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Application of Veno-Arterial ECMO Combined With Hemoperfusion in the Treatment of a Patient With Yunaconitine Poisoning: A Case Report

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ABSTRACT

A 40-year-old man, after accidentally ingesting aconitine in a herbal remedy, suffered severe poisoning symptoms and was diagnosed with multiple arrhythmias. He quickly received veno-arterial extracorporeal membrane oxygenation (VA-ECMO) and hemoperfusion in the ICU, along with continuous renal replacement therapy (CRRT) to stabilize his internal environment. The treatment controlled the arrhythmias, restored heart function, and alleviated acidosis. The patient was discharged feeling well after 9 days. This combined therapy is valuable for severe aconitine poisoning, especially when specific antidotes are lacking, as it provides critical life support and effectively removes toxins.

1 | Introduction

Yunaconitine can induce poisoning symptoms such as nausea, vomiting, limb numbness, dyspnea, and arrhythmias. Its toxic mechanism primarily affects voltage-dependent sodium channels, causing abnormal potassium and calcium ion influx. Studies show that Yunaconitine specifically binds to and activates these channels, leading to abnormal depolarization of cardiomyocytes and neurons, triggering malignant arrhythmias, respiratory suppression, and other severe reactions (Li et al. 2023). Aconite alkaloid poisoning has a short incubation period, acute onset, and rapid disease progression, so early intervention is necessary once diagnosed. Currently, there is no specific antidote for Yunaconitine poisoning. In the absence of a specific antidote, supportive care is provided, including cardiac monitoring, correction of electrolyte imbalances, and symptomatic management (Liu, Zhao, and Fang 2023). Common treatment methods include gastric lavage, emesis, catharsis, hemoperfusion, administration of antiarrhythmic drugs (atropine, lidocaine, amiodarone, etc.), magnesium ions, fat emulsions, cardiopulmonary resuscitation, defibrillation, renal replacement therapy, ECMO, and treatment with traditional Chinese medicine (Emergency Physician Branch, and of Chinese Medical Doctor Association, Professional Committee of Emergency Resuscitation and Disaster Medicine of Chinese Medical Doctor Association, Chinese Emergency Specialist Medical Association 2022).

Veno-arterial extracorporeal membrane oxygenation (VA-ECMO) is primarily used for treating refractory cardiac arrest. It works by extracting blood from the body, oxygenating it through an external oxygenator, and returning it to the body, thereby replacing or supporting heart and lung function. VA-ECMO provides temporary circulatory and oxygenation support for cardiac arrest patients, reducing ischemia–reperfusion injury. In particular, it allows for rapid implementation of targeted temperature management after cardiac arrest, helping to protect brain function (Bougouin et al. 2020). Hemoperfusion uses adsorbents to

remove toxins and waste from blood and is used to treat poison intoxications, renal failure, and hepatic encephalopathy (Ricci et al. 2022; Wu et al. 2023).

This report describes a severe case of Yunaconitine poisoning successfully treated with VA-ECMO combined with hemoperfusion.

1.1 | Case Presentation

1.1.1 | Chief Complaint

On September 22, 2022, a 40-year-old male was brought to our hospital by emergency services (120) after experiencing "limb numbness, vomiting, and palpitations 5 hours following alcohol consumption."

1.1.2 | History of Present Illness

The patient had attended a gathering with colleagues and consumed approximately 250 mL of liquor, 600 mL of beer, and 50 mL of homemade herbal liquor. Following this, he developed headache and dizziness, which were accompanied by numbness in the limbs and around the mouth, chest tightness, and shortness of breath. He also experienced nausea and vomiting, with symptoms progressively worsening to palpitations and difficulty breathing. The patient arrived at the hospital at 18:27 on September 22, 2022. Electrocardiography (ECG) indicated polymorphic ventricular tachycardia with conduction block, supraventricular tachycardia, and ventricular premature beats.

1.1.3 | Personal and Family History

The patient had no previous medical history or relevant family history.

