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Combined blood purification and antiarrhythmic therapy for acute aconitine poisoning with refractory arrhythmias: a case-based mechanistic evaluation and treatment strategy optimization

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Abstract

Introduction Aconitine poisoning from traditional Chinese medicine is life-threatening, associated with arrhythmias and shock. Early diagnosis and multidisciplinary treatment are essential due to the lack of specific antidotes. This study aimed to present a severe case of aconitine poisoning and to evaluate the effectiveness of combined blood purification and antiarrhythmic therapy, thereby providing practical insights for clinical management.

Case summary A 70-year-old man presented with coma, recurrent ventricular arrhythmias, and severe hypotension (nadir: 44/24 mmHg) after ingesting Fuzhi (*Aconitum taipaicum*). Toxicology confirmed high aconitine levels. He was treated with norepinephrine (0.17–0.33 µg/kg/min), intravenous amiodarone (150 mg bolus over 10–15 min, then 1 mg/min infusion), electrical cardioversion, and early blood purification (hemoperfusion + CVVH). He regained hemodynamic stability within 24 h and fully recovered at 6 months (LVEF 59–63%).

Conclusions Combined antiarrhythmic therapy and blood purification proved effective. Early recognition and integrated care are key to managing aconitine poisoning.

Keywords Aconitine poisoning, Arrhythmia, Hypotension, Blood purification, Electrical cardioversion, Comprehensive treatment strategy

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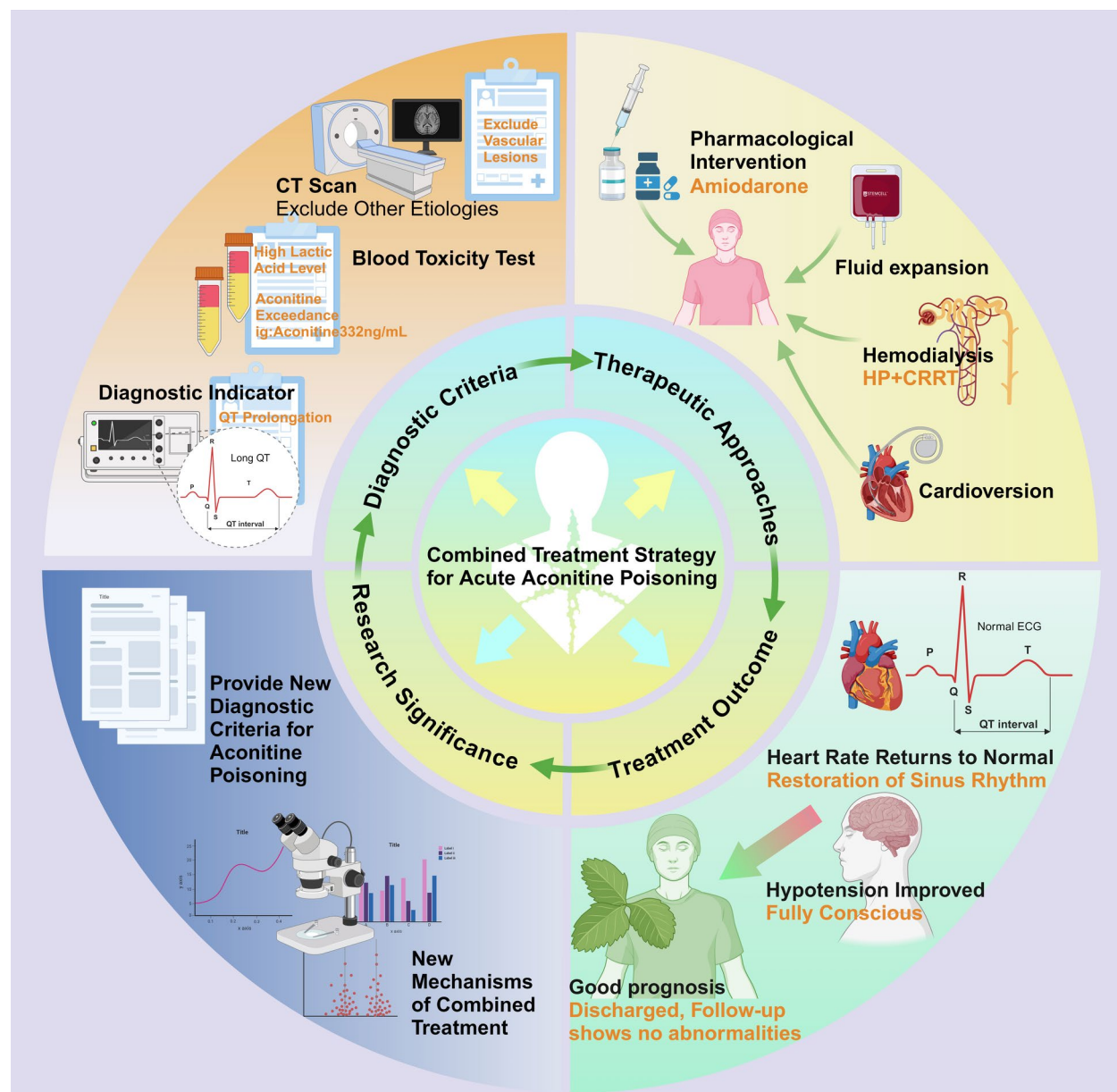
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Graphical abstract



