Critical Rounds Newsletter
VOL 6 | Issue 51 | 2025

Dear Doctor,

Greetings from Micro Labs Limited. We are happy to bring you **"Critical Rounds Newsletter"** which contain latest and interesting news in the field of Critical Care.

This issue contains:

1. Predictive value of erythrocyte distribution width-to-platelet ratio combined with procalcitonin on 28-day mortality in sepsis patients

2. Components and elements of glucose management in critically ill adult patients

3. Epithelial alterations in the distal airways of patients with acute respiratory distress syndrome

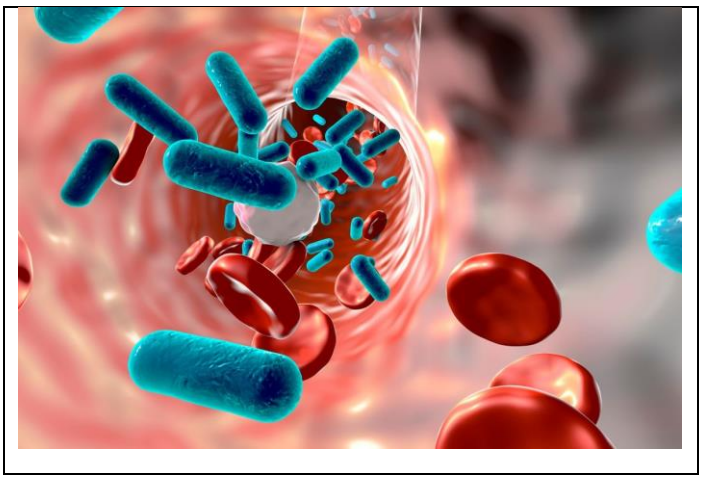
4. Case report on using thrombolysis and extracorporeal cardiopulmonary resuscitation to treat cardiac arrest caused by pulmonary embolism

Should you require any article or, medical information, please feel free to contact us medicalservices@microlabs.in
free to contact us at medicalservices@microlabs.in
Looking forward to your valuable feedback

Best Regards,
Medical Team, Micro Labs Limited

1. Predictive value of erythrocyte distribution width-to-platelet ratio combined with procalcitonin on 28-day mortality in sepsis patients

Introduction



Sepsis is a life-threatening organ malfunction induced by an abnormal host response to infection. Although the exact prevalence of sepsis is unclear, estimations show that it is a leading cause of mortality and serious illness globally. It was anticipated that sepsis would become more common, with increased public health and cost implications.[1] Sepsis causes organ dysfunction by disrupting the body's reaction to infection. So, screening for risk factors associated with sepsis prognosis is crucial for clinical diagnosis, treatment, and prognostic improvement. The ratio of red blood cell distribution width to platelet ratio (RPR) is a reliable indicator of systemic inflammation status. Procalcitonin (PCT) is a key clinical biomarker for evaluating sepsis patients and guiding the use of medications. Research has indicated a strong correlation between PCT and 28-day mortality in sepsis patients. The prognosis of sepsis patients was correlated with both RPR and PCT. However, no studies have shown that combining RPR with PCT can predict 28-day mortality in sepsis patients.[2] So, this study investigated the predictive value of PCT and RPR in sepsis patients and to understand more about the predictive impact of PCT and RPR together in sepsis patients.

This study showed that PCT and RPR as independent risk variables for the 28-day prognosis of sepsis patients.

Methods:

This retrospective cohort study included adult patients who met the diagnostic criteria for sepsis at admission and complete clinical data. Serum PCT level, red blood cell distribution width, and serum platelet level were measured in peripheral blood samples taken from individuals with a sepsis diagnosis. Patients were classified as survivors or non-survivors based on their death within 28 days of sepsis diagnosis. To understand the signs associated with the patients' 28-day prognosis, univariate and multivariate analyses were employed, and the ROC curve was further constructed. The patients' prognosis was assessed using the Kaplan-Meier curve.