1.1.4 | Physical Examination

Upon admission, the patient had a body temperature of 36°C, a respiratory rate of 28 breaths/min, and a blood pressure of 102/94 mmHg. Cardiac monitoring revealed chaotic arrhythmia. The patient was drowsy (E2V4M5), with no cyanosis of the lips or nail beds, and a noticeable smell of alcohol on his breath.

1.1.5 | Auxiliary Examinations

Laboratory analysis indicated lactic acidosis: pH 7.220, PaO2 552.5 mmHg↑, PaCO2 38.6 mmHg, K+ 3.79 mmol/L, Ca2+ 1.04 mmol/L↓, Lactate 7.9 mmol/L↑, HCO3–15.5 mmol/L, and BE −11.51 mmol/L.

1.1.6 | ECG Findings Suggested

Polymorphic ventricular tachycardia with conduction block, supraventricular tachycardia, and ventricular premature beats (Figure 1A).

1.1.7 | Imaging

Chest and abdominal X-rays showed no abnormalities.

1.1.8 | Echocardiography

Echocardiography revealed decreased systolic function of the left and right ventricles, left ventricular wall thickening, mild mitral regurgitation, mild tricuspid regurgitation, and an ejection fraction (EF) of 0.21.

1.1.9 | Toxicology Testing

Given the patient's symptoms and history of drinking homemade herbal liquor, toxicology testing was performed as part of the differential diagnosis.

1.2 | Final Diagnosis

1. Acute Yunaconitine poisoning and alcohol intoxication.
2. Cardiac and respiratory arrest, chaotic arrhythmia, ventricular fibrillation, ventricular tachycardia, ventricular premature beats, supraventricular tachycardia, and cardiogenic shock.
3. Metabolic acidosis and lactic acidosis.

1.3 | Treatment

The patient was admitted at 18:27 and immediately placed under cardiac monitoring and arterial blood pressure monitoring in the emergency department. Intravenous access was established, and amiodarone infusion (1 mg/min) was initiated. The electrocardiogram showed chaotic arrhythmias, including ventricular tachycardia, premature ventricular contractions, and supraventricular tachycardia. At 20:20, the patient lost consciousness, with no carotid pulse and a blood pressure of 34/17 mmHg. Ventricular fibrillation was detected (Figure 1B), prompting immediate CPR and two 200 J defibrillations at 20:21 and 20:23. The patient regained cardiac rhythm, though arrhythmias persisted, and was transferred to the ICU for VA-ECMO treatment in case of recurrent malignant arrhythmias or cardiac arrest.

Upon admission to the ICU at 21:31, the patient was immediately intubated and connected to a mechanical ventilator for assisted ventilation with the following settings: SIMV-VC, FiO_2: 100%, VT: 480 mL, f: 16 bpm, PEEP: 5cmH_2O, PS: 10cmH_2O. Hemoperfusion was also administered to rapidly remove aconitine, and bedside CRRT treatment (CVVH mode) was provided to stabilize the patient's internal environment. Twenty minutes after admission to the ICU (21:51), the ECG monitoring indicated that the patient had ventricular fibrillation, and the pulse was quickly restored after one electrical defibrillation. Considering that the patient might have a malignant arrhythmia or cardiac arrest again at any time, we placed guide wires in the hemofiltration tube and the invasive arterial pressure monitoring catheter to quickly complete the ECMO tube placement. The V-AECMO

