injury leads to shock or hypoxia, blood gas analysis may show acidosis (*e.g.*, metabolic acidosis, lactic acidosis, *etc.*).

(3) Imaging studies

a. X-ray examination: (1) pneumoperitoneum, characterized by free air beneath the diaphragm, typically observed near the liver margin or in the retroperitoneal space, suggesting a potential intestinal perforation; (2) bowel obstruction, identified by an abnormal distribution of gas in the intestines, bowel dilation and air-fluid levels indicating intestinal contents accumulation.

b. Ultrasound examination: (1) fluid accumulation, where free fluid, such as blood or pus, appears as hypoechoic areas in the abdominal cavity; (2) bowel wall thickening, suggesting intestinal inflammation, bleeding or damage.

c. CT scan: (1) bowel wall thickening, particularly with contrast enhancement, may indicate intestinal inflammation or bleeding; (2) gas or fluid accumulation in the bowel, suggesting perforation or hemorrhage; (3) intestinal perforation, evidenced by contrast leakage into the peritoneal cavity, with the presence of gas and fluid in the bowel.

(4) Endoscopy findings can help identify bleeding sites, ulcers, or other lesions in specific cases.

(5) Other assessment indicators: ① bowel function assessment, evaluating bowel ventilation, gas passage, and the frequency and nature of defecation; ② biomarkers: such as fecal calprotectin, to assess intestinal barrier function in AGI.

2.2.2 AGI grading

AGI grading was based on a comprehensive evaluation of clinical manifestations, laboratory test results and imaging findings: (1) clinical symptoms: including abdominal pain, nausea and vomiting; (2) laboratory assessments: (a) inflammatory markers: levels of C-reactive protein (CRP), white blood cell count (WBC), Tumor Necrosis Factor-alpha (TNF-α), and IL-6 assess inflammation; (b) biochemical markers: indicators of electrolyte imbalances and renal function, such as creatinine and blood urea nitrogen; (3) imaging examinations: abdominal ultrasound or CT scans are used to identify complications, such as intestinal obstruction or perforation; (4) histological results: when available, gastrointestinal biopsies provided additional references for grading.

2.2.3 Reliability of classification

The reliability of the AGI classification was assessed using several verification methods. First, multiple reviewers, including gastroenterologists and intensivists, independently assessed the AGI grade and cross-verified the results. The Kappa coefficient (Kappa = 0.87) was used to evaluate the consistency among reviewers. Retrospective quality control measures were also implemented during case reviews to maintain high standards in AGI grading and ensure adherence to predefined criteria, enhancing the reliability of the process. Additionally, regular checks were conducted to verify the accuracy and integrity of clinical symptoms, laboratory results, imaging findings and histological data entries.

2.2.4 Data analysis

Descriptive statistics were used to summarize the basic characteristics of patients, including age, gender and underlying conditions. For group comparisons, continuous variables were analyzed using *t*-tests or Mann-Whitney U tests, depending on data distribution. Categorical variables, such as complication incidence and mortality rate, were assessed using Chi-square tests.

2.2.5 Grading criteria

The grading criteria for AGI help physicians assess the severity of the injury, typically including the following levels: see Table 1 below.

The establishment of ECMO and detailed nutritional management process can be found in **Supplementary material**.

2.3 Observational indicators

As a retrospective study, the results of the outcome indicators have already been recorded in the existing case records.

(1) Inflammatory factors

Inflammatory factor levels were assessed through blood samples collected at specific time points during ECMO support: the start of treatment (T0), the first day (T1), second day (T2), third day (T3) and fourth day (T4). At noon each day, 5 mL of radial artery blood was drawn for analysis. Serum concentrations of IL-1β, IL-6, IL-10 were measured using the radioimmunoassay method.

(2) Hemodynamic indicators

Hemodynamic parameters, including systolic blood pres-

TABLE 1. Grading criteria for acute gastrointestinal injury.

Specific manifestations	Grade I (Mild)	Grade II (Moderate)	Grade III (Severe)	Grade IV (Critical)
Gastrointestinal tract	Minor injury	Moderate injury	Severe injury	Extremely severe injury
Abdominal pain	Mild abdominal pain or discomfort	Noticeable abdominal pain	Significant abdominal pain	Severe abdominal pain
Nausea and vomiting	Possible mild nausea or vomiting	Possible nausea, vomiting and diarrhea	Possible severe nausea, vomiting and diarrhea	Accompanied by persistent vomiting and severe diarrhea
Bleeding	No bleeding	Possible mild bleeding	Noticeable gastrointestinal bleeding	Possible massive gastrointestinal bleeding
Abdominal examination	No obvious features	No obvious features	Significant tenderness, rebound tenderness, and abdominal rigidity, accompanied by noticeable abdominal distension	May show clear signs of peritoneal irritation, accompanied by acute abdominal pain
Complications	No severe complications	No severe complications	No severe complications	May include multiple organ dysfunction syndrome (MODS)
Health status	Normal	Generally not life-threatening	Possible symptoms of shock (such as hypotension, tachycardia)	Leads to severe shock

sure (SBP), diastolic blood pressure (DBP), MAP and central venous pressure (CVP), were measured daily at T0, T1, T2, T3 and T4. Average daily levels of these parameters were recorded for comparison between groups.