Results:

This study comprised 193 patients in total. Patients with sepsis were classified into two groups: survival (n = 156) and non-survival (n = 37), depending on their survival within 28 days of diagnosis. The median age was 62.5 years, and 64.7% were men. PCT and RPR were independent risk variables for the 28-day prognosis in sepsis patients, according to multivariate analysis. PCT and RPR had respective cutoff values of 27.04 and 0.12, and their respective area under the ROC curves was 0.894 and 0.861. According to survival curve analysis, PCT and RPR were linked to patients' 28-day prognosis, and their combination had a stronger prognostic impact (Figure 1).

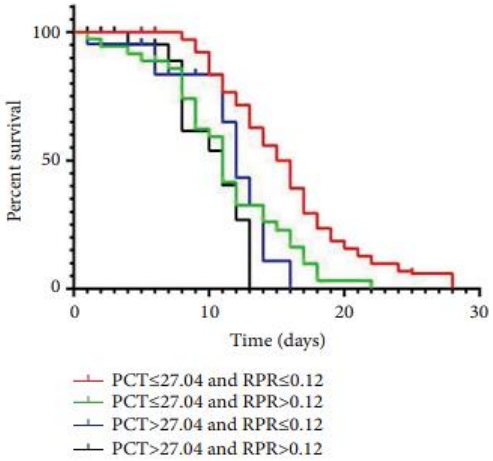


Figure 1: Improves prognostic accuracy for sepsis patients with the combination of PCT and RPR

Discussion:

This study showed that PCT and RPR as independent risk variables for the 28-day prognosis of sepsis patients. In this study, RPR was found to be an independent prognostic factor for sepsis patients. Patients with low RPR had a poorer prognosis, according to survival analysis. This study found that the ideal critical value of RPR is 0.12.[2] This was in consistent with previous critical RPR values of 0.06 to 0.14.[3,4]

Conclusion:

The prognosis for sepsis can be independently predicted by PCT and RPR. The combined use of PCT and RPR (PCT-RPR) can enhance predictive performance even further and serve as a guide for clinical diagnosis, treatment, and prognosis assessment of patients with sepsis.

References

1. Torres LK, Pickkers P, van der Poll T. Sepsis-Induced Immunosuppression. Annu Rev Physiol. 2022 Feb 10;84:157-181.
2. Si Y, Sun B, Huang Y, Xiao K. Predictive Value of Red Cell Distribution Width-to-Platelet Ratio Combined with Procalcitonin in 28-day Mortality for Patients with Sepsis. Crit Care Res Pract. 2024 Aug 12;2024:9964992.
3. Yuyun D et al. Predictive value of the red blood cell distribution width-to-platelet ratio for hepatic fibrosis. Scand J Gastroenterol. 2019 Jan;54(1):81-86.
4. Zhou WJ et al. Association between red cell distribution width-to-platelet ratio and hepatic fibrosis in nonalcoholic fatty liver disease: A cross-sectional study. Medicine (Baltimore). 2019 Jul; 98(30): e16565.

2. Components and elements of glucose management in critically ill adult patients

Introduction

Stress hyperglycemia (SHG) is quite common among critically ill patients. Several studies have shown that uncontrolled blood glucose levels might adversely impact ICU patients with various conditions.[1] SHG is a metabolic reaction to physiological stress that worsens stroke complications and predicts poor outcomes. SHG causes severe metabolic reactions, vascular endothelial dysfunction, and immune-inflammatory responses that impair outcomes in patients with traumatic brain injuries.[2] SHG in acute stroke patients was linked to mortality within 30 days, mechanical ventilation, vasopressor use, and hemorrhagic transformation. It is also a significant predictor of poor prognosis. The current treatment for glucose management in critically ill patients is continuous insulin administration using the insulin infusion protocol (IIP).[1] Assessing SHG has proven hard due to varying baseline glucose levels, which can lead to inaccurate stress measurements.[2] This study aimed to explain glucose control in critically ill adults based on healthcare providers' experiences.