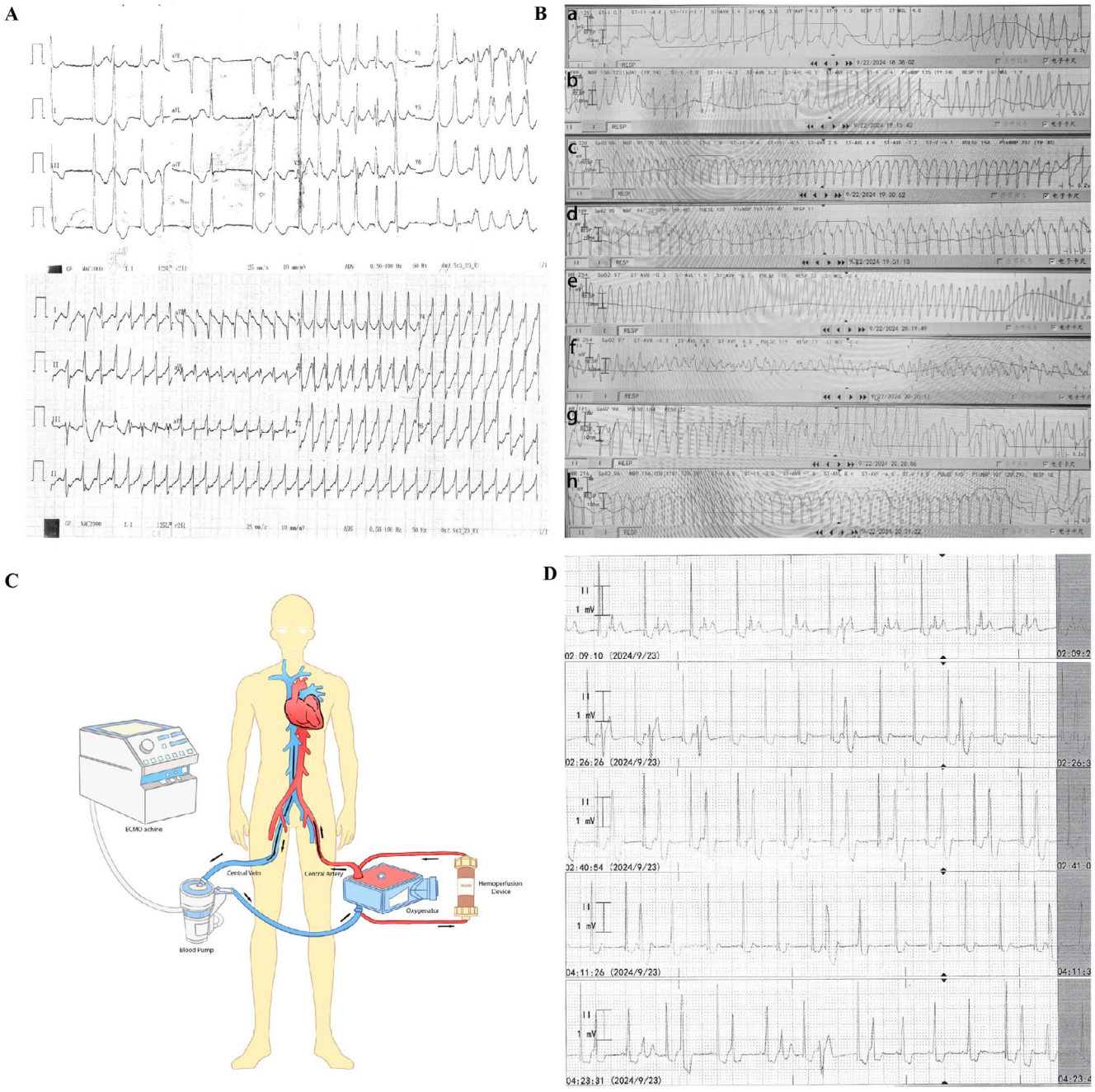

FIGURE 1 | (A) Electrocardiogram revealed multifocal ventricular tachycardia with conduction block, supraventricular tachycardia and premature ventricular contractions. (B) ECG monitoring of patients during emergency treatment: (a, b): Ventricular premature beats, ventricular tachycardia. (c–e): Ventricular tachycardia. (f): Ventricular fibrillation. (g, h): The patient recovered ventricular premature beats and ventricular tachycardia after rescue. (C) CRRT and hemoperfusion are connected in parallel following ECMO to maintain the stability of the patient's internal environment and facilitate the removal of toxic substances. (D) Arrhythmia was gradually improved and ventricular premature beats were reduced after combined treatment of ECMO and hemoperfusion.

machine was installed 42 min after ICU admission (22:13). The tank temperature was 37°C, the ECMO speed was 2900 RPM, the blood flow rate was 2.32 L/min, the gas volume was 2.5 L/min, and the oxygen concentration was 100% (Table 1).