(3) ECMO-related parameter indicators

ECMO-related outcomes included the success rate of ECMO weaning, mortality rate and discharge rate.

(4) Follow-up

Patients who were successfully weaned from ECMO and discharged were followed up for three months to evaluate survival status. Data on mortality rate, complications, readmission and disease recovery were statistically recorded to assess post-ECMO outcomes.

2.4 Statistical methods

Data analysis was performed using SPSS 21.0 statistical software (IBM, Armonk, NY, USA) and GraphPad Prism 8.0.2 (GraphPad Software Inc, San Diego, CA, USA). The Shapiro-Wilk test was employed to assess the normality of the data distribution. This test is commonly used for small sample sizes, with the null hypothesis stating that the data follow a normal distribution. A p-value less than 0.05 indicated rejection of the null hypothesis, suggesting that the data did not follow a normal distribution. For normally distributed continuous data, results were presented as mean $\pm$ standard deviation (SD). Independent samples t-tests were used for comparisons between groups, while paired samples t-tests were used for within-group comparisons. For data with skewed distributions or unequal variances, the Mann-Whitney U test was applied, and the results were expressed as median (M) and interquartile range (P25, P75). Categorical data were summarized as frequencies and percentages. Between-group comparisons were conducted using chi-square tests or Fisher's exact test, as appropriate. A p-value of < 0.05 was considered indicative of statistical significance.

3. Results

3.1 Baseline characteristics

In order to reduce confounding bias and more accurately assess the effect of an intervention or treatment, this study utilized Propensity Score Matching (PSM) to control for covariates that might influence the treatment outcomes of the two patient groups. By calculating the propensity score for each individual, it was possible to match treated individuals with untreated individuals, creating a similar control group after matching.

The general information of the two groups is shown in Table 2, with no significant differences between the groups ($p > 0.05$).

3.2 Inflammatory factors

From time points T0 to T4, inflammatory factors in Group A were consistently lower than those in Group B ($p < 0.05$). Additionally, the variation in inflammatory factors within Group A remained relatively stable over time ($p > 0.05$), whereas Group B exhibited significant increases in these factors ($p < 0.05$, Table 3).

3.3 Hemodynamics indicators

At time points T0 to T4, SBP, DBP and MAP in Group A were consistently higher than those in Group B, while CVP was lower in Group A ($p < 0.05$). Hemodynamic variations in Group A were relatively stable over time ($p > 0.05$), whereas

TABLE 2. Baseline characteristics of patients in the two groups.

Observation indicators	Pre-PSM				Post-PSM			
	Group A (n = 66)	Group B (n = 63)	t	χ^2/p	Group A (n = 55)	Group B (n = 50)	t	χ^2/p
Gender								
Male (n (%))	36 (54.55)	29 (46.03)	0.935	0.334	31 (56.36)	30 (60.00)	0.142	0.706
Female (n (%))	30 (45.45)	34 (53.97)			24 (43.64)	20 (40.00)		
Age (yr) (mean ± SD)	56.57 ± 12.64	54.97 ± 12.01	0.463	0.736	55.20 ± 13.10	54.51 ± 12.86	0.786	0.272
BMI (kg/m^2) (mean ± SD)	21.66 ± 1.75	21.84 ± 2.06	0.594	0.535	22.84 ± 2.76	22.95 ± 2.82	0.854	0.184
ECMO Indications (n (%))								
Cardiogenic	55 (83.33)	50 (79.37)	0.335	0.563	52 (94.55)	47 (94.00)	0.014	0.904
pulmonary	11 (16.67)	13 (20.63)			3 (5.45)	3 (6.00)		
Basic disease (n (%))								
Diabetes mellitus	43 (65.16)	37 (58.73)	0.564	0.453	25 (45.45)	22 (44.00)	0.022	0.881
Hypertension	40 (60.61)	19 (30.16)	12.040	0.001	19 (34.55)	18 (36.00)	0.024	0.876
Angiocardiopathy	43 (65.15)	22 (34.92)	11.784	0.001	28 (50.91)	20 (40.00)	1.256	0.262
Cerebrovascular disease	36 (54.55)	38 (60.32)	0.439	0.508	19 (34.55)	22 (44.00)	0.984	0.321
Renal failure	35 (53.03)	40 (63.49)	1.450	0.229	15 (27.27)	13 (26.00)	0.022	0.883
COPD	25 (37.88)	34 (53.97)	3.362	0.067	20 (36.36)	18 (36.00)	0.001	0.969
Asthma	23 (34.85)	30 (47.62)	2.172	0.141	16 (29.09)	20 (40.00)	1.383	0.240
Cancer	15 (22.73)	16 (25.40)	0.126	0.723	9 (16.36)	12 (24.00)	0.955	0.329