The glucose management practice model's elements and components could be used as a reference for physicians and nurses when managing glucose levels in critically ill patients.

Methods

This was a qualitative investigation that adhered to the Consolidated Criteria for Reporting Qualitative Research (COREQ) standards. Researchers developed a semi-structured interview guide with a total of ten questions. The first author conducted formal, in-depth interviews from January to April 2022. Fifteen doctors and nurses were chosen from ten hospitals. After obtaining consent, both demographic and professional data about the physicians and nurses were collected prior to the interview, including their age, gender, education, ICU type, current role in the ICU, and ICU experience. Interviews lasted 30-60 minutes and were audio-recorded, with field notes taken. Data were analyzed by inductive thematic analysis.

Results

A total of 15 interviews with ICU nurses (N = 10) and physicians (N = 5) were done at 10 tertiary hospitals, either in person or via video conference. Participants had an average age of 35.37 ± 5.14 years, a work experience of 10.87 ± 6.13 years, and 9.33 ± 4.76 years of ICU work. Glucose management in critically ill adult patients has two parts: the inner ring (practice behaviors) and the external space (methods and drivers). The inner ring focuses on practice behaviors, while the outside region concentrates on glucose control approaches and process drivers (Figure 1). Glucose management practices consist of five parts, whereas methods and drives focus on three.

Figure 1: A model for glycemic management in critically ill adult patients.

Discussion

This study gives clear and significant evidence for managing hyperglycemia in critically ill adult patients. The study's glycemic evaluation part revealed that ICU healthcare providers are more sensitive to patients with diabetes.[1] One of the studies found that managing glycemic levels in critically ill individuals is a difficult procedure with various factors, including a history of diabetes.[3] Patients with a history of diabetes are more likely to experience glycemic-related problems in the ICU, regardless of their glycemic control techniques.[4]

Conclusion

This study designed a glycemic management model for critically ill adult patients, clarified its elements based on healthcare providers' perceptions, and elaborated on the methods and drivers covered under each element. This will serve as a reference for physicians and nurses to create a comprehensive glycemic management guideline for critically ill adults.

References

1. Huang M et al. Glucose management in critically ill adults: A qualitative study from the experiences of health care providers. Heliyon. 2024 Jan 23;10(3):e24545.
2. Zhang Y et al. Association between the stress hyperglycemia ratio and all-cause mortality in critically ill patients with Acute Ischemic Stroke: a retrospective cohort study.2024 Sep;14: 20962
3. Munan M et al. Acute and Chronic Effects of Exercise on Continuous Glucose Monitoring Outcomes in Type 2 Diabetes: A Meta-Analysis. Front Endocrinol (Lausanne). 2020 Aug 4;11:495.
4. Long B et al. Evaluation and Management of the Critically Ill Adult With Diabetic Ketoacidosis. J Emerg Med. 2020 Sep;59(3):371-383.

3. Epithelial alterations in the distal airways of patients with acute respiratory distress syndrome

Introduction

Patients with ARDS have complex changes in their airway epithelium, which can result in increased permeability with pneumoproteinemia, altered mucociliary differentiation, and reduced defensive activities.

Acute respiratory distress syndrome (ARDS) is characterized by general lung inflammation and edema, leading to acute respiratory failure. In 2016, ARDS was diagnosed in 10.4% of intensive care unit admissions. ARDS pathogenesis involves several overlapping and interacting mechanisms of damage, inflammation, and coagulation, both in the lung and systemically.[1] The airway epithelium (AE) plays multiple roles in maintaining pulmonary homeostasis, including barrier function, cell differentiation and polarization, and transporting IgA, the primary mucosal immunoglobulin in the airway lumen, through the polymeric immunoglobulin receptor (pIgR). ARDS causes morphological alterations in the airways, although their impact on mucosal immunity and causal processes is unclear.[2] The purpose of this study was to evaluate epithelial changes in ARDS patients' distal airways.