To facilitate hemoperfusion, the research team innovatively used ECMO to power the hemoperfusion process, while CRRT was used in parallel for toxin removal and internal environment stabilization (Figure 1C). Hemoperfusion cartridges were replaced every 2 h, with a total of three cartridges used. The patient was sedated, given analgesia, muscle relaxants, potassium and magnesium supplementation, and lidocaine at 150 mg/h to control the ventricular rate. Norepinephrine (0.6 μg/kg/min) was administered to maintain hemodynamic stability, and target temperature management was applied.

After the combined treatment of ECMO, CRRT, and hemoperfusion, the patient's condition rapidly stabilized, with a significant reduction in poisoning symptoms. The patient returned to sinus rhythm with ventricular premature beats 4.5 h after ICU

TABLE 1 | VA-ECMO treatment parameters and changes in cardiac function and hemodynamic parameters before and after treatment.

ICU stay duration	ECMO speed (RPM)	ECMO flow (L/min)	Gas flow (L/min)	Oxygen concentration (%)	MAP (mmHg)	EF	Left ventricular myocardial contraction function	Right ventricular myocardial contraction function	E/A	E/Ea	Others
3 h	2900	2.32	2.5	100	61	0.21	Decreased contraction function	Decreased contraction function			Left ventricular wall thickening
8 h	2900	2.27	2.5	80	93	\	\	\	\	\	\
13 h	3100	2.5	2.5	60	82	\	\	\	\	\	\
19 h	2940	2.32	2.5	50	91	\	\	\	\	\	\
20 h	2665	1.87	2	45	85	0.4	Mildly decreased contraction function	Decreased contraction function	0.8	12.9	TAPSE: 19 mm
27 h	2720	1.72	2	45	96	\	\	\	\	\	\
35 h	2500	1.55	1.6	40	89	\	\	\	\	\	\
37 h	2195	1.31	1.6	40	94	\	\	\	\	\	\
38 h	\	\	\	\	\	0.42	Mildly decreased contraction function with poor coordination	Decreased contraction function	0.8	8	TAPSE: 19 mm
60 h	\	\	\	\	\	0.53	No significant abnormality	No significant abnormality	0.9	6	TAPSE: 21 mm

admission (September 23, 2022, 2:01 AM), and hemodynamics gradually stabilized, with the heart rate decreasing to approximately 100 beats per minute (Figure 1D). The norepinephrine was gradually discontinued. During this period, the patient's blood pressure was maintained with SBP at 140–160 mmHg and MAP at 80–100 mmHg. After 8.5 h (September 23, 2022, 6:01 AM), ventricular premature beats largely disappeared, and the heart rate stabilized between 70 and 80 beats per minute, allowing for the discontinuation of lidocaine. Continuous ECG monitoring after ICU is getting better (Figure 2A). Echocardiography showed that the patient's cardiac contractility gradually improved, with abnormal ventricular wall motion disappearing 60 h after ICU admission (September 25, 2022, 9:31 AM) and the ejection fraction (EF) increasing from 0.21 to 0.53 (Table 1, Figure 2B).

At this time, the toxicology report confirmed our previous suspicion of Yunaconitine poisoning, as indicated by the toxicology test results from Nanjing Medical University (Figure 3A). During the subsequent treatment, the patient's vital signs remained stable. At 36 h post-ICU admission(September 24, 2022, 9:31 AM), we re-evaluated the patient: MAP was between 80 and 100 mmHg, no vasopressors were used, SpO2 was 100%, ScvO2 was 73.5%, and right radial artery PaO2 was 122.5 mmHg, with an oxygenation index of 306.25. At that point, the patient met the standard criteria for ECMO weaning (Charbonneau et al. 2022): 1. Mean arterial pressure > 70 mmHg; 2. Low-dose vasopressors (inotropic score < 10); 3. SpO2 > 95%; 4. ScvO2 > 70%; 5. Adequate intrinsic pulmonary oxygenation; 6. Improved two-dimensional echocardiography with EF > 25%–30%. We gradually reduced VA-ECMO support (Table 1), and at 40 h post-ICU admission (September 24, 2022, 13:31), ECMO was successfully weaned under DSA guidance. The ventilator was removed at 62 h (September 25, 2022, 11:31 AM), and the endotracheal tube was extubated. ECG indicated (Figure 3B): sinus rhythm, Wolff–Parkinson–White syndrome, counterclockwise rotation, and ST-T changes. The patient was transferred to the cardiology department on day 5 after ICU admission(September 27, 2022) and discharged on day 9 in improved condition(October 1, 2022). A follow-up phone call 1 week after discharge revealed no specific discomfort.