Abbreviations: Pre-PSM: Before Propensity Score Matching; Post-PSM: After Propensity Score Matching; BMI: Body Mass Index; ECMO: Extracorporeal Membrane Oxygenation; COPD: Chronic Obstructive Pulmonary Disease; SD: standard deviation.

TABLE 3. Comparison of inflammatory factors between the two groups ($\bar{x} \pm s$).

Group	n	IL-1β (ng/L)				
		T0	T1	T2	T3	T4
Group A	55	1.23 ± 0.34	1.32 ± 0.44*	1.35 ± 0.45*	1.25 ± 0.57*	1.31 ± 0.39*
Group B	50	1.50 ± 0.54	1.65 ± 0.62	1.82 ± 0.68	1.84 ± 0.75$^{\#}$	1.88 ± 0.87$^{\#}$
t-values	-	2.905	−3.139	4.042	4.518	4.322
p-values	-	0.005	0.002	<0.001	<0.001	<0.001
Group	n	IL-6 (pg/mL)				
		T0	T1	T2	T3	T4
Group A	55	120.25 ± 16.93	118.57 ± 13.89*	119.64 ± 11.68*	121.34 ± 8.50*	120.27 ± 9.60*
Group B	50	125.87 ± 8.09	126.84 ± 8.68	128.13 ± 8.05	131.54 ± 10.85$^{\#}$	133.59 ± 9.83$^{\#}$
t-values	-	2.202	3.620	4.373	5.326	7.019
p-values	-	0.031	<0.001	<0.001	<0.001	<0.001
Group	n	IL-10 (pg/mL)				
		T0	T1	T2	T3	T4
Group A	55	5.37 ± 0.75	5.61 ± 0.62*	5.55 ± 0.74*	5.32 ± 0.78*	5.54 ± 0.60*
Group B	50	5.96 ± 0.86	6.51 ± 1.68	6.35 ± 1.35	6.71 ± 1.53$^{\#}$	6.50 ± 1.19$^{\#}$
t-values	-	3.714	3.625	3.718	5.839	5.151
p-values	-	<0.001	0.001	<0.001	<0.001	<0.001

**Compared to T0, $p > 0.05$; $^{\#}$Compared to T0, $p < 0.05$.*
IL: Interleukin; T0: the start of treatment; T1: the first day; T2: second day; T3: third day; T4: fourth day.

Group B demonstrated significant fluctuations in these indicators ($p < 0.05$, Table 4).

3.4 ECMO-related indicators

The success rate of ECMO weaning and the discharge rate in Group A were significantly higher compared to Group B, while the in-hospital mortality rate in Group A was significantly lower than that in Group B ($p < 0.05$, Table 5).

3.5 Follow-up

During follow-up, patients from Group A were found to have significantly better outcomes compared to those in Group B in terms of post-discharge mortality, complications, readmissions, and disease recovery ($p < 0.05$, Table 6).

3.6 Analysis of the factors affecting the early onset of moderate to severe AGI in ECMO patients

Univariate analysis showed that in Group B (mild AGI group), the systolic blood pressure (SDP), diastolic blood pressure (DBP), mean arterial pressure (MAP), pH at the time of ECMO initiation were higher compared to Group A (moderate to severe AGI group), while Body Mass Index (BMI), Vascular Insulin Sensitivity (VIS), lactic acid level and total bilirubin levels were lower in Group B compared to Group A. These differences were statistically significant ($p < 0.05$, Table 7). Logistic regression analysis was performed with significant variables from the univariate analysis, including SDP, DBP, MAP, BMI, VIS, pH, lactate, and total bilirubin, with the occurrence of AGI as the dependent variable (mild AGI assigned as 0, moderate to severe AGI as 1). The results indicated that MAP and VIS were included in the model as independent variables. The comparison of BMI showed a statistically significant difference ($p < 0.05$, Table 8), indicating that BMI is an independent risk factor for the early onset of moderate to severe AGI in ECMO patients.

4. Discussion

ECMO is an advanced technology that provide respiratory and circulatory support [21]. AGI is a common complication in ECMO patients, with studies showing a significantly higher incidence of AGI in ECMO-treated patients compared to those who do not receive ECMO support. The reported mortality

TABLE 4. Comparison of hemodynamic indicators of the two groups ($\bar{x} \pm s$).