Methods

This retrospective study investigated lung tissue samples from ARDS patients and controls to quantify structural and functional alterations in the small airways. The study used multiplex fluorescence immunostaining and computer-assisted quantification on entire tissue sections. Additionally, this study analyzed mucosal immune markers (IgA and pIgR), as well as epithelial markers, in serum and broncho-alveolar lavage fluid (BALF) samples from ARDS patients and controls.

Results

A total of 25 patients with ARDS were included in this study. ARDS patients had more epithelial denudation ($p=0.0003$) and diffuse neutrophil infiltration ($p=0.0005$) in their airways compared to controls (Figure 1). Multiplex fluorescence immunostaining showed a loss of ciliated cells ($p=0.0317$), a decrease in goblet cells ($p=0.056$), and no change in cell progenitors (basal and club cells), indicating altered mucociliary differentiation. ARDS was associated with increased epithelial permeability and a significant decrease in tight ($p<0.0001$) and adherens junctional proteins ($p=0.025$). Additionally, this study found a significant decrease in pIgR expression ($p<0.0001$), indicating decreased mucosal IgA immunity. ARDS was associated with elevated serum concentrations of secretory component (SC) and S-IgA, as well as other lung-derived proteins (CC16, SP-D, sRAGE) ($p<0.0001$). Their BALF contents, however, did not decrease, indicating that a broken AE caused the proteins in the airways and alveoli to flow over.

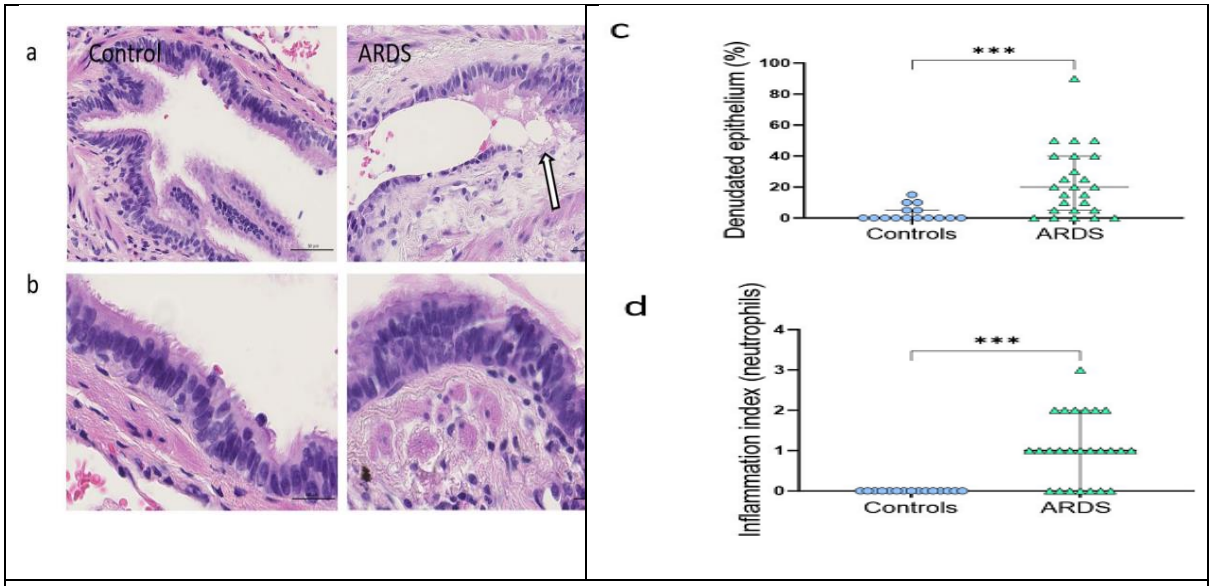


Figure 1: Airway structural alterations in patients with ARDS. **A, B-** HE staining of samples from a control (left) and an ARDS patient (right) reveal epithelial denudation (a, white arrow) and neutrophilic infiltration (b). **C, D-** Quantitative analysis compares the degree of epithelial denudation (c) and the presence of inflammatory cells infiltrating airway epithelia using a four-grade scale (d), in controls (n = 15) and ARDS (n = 25). The nonparametric Mann-Whitney U test was used to assess between-group differences. ***denotes p< 0.0005.