2 | Discussion

Yunaconitine, an alkaloid derived from *Aconitum carmichaelii*, primarily found in the mountainous regions of China, particularly Yunnan Province, is one of the most active components in the plant's roots and rhizomes (Yu et al. 2021). Known for its potent neurotoxicity, Yunaconitine can cause various poisoning symptoms, including nausea, vomiting, limb numbness, dyspnea, and arrhythmias, with symptom severity depending on dosage and individual sensitivity (Li et al. 2023). The toxicological mechanism of Yunaconitine is similar to that of aconitine, primarily affecting voltage-gated sodium channels. Studies have shown that Yunaconitine specifically binds to and activates these channels, leading to abnormal depolarization of cardiomyocytes and neurons, causing malignant arrhythmias, respiratory depression, and other severe physiological reactions. It can also disrupt potassium and calcium ion influx, further exacerbating cardiac and neurological damage (Wang et al. 2021). Currently, no specific antidote exists for Yunaconitine poisoning. In this case, Yunaconitine poisoning presented earlier than typical aconitine poisoning, with more severe neurological and gastrointestinal symptoms, and research has confirmed its higher toxicity (Hai et al. 2017).

In this case, after detailed history-taking and clinical evaluation, the diagnosis of aconitine poisoning was confirmed, accompanied by malignant arrhythmias and hypokalemia.