Introduction

Aconite species, such as Chuanwu (*Aconitum carmichaelii*), Caowu (*Aconitum kusnezoffii*), and Fuzhi (*Aconitum taipeicum*), are widely used in traditional Chinese medicine due to their significant pharmacological effects [1]. The alkaloids in these plants exhibit a range of therapeutic properties, including anti-inflammatory, analgesic, cardiotonic, and anticancer

activities, and are commonly used to treat rheumatic diseases, cardiovascular disorders, and certain types of cancer [2]. Particularly in traditional Chinese medicine formulations, Chuanwu and Fuzhi are highly regarded for their synergistic effects in enhancing therapeutic efficacy. However, the toxicity of these substances is substantial [3, 4], with a narrow therapeutic-to-toxic dose range. Even slight mismanagement can result in severe adverse effects.

The toxic mechanism of aconite alkaloids primarily involves the prolonged activation of voltage-gated sodium channels, which leads to sustained cellular depolarization at the plateau level of the action potential. This process disrupts the normal functioning of myocardial cells and the nervous system, manifesting as severe arrhythmias, hypotension, and central nervous system (CNS) depression [5]. Research indicates that the oral dose of aconitine required to induce poisoning is as low as 0.2 mg, with doses exceeding 2–4 mg potentially being fatal [6]. The extremely narrow dose range significantly increases the clinical risks associated with the use of aconite-based medications, particularly when the plant is improperly processed or used incorrectly, making severe poisoning more likely.

In recent years, both isolated and mass-poisoning incidents involving aconite have been frequently reported worldwide [7]. This trend is related to the widespread use of aconite in traditional Chinese medicine and reflects the public’s insufficient awareness of its toxicity [8]. Common causes of poisoning include the ingestion of improperly processed aconite herbs, homemade alcoholic tinctures containing aconitine, and improper use of compound traditional medicines containing aconite alkaloids [8, 9]. Acute aconite poisoning presents diverse clinical manifestations, primarily involving acute cardiovascular damage, which typically includes symptoms such as nausea, vomiting, hypotension, ventricular arrhythmias, and CNS depression [9]. Severe cases may progress to circulatory failure and cardiac arrest, resulting in a high mortality rate [10, 11]. Treatment of poisoning cases generally requires multidisciplinary intervention, but challenges such as delayed diagnosis, limited treatment options, and difficulties in toxin elimination persist.

This report presents a case of acute aconitine poisoning with severe hypotension and coma caused by recurrent ventricular arrhythmias. Through analysis of the clinical course and outcome, combined with a review of relevant literature, this study aims to provide practical guidance for the diagnosis, treatment, and prevention of aconite poisoning and to inform safer clinical use and regulation of aconite-containing medicines.

Case report

A 70-year-old male with a history of hypertension was admitted to the emergency department of the First Hospital of Jiaxing, China, at 10:50 A.M., after 1.5 h of unconsciousness (hospital timeline shown in Fig. 1, Table 1). On admission, vital signs were: temperature 36°C, pulse 81 beats/min, respiration 20 breaths/min, and blood pressure 107/80 mmHg. The patient was comatose (GCS E1V2M4, score 7). Physical examination revealed equal and round pupils with sluggish light reflexes, clear