Group	n			SBP (mmHg)		
		T0	T1	T2	T3	T4
Group A	55	120.97 ± 8.19	118.13 ± 7.35*	117.23 ± 8.15*	118.39 ± 8.06*	117.93 ± 7.55*
Group B	50	115.34 ± 10.53	113.34 ± 7.16	112.60 ± 9.08	109.84 ± 9.12$^{\#}$	103.83 ± 8.65$^{\#}$
t-values	-	3.037	3.376	2.754	5.105	8.917
p-values	-	0.003	0.001	0.007	<0.001	<0.001
Group	n			DBP (mmHg)		
		T0	T1	T2	T3	T4
Group A	55	81.37 ± 5.83	79.34 ± 6.20*	80.24 ± 5.34*	78.54 ± 6.02*	79.65 ± 4.84*
Group B	50	77.93 ± 6.72	75.67 ± 6.31	75.36 ± 6.05	74.64 ± 5.63$^{\#}$	69.98 ± 5.35$^{\#}$
t-values	-	2.808	3.006	4.403	3.416	9.725
p-values	-	0.006	0.003	<0.001	0.001	<0.001
Group	n			MAP (mmHg)		
		T0	T1	T2	T3	T4
Group A	55	90.25 ± 9.65	89.91 ± 10.42*	91.53 ± 10.20*	89.10 ± 10.02*	90.15 ± 8.65*
Group B	50	85.25 ± 9.26	84.31 ± 8.65	85.31 ± 7.70	80.95 ± 6.75$^{\#}$	78.68 ± 6.05$^{\#}$
t-values	-	2.696	2.978	3.493	4.926	7.929
p-values	-	0.008	0.004	0.001	<0.001	<0.001
Group	n			CVP (cmH_2O)		
		T0	T1	T2	T3	T4
Group A	55	8.50 ± 1.35	8.23 ± 1.51*	8.65 ± 0.85*	8.30 ± 1.03*	8.27 ± 1.36*
Group B	50	9.27 ± 1.33	9.36 ± 1.53	9.86 ± 1.86	10.13 ± 1.28$^{\#}$	11.07 ± 1.01$^{\#}$
t-values	-	2.950	3.818	4.265	8.077	11.885
p-values	-	0.004	<0.001	<0.001	<0.001	<0.001

**Compared to T0, $p > 0.05$; $^{\#}$Compared to T0, $p < 0.05$.*
SBP: systolic blood pressure; DBP: diastolic blood pressure; MAP: mean arterial pressure; CVP: central venous pressure; T0: the start of treatment; T1: the first day; T2: second day; T3: third day; T4: fourth day.

TABLE 5. Comparison of ECMO-related parameter indicators (n (%)).

Group	n	ECMO successful withdrawal probability	Hospital mortality	Hospital discharge rate
Group A	55	30 (54.55)	23 (41.82)	28 (50.91)
Group B	50	15 (30.00)	35 (70.00)	13 (26.00)
χ^2	-	6.443	8.413	8.413
p-values	-	0.011	0.004	0.004

ECMO: Extracorporeal Membrane Oxygenation.

TABLE 6. Comparison of follow-up outcomes of two groups (n (%)).

Group	n	Mortality after discharge	Complication	Readmission	Disease recovery
Group A	55	8 (14.55)	8 (14.55)	5 (9.09)	27 (49.09)
Group B	50	18 (36.00)	25 (50.00)	20 (40.00)	10 (20.00)
χ^2	-	6.471	15.276	13.793	9.712
p-values	-	0.011	<0.001	<0.001	0.002

TABLE 7. Two groups of related indicators.