Discussion

This study found significant epithelial alterations in the airways of patients with ARDS and the loss of tight and adherens junctions during ARDS impairs the barrier function of the airway epithelium, potentially affecting paracellular permeability and exposing subepithelial tissues to luminal pathogens.[2] Previous studies showed the dissociation of intercellular connections between epithelial alveolar and endothelial cells during acute lung damage and in culture models of viral/bacterial infection of human airway epithelial cells.[3,4]

Conclusion

Patients with ARDS have altered airway epithelium, resulting in mucociliary differentiation, reduced defensive mechanisms, and increased permeability with pneumoproteinemia.

References

1. Bos LDJ, Ware LB. Acute respiratory distress syndrome: causes, pathophysiology, and phenotypes. Lancet. 2022 Oct 1;400(10358):1145-1156.
2. Gerard L et al. Airway epithelium damage in acute respiratory distress syndrome. Crit Care. 2024 Oct 30;28(1):350.
3. Matthay MA et al. acute respiratory distress syndrome. Nat Rev Dis Prim. 2019;5(1):18.
4. Gao N, Rezaee F. Airway epithelial cell junctions as targets for pathogens and antimicrobial therapy. Pharmaceutics. 2022;14(12):2619.

4. Case report on using thrombolysis and extracorporeal cardiopulmonary resuscitation to treat cardiac arrest caused by pulmonary embolism

Introduction

The use of Extracorporeal cardiopulmonary resuscitation (ECPR) in conjunction with systemic thrombolysis to treat cardiac arrest caused by acute pulmonary embolism (PE) has complications like fatal intracranial hemorrhage.

Acute pulmonary embolism (PE) is a life-threatening condition that can lead to cardiac arrest or circulatory collapse, requiring clinicians to make quick decisions in an emergency.[1] PE causes significant morbidity and mortality. Early and precise diagnosis allows for faster therapy. This is crucial as it may significantly reduce mortality and improve outcomes. For the diagnosis of PE, computed tomographic pulmonary angiography (CTPA) has emerged as the gold standard.[2] Clinical practice guidelines suggest systemic thrombolysis with anticoagulation for patients with high-risk or large PE and hemodynamic impairment. Extracorporeal cardiopulmonary resuscitation (ECPR) with veno-arterial extracorporeal membrane oxygenation (ECMO) is used as a rescue therapy for refractory cardiac arrest caused by PE. Conventional CPR measures are often insufficient to restore spontaneous circulation. Using systemic thrombolysis during CPR, particularly ECPR can raise the risk of cerebral hemorrhage and other complications.[1] In this case report, a patient with acute PE and cardiac arrest experienced intracerebral hemorrhage following tPA administration during mechanical chest compression and ECPR.

Case Presentation:

A 69-year-old male had a prolonged cardiac arrest in an ambulance with pulseless electrical activity. The emergency medical team administered continuous manual CPR. Five minutes after arriving at the emergency department, the patient underwent emergency tracheal intubation, epinephrine injections, and mechanical chest compressions with the LUCAS 2 device. A bedside point-of-care ultrasonography (POCUS) showed an enlarged right ventricle without contractility. Cardiac arrest was considered to be caused by acute PE due to significant RV dilatation. During mechanical chest compressions, 50 mg of tissue plasminogen activator (tPA) was provided as rescue therapy. The dose was divided into a 10 mg bolus and a 40 mg infusion over 2 hours. The extracorporeal membrane staff was asked to start ECPR. Manual or mechanical chest compressions had been occurring for approximately 31 minutes before the ECMO team established cannulation. However, sustained return of spontaneous circulation (ROSC) did not occur until 8 minutes after the commencement of Veno-arterial ECMO. After the first collapse, it took around 39 minutes to achieve ROSC. The patient required ECMO support and vasoactive medication infusions (epinephrine at 0.2 µg/kg/min, norepinephrine at 0.5 µg/kg/min, and dobutamine at 5 µg/kg/min) to stabilize after experiencing sustained ROSC. An urgent computed tomography (CT) with contrast medium revealed a thrombus in both pulmonary arteries, including the distal right interlobar artery and the left interlobar pulmonary artery (Figure 1A–C). CT angiography demonstrated RV dilation with a diameter of 46.6 mm (Figure 1D). The patient had a medical history of Hypertension. On admission, the patient was in a coma with normal pupil diameters on both sides and poor light responses. He was transported to the intensive care unit and cooled to a target temperature of 34 °C with a surface cooling pad for brain protection. POCUS in the emergency department indicated an enlarged RV with no contractility. The CT scan revealed thrombus in both pulmonary arteries. After admission, the patient's hemodynamic condition improved dramatically, allowing for the withdrawal of vasoactive medications like epinephrine and norepinephrine.