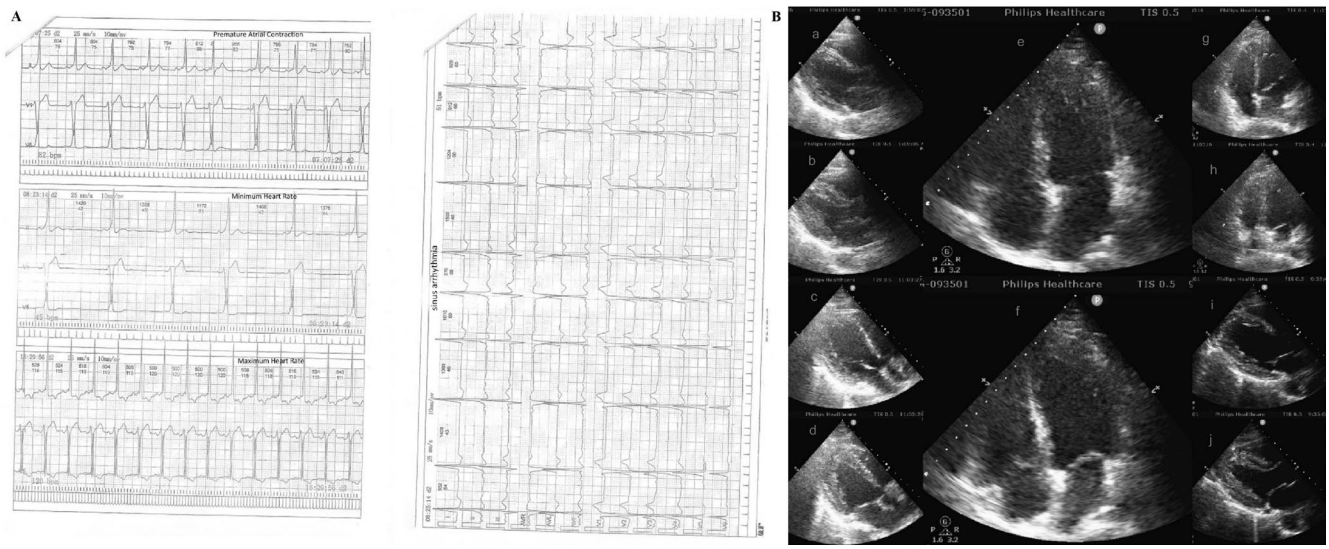

FIGURE 2 | (A) Dynamic electrocardiogram: Sinus rhythm with sinus arrhythmia, Wolff-Parkinson-White syndrome, occasional atrial premature beats, occasional ventricular premature beats. (B) Echocardiography: (a, b): Emergency bedside ultrasound, decreased left ventricular and right ventricular systolic function, left ventricular wall thickening, mild mitral regurgitation, mild tricuspid regurgitation. (LVEF21%). (c–f): Generally slightly reduced and less coordinated left ventricular wall systolic motion amplitude, mitral regurgitation (mild-to-moderate) tricuspid regurgitation (mild), decreased left ventricular systolic function (LVEF42%). (g–j): No significant wall motion abnormalities at rest, tricuspid regurgitation (mild) (LVEF53%).

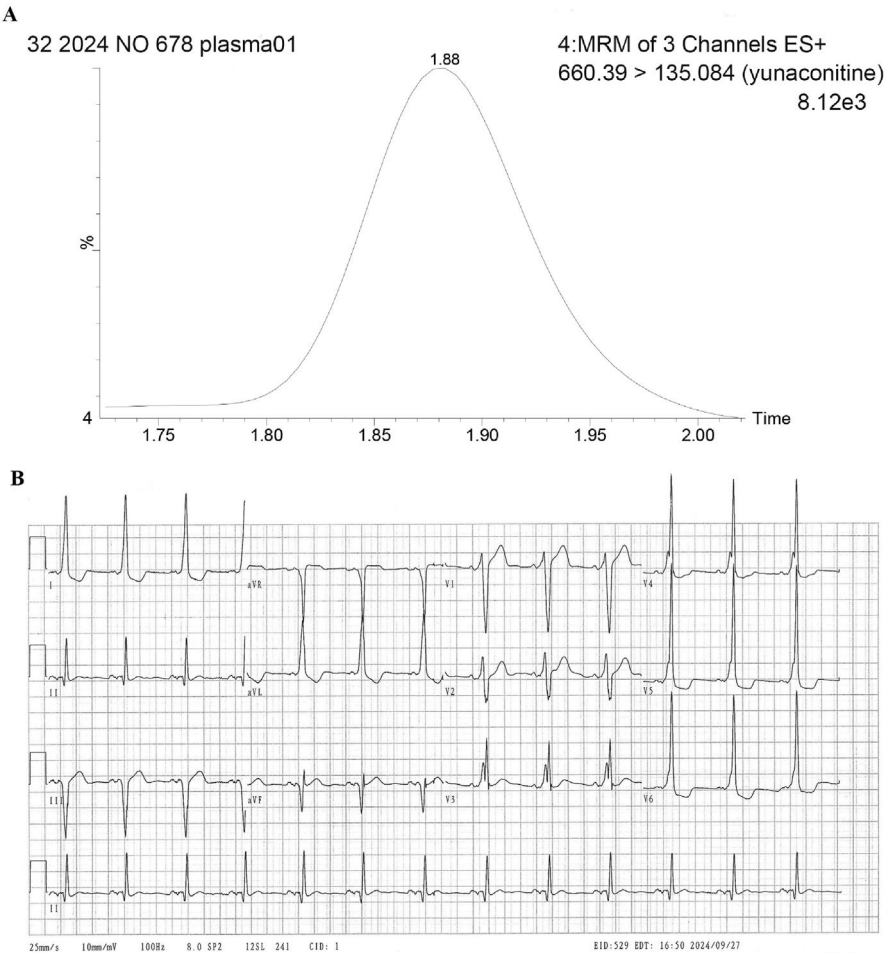

FIGURE 3 | (A) Toxin detection report. (B) ECG on day 5: Sinus rhythm, Wolff-Parkinson–White syndrome, counterclockwise rotation, ST-T changes.