Indicators	Group A (n = 55)	Group B (n = 50)	Statistical value	*p*
BMI (kg/m^2) (mean ± SD)	23.68 ± 3.20	21.85 ± 2.96	3.054	0.003
ECMO on machine indicators (mean ± SD)				
ECMO speed (r/min)	3020.80 ± 446.56	3067.50 ± 422.19	0.550	0.584
ECMO flow rate (L/min)	3.19 ± 0.57	3.21 ± 0.69	0.084	0.934
Systolic pressure (mmHg) (mean ± SD)	93.86 ± 34.31	105.60 ± 12.38	2.362	0.021
Diastolic pressure (mmHg) (mean ± SD)	58.48 ± 21.57	68.90 ± 10.37	3.199	0.002
MAP (mmHg) (mean ± SD)	69.64 ± 21.46	79.85 ± 10.66	3.133	0.002
CVP (mmHg)	10 (8.00, 12.25)	10 (8.00, 11.00)	0.640	0.522
VIS (point) (mean ± SD)	87.40 ± 67.88	54.25 ± 44.00	2.995	0.004
pH (mean ± SD)	7.33 ± 0.13	7.40 ± 0.11	2.846	0.005
Lactic acid (mmol/L) (mean ± SD)	7.26 ± 6.11	4.64 ± 3.68	2.691	0.008
PO_2 (mmHg)	146.5 (71.50, 323.75)	126 (92.00, 262.00)	1.923	0.057
PCO_2 (mmHg)	35 (29.00, 43.50)	34 (28.25, 40.40)	1.765	0.081
Oxygenation index (mmHg)	168 (117.75, 375.75)	175 (126.00, 328.00)	0.702	0.484
Glucose (mmol/L) (mean ± SD)	12.56 ± 5.83	10.49 ± 4.83	1.977	0.051
White blood cell (×10^9/L)	13.76 (9.72, 21.13)	14.43 (11.15, 22.79)	1.191	0.236
CRP (mg/L)	75.68 (26.82, 164.30)	69.15 (15.72, 191.69)	0.970	0.334
PCT (ng/mL)	1.76 (0.46, 6.93)	1.25 (0.28, 3.93)	1.963	0.052
Hemoglobin (g/L) (mean ± SD)	91.58 ± 27.10	86.90 ± 24.77	0.923	0.358
Total bilirubin (μmol/L)	16.55 (9.23, 46.93)	25.75 (9.60, 38.08)	7.410	<0.001
Albumin (g/L) (mean ± SD)	27.57 ± 8.67	30.21 ± 6.78	1.726	0.087
Creatinine (μmol/L)	147 (107.75, 273.00)	148 (100.25, 264.25)	0.124	0.902
AST (U/L)	169 (43.00, 417.00)	190 (51.25, 544.50)	0.976	0.332

BMI: Body Mass Index; SD: standard deviation; ECMO: Extracorporeal Membrane Oxygenation; MAP: mean arterial pressure; CVP: central venous pressure; CRP: C-reactive protein; PCT: procalcitonin; AST: Aspartate Aminotransferase; VIS: Vascular Insulin Sensitivity; PO_2: Partial Pressure of Oxygen; PCO_2: Partial Pressure of Carbon Dioxide.

TABLE 8. Logistic regression analysis.

Variable	B	*p*	Exp(B)	95% CI	
				Lower limit	Upper limit
BMI	−2.100	0.028	0.811	0.672	0.978
MAP	0.050	0.051	1.052	1.000	1.106
VIS	−0.010	0.101	0.990	0.977	1.002
Constant	0.739	0.783	2.094	-	-

BMI: Body Mass Index; MAP: mean arterial pressure; CI: confidence interval; VIS: Vascular Insulin Sensitivity; B: Regression Coefficient.

rate for AGI ranges from 20% to 40% [22]. Furthermore, it has been suggested that patients undergoing ECMO who develop early AGI exhibit significantly elevated levels of the pro-inflammatory cytokine IL-6 compared to those without AGI, reflecting notable differences in their cytokine profiles.

ECMO serves as a stable cardiopulmonary support system and acts as a bridge to recovery for patients with acute cardiopulmonary failure [23]. In this study, inflammatory markers in Group A were significantly lower than those in Group B from T0 to T4, with smaller fluctuations in Group A compared to the larger variations observed in Group B. These findings suggest that mild AGI has a relatively minor impact on the inflammatory response in ECMO-support patients. The results indicate that mild AGI may not trigger a strong systemic inflammatory response (SIRS), which helps maintain overall patient stability. Clinical significance: The elevation of inflammatory markers is closely associated with multi-organ dysfunction and mortality. Mild gastrointestinal injury appears to maintain lower levels of inflammation during ECMO treatment, potentially reducing the incidence of complications. This finding offers a new perspective on managing acute gastrointestinal injury in clinical practice, emphasizing the importance of preventing or promptly identifying and addressing gastrointestinal injury in ECMO-supported patients. In the study by Wang J *et al.* [24] 20 patients from each AGI severity level (I, II, III and IV) were randomly selected from the Intensive Care Unit (ICU) of Yantai Yuhuangding Hospital. The study measured serum levels of tumor necrosis factor (TNF)-α, interleukin (IL)-6, procalcitonin (PCT) and C-reactive protein (CRP), followed by statistical analysis. The results indicated that as AGI severity increased, TNF-α and IL-6 secretion significantly increased, and PCT and CRP levels were also notably elevated. Consistent with the findings of this study, these results further confirm the correlation between AGI grading and inflammatory markers in critically ill patients, suggesting that gastrointestinal dysfunction may trigger the systemic inflammatory response syndrome (SIRS) and multiple organ dysfunction syndrome. This provides clinical evidence for evaluating the degree of inflammatory response in critically ill patients based on AGI grading.