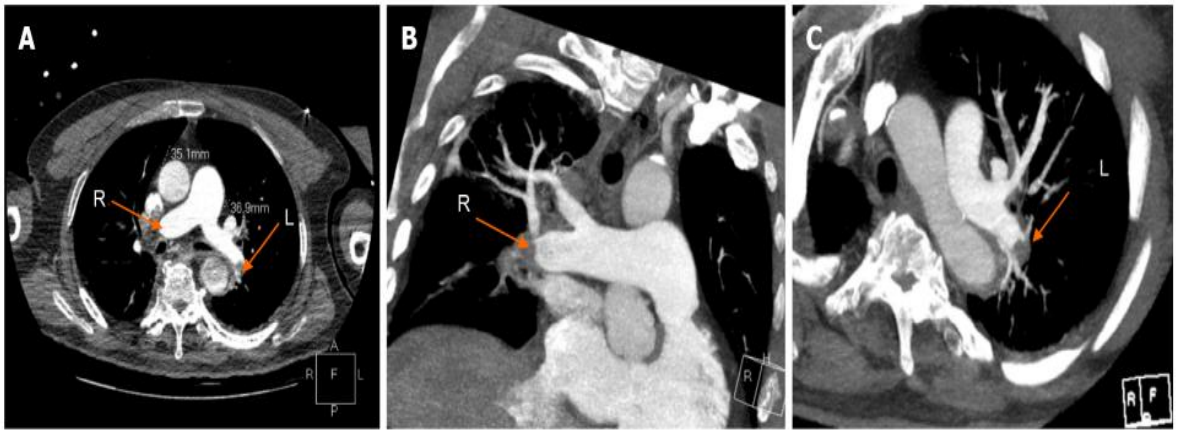


Figure 1: Contrast-enhanced computed CT findings. Orange arrows indicate thrombosis in both pulmonary arteries (A), extension to the distal right interlobar artery (B), and extension to the left interlobar pulmonary artery (C).

Bedside POCUS showed a considerable improvement in the dilated RV and myocardial contractility. The ECMO flow was lowered from 3.5 L/min to 2 L/min then removed on the third day of admission. Despite effective decannulation, the patient remained in a deep coma, with uneven pupil diameters and no light sensitivity. A CT scan indicated left frontal lobe, parietal lobe, and subarachnoid hemorrhage (Figure 1E). A diagnosis of Acute PE was made. To treat suspected PE, tPA was used as a rescue therapy during mechanical chest compressions. ECPR with venoarterial ECMO was started. The patient did not survive. The patient died in the hospital after suffering from intracerebral hemorrhage and severe hypoxic brain injury.