Toxicological tests later identified the toxin as Yunaconitine. For treatment, no specific antidote exists, so supportive care was provided, including cardiac monitoring, electrolyte correction, and symptomatic management. With advancements in medical technology, the combined use of ECMO and hemoperfusion has emerged as a new therapeutic option for severe aconitine poisoning. ECMO provides crucial life support by maintaining cardiopulmonary function, ensuring oxygen supply, and stabilizing circulation, thus reducing the risk of multi-organ failure caused by hypoperfusion. More importantly, ECMO provides a time window for arrhythmia management, allowing the toxin to be metabolized gradually, reducing the risk of ischemic encephalopathy and myocardial injury, thereby lowering the likelihood of fatal complications (Schreiber et al. 2022).

Hemoperfusion, as a technique that uses specific adsorbents to rapidly remove toxic substances from the blood, effectively reduces aconitine concentrations in the body and shortens the duration of its toxic effects. Hemoperfusion also reduces the metabolic burden on organs like the liver and kidneys, lowering the incidence of multi-organ dysfunction syndrome (Chen et al. 2023). Therefore, the combined use of ECMO and hemoperfusion not only provides strong supportive treatment but also effectively removes toxins, significantly improving survival and recovery outcomes in aconitine poisoning cases. Furthermore, we innovatively use ECMO to provide blood flow power to the blood manager during treatment (Figure 1C). The high power provided by ECMO can make the blood manager run smoothly, and the poison and inflammatory factors are removed through hemoperfusion. CRRT is no longer a necessary combination for hemoperfusion. These techniques represent new therapeutic options for severe aconitine poisoning and highlight the potential value of modern medical technology in managing toxic crises.

Research on Yunaconitine poisoning is relatively limited, and clinical experience is still lacking. Therefore, individualized treatment based on specific clinical situations is necessary. In the absence of specific antidotes, the combined use of ECMO and hemoperfusion offers a treatment option for severe aconitine poisoning. ECMO provides critical life support by maintaining cardiopulmonary function, ensuring oxygen supply, and stabilizing circulation, while hemoperfusion rapidly removes toxic substances from the blood using specific adsorbents. According to the study results and the current medical literature, this combined approach can potentially improve the prognosis for patients with malignant arrhythmias due to aconitine poisoning. However, it is important to note that the impact on survival, treatment duration, and costs may vary and should be considered within the context of individual patient cases and treatment settings. The effectiveness of ECMO and hemoperfusion is influenced by several factors, including the timing of intervention, the severity of the poisoning, and the availability of

advanced medical technology. Therefore, while this combined treatment may offer benefits, it is not universally applicable, and its efficacy should be assessed on a case-by-case basis.

3 | Conclusion

In the absence of specific antidotes, the combined use of ECMO and hemoperfusion provides a new treatment option for severe aconitine poisoning. ECMO provides critical life support by maintaining cardiopulmonary function, ensuring oxygen supply, and stabilizing circulation, while hemoperfusion rapidly removes toxic substances from the blood by using specific adsorbents.

Author Contributions

Zhuo Jiang and Yue Zhuang: Conception and design. Xueting Hu and Wei Chen: Administrative support. Xiaoxia Hu and Aixiang Yang: Provision of study materials or patients. Weiyi Tao and Zhuo Jiang: Collection and assembly of data. Yue Zhuang, Aixiang Yang, and Weiyi Tao: Data analysis and interpretation. All authors: Manuscript writing. All authors: Final approval of manuscript.

Acknowledgments

The authors have nothing to report.

Ethics Statement

This study was conducted in accordance with the Declaration of Helsinki. This study was conducted with approval from the Ethics Committee of The Affiliated Suzhou Hospital of Nanjing Medical University, Suzhou Municipal Hospital, Gusu School of Nanjing Medical University, and Suzhou Clinical Medical Center of Critical Care Medicine.

Consent

The manuscript is not submitted for publication or consideration elsewhere.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

All data generated or analyzed during this study are included in this published article.

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