From T0 to T4, SBP, DBP and MAP were significantly higher in Group A than in Group B, while CVP was lower in Group A. The smaller hemodynamic fluctuations observed in Group A, compared to Group B, further suggest that mild AGI has a less pronounced effect on the hemodynamics of ECMO-treated patients. Patients with mild AGI exhibit more stable hemodynamics. Clinical significance: Stable hemodynamics are crucial for ECMO-supported patients, as instability in hemodynamics can lead to a higher risk of complications and decreased treatment efficacy. The stability observed in Group A may be attributed to its relatively low inflammatory response, which helps maintain organ perfusion and systemic blood flow stability.

Analysis of the Biological Mechanisms Behind the Inflammatory Response and Hemodynamic Differences: (1) Activation Mechanism of Inflammatory Response. The inflammatory response triggered by AGI is primarily caused by damage to the intestinal barrier and dysbiosis of the gut microbiota. In mild AGI, although the intestinal barrier is somewhat compromised, it still maintains a certain level of integrity, limiting the translocation of bacteria and endotoxins into the bloodstream. This helps reduce the activation of the systemic inflammatory response. In contrast, moderate to severe injury can lead to greater intestinal permeability, allowing bacteria and endotoxins to enter the bloodstream, triggering systemic inflammatory response syndrome (SIRS) and increasing the release of inflammatory mediators such as TNF-α, IL-6 and IL-1β. The increase in these inflammatory factors is closely associated with poor clinical outcomes. (2) Production and Regulation of Cytokines. The regulation of inflammatory factors at different levels of AGI may depend on the host's immune response. In patients with mild injury, the immune system is generally able to control the inflammatory response more effectively, resulting in relatively stable production of inflammatory mediators with minimal fluctuations. In contrast, patients with moderate to severe injury may experience an excessive immune response, leading to upregulation of cytokine production, accompanied by large fluctuations in cytokine levels. This imbalance is often linked to the activation of immune cells such as macrophages and T cells. (3) Neuroendocrine Response. Acute gastrointestinal injury also activates the neuroendocrine system, leading to a stress response. In cases of mild injury, the sympathetic nervous system and adrenal response may be moderate, helping to maintain hemodynamic stability. However, in moderate to severe injuries, activation of the sympathetic nervous system is more pronounced, resulting in an exaggerated endocrine response. This exacerbates the systemic inflammatory response and significantly increases the release of inflammatory mediators. (4) Hemodynamic Impact. Studies have shown that hemodynamic indicators (such as SBP, DBP and MAP) in Group A are significantly higher than those in Group B, while CVP is lower in Group A.

This difference may be related to the release of inflammatory factors and circulating blood volume. Better hemodynamic stability helps maintain organ perfusion and function, reducing tissue hypoxia and cellular damage, thereby decreasing the release of inflammatory mediators. Conversely, patients with moderate to severe injury, due to unstable hemodynamics, may experience organ ischemia and hypoxia, which further exacerbates the inflammatory response.

In summary, the biological mechanisms underlying the differences in inflammatory markers between mild and moderate-to-severe AGI patients are multifactorial. These include intestinal barrier function, immune response regulation, neuroendocrine regulation, hemodynamic status.

CVP is a key parameter for assessing cardiac preload and fluid status. In this study, statistical analysis revealed significant differences in CVP between Groups A and B ($p < 0.001$). The observed difference of approximately 1 cmH_2O, however, may not have substantial clinical implications when considered in isolation. Several factors must be interpreted to understand the significance of this difference. First, the clinical status of the patients is crucial. If CVP values in Group A fall within the normal range, whereas those in Group B exceed it, this may signal the need for closer monitoring and more intensive management for Group B patients. Second, clinical interventions may be influenced by these CVP differences. For instance, an elevated CVP in Group B could necessitate more aggressive fluid management or pharmacological interventions, while patients in Group A may require less intensive adjustments, reflecting their relatively stable condition.

The clinical significance of the CVP differences between the two groups is outlined as follows: (1) Hemodynamic Stability: CVP is an important indicator of right heart function, circulatory blood volume and fluid status. The lower CVP in Group A may suggest a relatively lower circulatory blood volume, but it could also indicate more stable hemodynamic status. Lower CVP is typically associated with better cardiac function, blood flow return, and effective fluid management. This may suggest that patients in Group A experience a lighter circulatory load and reduced cardiac burden under ECMO support, thereby decreasing the risk of heart failure. (2) Impact of Inflammatory Response: Studies indicate that inflammatory marker levels are lower in Group A compared to Group B, which may be linked to the lower CVP observed in Group A. Reduced CVP could be associated with decreased tissue perfusion pressure, potentially leading to a lower inflammatory response. This relationship suggests that patients with mild acute gastrointestinal injury (AGI) may have a milder inflammatory response, which could result in better outcomes. (3) Prognostic Assessment: The differences in CVP may serve as a useful indicator for prognostic evaluation. The lower CVP in Group A, coupled with higher ECMO weaning success rates and lower mortality, suggests that patients with mild AGI have a better prognosis under ECMO support. Clinically, CVP can be a key reference for monitoring and assessing the prognosis of ECMO-supported patients. Mortality rates, complication rates, readmission rates, and recovery outcomes were significantly more favorable in Group A than in Group B, suggesting that mild AGI is associated with a better prognosis in ECMO-supported patients.