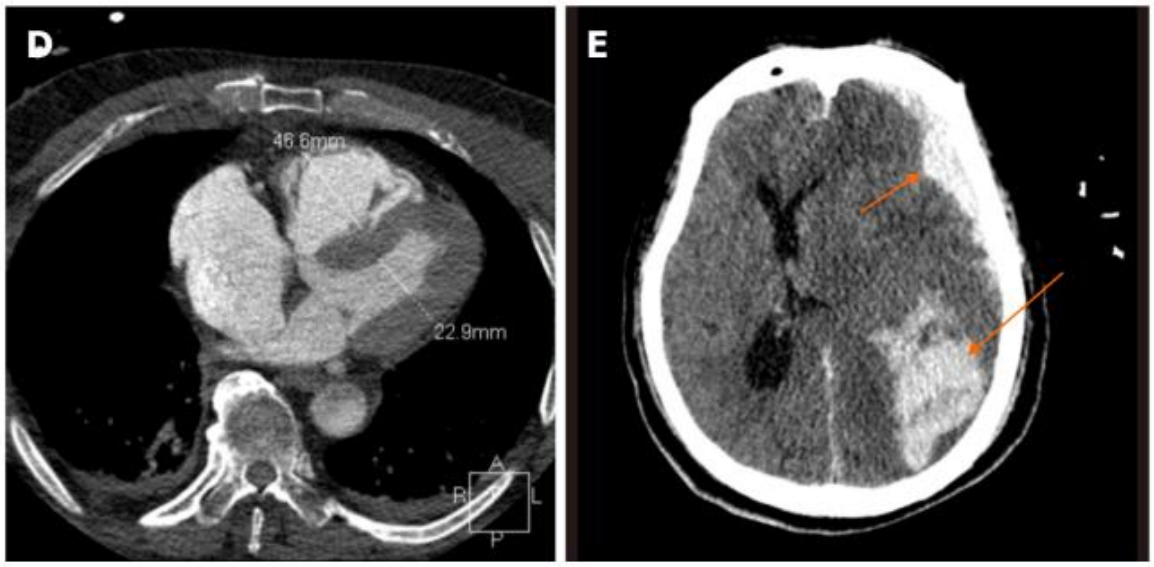


Figure 1: D) 46.6 mm right ventricle dilation as shown in CECT; E) CT scan reveals left frontal, parietal, and subarachnoid hemorrhage (orange arrows).

Discussion

This case report highlights the importance of thrombolytic treatment, mechanical chest compression, and ECPR in treating acute PE that causes refractory cardiac arrest. Veno-arterial ECMO offers cardiopulmonary support, relieving right ventricular strain and acting as a salvage therapy for PE. It can resolve clots with anticoagulation alone or as a bridge to surgical or catheter-directed therapy. Early and aggressive use of veno-arterial ECMO can effectively manage large PE, with positive outcomes.[1] ECMO is an essential life-support strategy; yet it fails to remove the outflow restriction produced by embolized thrombi.[3] Further treatment, either pharmacological or invasive, is required for relieving the thromboembolic burden.[4]

Conclusion

Combining systemic thrombolysis and ECPR to treat cardiac arrest caused by acute PE has possible disadvantages. Although it may enhance survival rates, it also raises the risk of serious bleeding problems, such as cerebral hemorrhage. Making the optimal treatment selection requires a thorough assessment of the patient's condition, prospective benefits and hazards, and available resources.

References

1. Yuan GX, Zhang ZP, Zhou J. Thrombolysis and extracorporeal cardiopulmonary resuscitation for cardiac arrest due to pulmonary embolism: A case report. World Journal of Critical Care Medicine. 2025 Mar 9;14(1):1-7
2. Soffer S et al. Deep learning for pulmonary embolism detection on computed tomography pulmonary angiogram: a systematic review and meta-analysis. Scientific reports. 2021 Aug 4;11(1):15814.
3. Frenk NE et al. Large-bore Suction Thrombectomy Therapy for Massive Pulmonary Embolism on Extracorporeal Membrane Oxygenation. J Vasc Interv Radiol. 2020 Nov;31(11):1932-1934.
4. Elbadawi A et al. National trends and outcomes for extra-corporeal membrane oxygenation use in high-risk pulmonary embolism. Vasc Med. 2019 Jun;24(3):230-233.