The mortality rate, complications, readmission rates and recovery outcomes were significantly more favorable in Group A than in Group B, suggesting that mild AGI is associated with a better prognosis in ECMO-supported patients. Mild AGI usually induces a localized, relatively mild inflammatory response with minimal systemic inflammation [25], allowing patients to maintain normal physiological conditions and experience fewer severe complications. These patients generally exhibit stronger tissue repair capacities and immune function, which enables them to adapt more quickly to the physiological changes induced by ECMO support. As a result, they demonstrate higher rates of successful ECMO weaning, hospital discharge and lower mortality rates. In contrast, moderate to severe AGI triggers a pronounced systemic inflammatory response, significantly increasing the risk of disease recurrence. Patients with severe AGI often exhibit elevated levels of inflammatory markers, which may progress to systemic inflammatory response syndrome (SIRS) or sepsis [26]. Under ECMO support, systemic inflammation in these patients is exacerbated, leading to widespread organ dysfunction and poor clinical outcomes. Severe AGI is frequently associated with complications such as vasodilation, reduced peripheral resistance, and hypotension, which may arise from SIRS. Furthermore, translocation of toxins and bacteria from the intestine into the bloodstream can lead to sepsis, further destabilizing hemodynamics. High-grade AGI often involves extensive tissue necrosis, intestinal perforation, or severe hemorrhage, resulting in multiple organ dysfunction syndrome (MODS) [27], which significantly increases mortality and predisposes patients to complications such as infection and shock. These complications not only worsen the clinical condition but may also reduce the effectiveness of ECMO support [28]. Severe AGI can lead to secondary complications, including peritonitis and sepsis, which amplify patient suffering and hinder recovery. Additionally, patients with severe AGI are more likely to experience recurrent health issues and complications after discharge, leading to higher readmission rates, additional strain on healthcare resources, and substantial psychological and physiological burdens. Recovery in these cases is often prolonged, requiring more intensive and targeted interventions. Severe AGI may also result in long-term challenges, such as malabsorption and intestinal dysfunction, complicating rehabilitation after ECMO withdrawal and potentially affecting the patient's quality of life [29].

Hua Xu collected clinical data from patients with infectious acute respiratory distress syndrome (ARDS) admitted to the intensive care unit (ICU) of Tianjin First Central Hospital between March and October 2023. Based on the AGI classification system, the patients were categorized into AGI grades 0–IV. The clinical features and 28-day clinical outcomes were observed, and both univariate and multivariate logistic regression analyses were performed to identify risk factors associated with the prognosis of ARDS patients with concomitant AGI. The results showed that, as AGI grade increased, the ARDS grade also increased, while white blood cell count, neutrophil count, lymphocyte count, lymphocyte percentage, and 28-day mortality all exhibited significant upward trends. The conclusion of the study indicated that the incidence of AGI in infectious ARDS patients was approximately 90%, and

that higher AGI grades were associated with worse patient prognosis. This finding is consistent with the current study and further confirms that AGI grade is negatively correlated with patient prognosis.

In this study, addressing confounding factors was essential for accurately evaluating the relationship between AGI and patient outcomes. Several potential confounders and their mechanisms of influence were identified. Therapy variability-including differences in treatment protocols or clinical decisions-can lead to inconsistencies in patient management and influence clinical outcomes such as ECMO weaning rates, mortality and discharge success. Variations in ECMO use, anticoagulation strategies, and the intensity of nutritional support can significantly affect these outcomes. Consequently, discrepancies in therapeutic approaches may considerably influence study results. Comorbidities, including diabetes, liver disease and pulmonary disease, may also influence patient outcomes. For instance, diabetes is associated with an increased risk of infection and chronic inflammation, which may hinder recovery and worsen prognosis. Similarly, other comorbid conditions can impact ECMO tolerance and the severity of the inflammatory response, introducing variability in outcomes. Infection incidence is another key factor to consider. ECMO patients are particularly susceptible to infections, including respiratory and bloodstream infections. Variations in the type, timing, and severity of these infections can significantly influence inflammatory marker levels and clinical outcomes, further complicating the study findings.

To mitigate the influence of confounding factors, statistical techniques such as multivariable regression analysis should be applied to account for their effects. Additionally, conducting stratified analyses based on comorbidities and age may provide more detailed insights into how prognostic outcomes differ among subgroups. Prospective study designs could further reduce selection bias and minimize the confounding effects often present in retrospective studies. By carefully identifying and adjusting for these factors, the study can more accurately assess the relationship between AGI, inflammatory markers, and disease outcomes in ECMO patients, thereby enhancing the validity and reliability of its conclusions.

Since this study relied on retrospective analysis, it is inherently prone to biases and data inaccuracies. Prospective studies could help mitigate these biases and improve the reliability of the findings. Several strategies can be employed to enhance the credibility of future research: Clear inclusion and exclusion criteria: In prospective studies, researchers can establish precise criteria for patient selection to ensure consistency and objectivity. Standardized data collection processes: The study can implement standardized procedures for data collection to ensure accuracy, consistency and minimize bias due to inconsistent data recording. Real-time monitoring and follow-up: Prospective studies allow for real-time monitoring of patients' inflammatory marker levels and clinical status during ECMO support. This provides more reliable data and allows for prompt capture of changes in patient status. Random grouping: Where feasible, random grouping could be used to divide patients into different AGI grade groups. Randomization can help minimize the impact of inter-group differences and strengthen causal inferences. Multicenter studies: Conducting prospective studies across multiple hospitals increases sample size, enhances the external validity of the findings, reduces single-center bias, and improves the generalizability of the results. Statistical analysis plans: Detailed statistical analysis plans should be developed before the study begins, including the clear identification of primary and secondary endpoints, as well as pre-determined data analysis methods. This helps reduce selective bias during analysis and ensures reliable results. Long-term follow-up: Observing long-term outcomes of patients post-ECMO support, such as complications and readmission rates, provides a more comprehensive understanding of AGI grading's impact on patient prognosis.

In retrospective studies, missing data or incomplete records are common issues that can affect the reliability and validity of research results. To address potential data gaps, the following strategies can be implemented: (1) Establish detailed data collection standards: Before the study begins, develop clear standards for data collection and documentation to ensure all relevant information is systematically recorded and archived. This should include basic patient information, medical records, treatment processes, inflammatory factor levels, and hemodynamic indicators. (2) Use data verification tools: During data collection, employ standardized forms and databases to ensure data completeness. After data entry, verify and correct any errors or inconsistencies. (3) Review case records multiple times: Arrange for team members to regularly review case records to ensure completeness and accuracy. Cross-checking data from multiple sources helps ensure consistency. (4) Supplementary surveys and interviews: For cases with missing data, supplementary surveys or interviews with medical staff, patients or families can be conducted to fill in gaps. (5) Use electronic health record (EHR) systems: If available, utilize the querying and reporting functions of EHR systems to quickly extract relevant data, reducing manual errors or omissions. (6) Data management plan: Develop a comprehensive plan for data management, covering collection, storage, cleaning and analysis. Ensure that all team members understand the process and standards to minimize data gaps. (7) Document missing data: Clearly document missing data in the research report, including reasons for its absence, the quantity, and the handling methods. This increases transparency and helps readers understand the study's limitations.

Despite these strategies, the study has some limitations, such as the inherent biases of retrospective research, the small sample size, and the restrictions of a single-center setting, which limit the generalizability of the results. Additionally, the lack of long-term follow-up data prevents us from assessing the lasting effects of treatment or potential complications, hindering a comprehensive understanding of AGI's long-term impact on ECMO patients. Future studies should aim to: • Expand the sample size to improve the reliability of the results. • Conduct multicenter collaborative research to enhance sample representativeness and result extrapolation. • Design long-term follow-up studies to evaluate patients' long-term prognosis, including survival rates, quality of life, and complication incidence.

5. Conclusions

Mild AGI has a limited impact on inflammatory factor levels and hemodynamic stability in ECMO-support patients. These findings suggest that recovery prospects for patients with mild AGI are more favorable than for those with moderate to severe AGI.

AVAILABILITY OF DATA AND MATERIALS

The authors declare that all data supporting the findings of this study are available within the paper and any raw data can be obtained from the corresponding author upon request.

AUTHOR CONTRIBUTIONS

XL, XCJ—designed the study and carried them out; XL, XCJ—prepared the manuscript for publication and reviewed the draft of the manuscript. XL, XCJ, TTF, LLD, YZ, HJS—supervised the data collection, XL, XCJ, TTF, LLD, YZ—analyzed the data, XL, XCJ, TTF, LLD—interpreted the data. All authors have read and approved the manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Ethical approval was obtained from the Ethics Committee of The Affiliated Suqian First People's Hospital of Nanjing Medical University (Approval no. KYSB20240181). Written informed consent was obtained from a legally authorized representative for anonymized patients' information to be published in this article.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

SUPPLEMENTARY MATERIAL

Supplementary material associated with this article can be found, in the online version, at https://oss.signavitae.com/mre-signavitae/article/1909484901471535104/attachment/Supplementary%20material.docx.

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