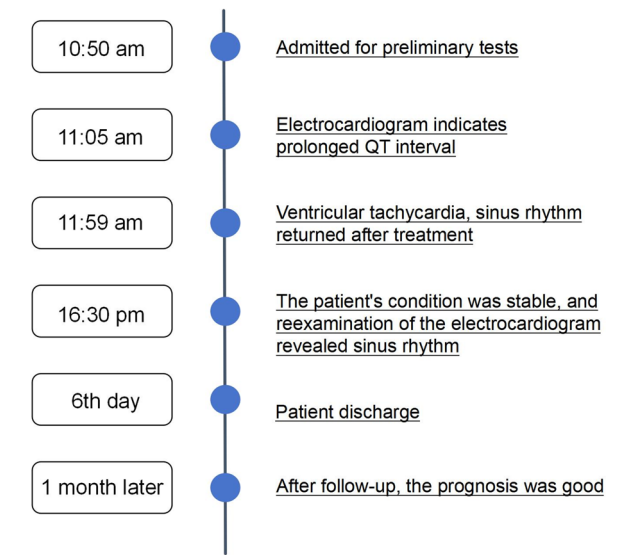


Fig. 1 Patient’s hospitalization timeline

Table 1 Patient timeline

Timepoint	Event
10:50 A.M.	Admission, initial examination
11:05 A.M.	ECG shows prolonged QT interval
11:59 A.M.	Ventricular tachycardia, restored to sinus rhythm after treatment
4:30 P.M.	Condition stabilized, follow-up ECG shows sinus rhythm
Day 6	Patient discharged
1 month later	Follow-up, favorable prognosis

bilateral lung sounds, irregular heart rhythm without murmurs, a soft abdomen, and non-cooperation with abdominal and limb strength testing. Pain stimulation elicited no movement in the left limbs, and bilateral Babinski signs were positive.

Following admission to the intensive care unit, urgent laboratory testing revealed a blood glucose of 6.3 mmol/L, arterial pH 7.334, base excess (BE) – 8.2 mmol/L, and lactate 3.2 mmol/L (Fig. 2, Table 2). D-dimer was 3,180 ng/mL. Liver, kidney function, and cardiac enzymes (troponin I, creatine kinase) were unremarkable. Cranial CT was normal. Given coma, hemiplegia, and bilateral Babinski signs, ischemic stroke was initially suspected. However, after the CT scan, blood pressure dropped to a nadir of 44/24 mmHg, and consciousness fluctuated. Neurology consultation suggested ischemic stroke was unlikely and prioritized the management of hypotension.

To further investigate, contrast-enhanced CT angiography excluded pulmonary embolism, aortic dissection, and

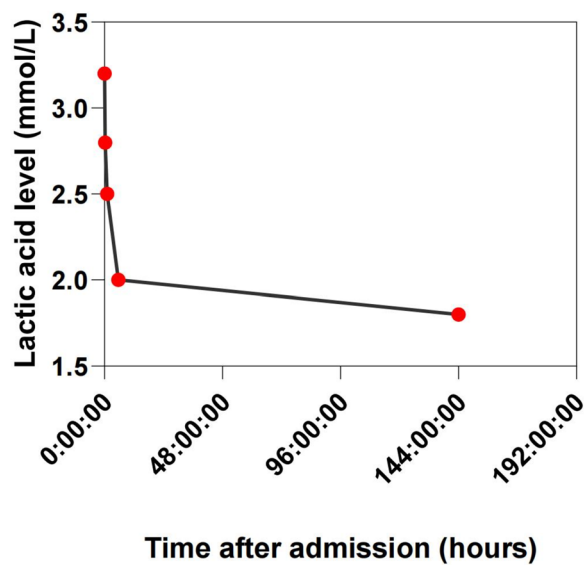


Fig. 2 Changes in lactate levels during the patient's treatment

Table 2 Lactate level changes during treatment

Timepoint	Lactate level (mmol/L)
Admission (day 1)	3.2
11:05 A.M.	2.8
11:59 A.M.	2.5
4:30 P.M.	2.0
Discharge (day 6)	1.8

acute myocardial infarction. Echocardiography showed a full left atrium, mild tricuspid regurgitation, and preserved LVEF (59%) with no regional wall motion abnormalities. Electrocardiogram (ECG, Fig. 3) demonstrated sinus rhythm, frequent multifocal premature ventricular contractions (some in couplets, others with accelerated ventricular escape), ST-T changes, and a prolonged QT interval (QT 520 ms, QTc 560 ms). No torsade de pointes arrhythmias were observed during hospitalization.

Initial treatment included central venous catheterization, fluid resuscitation, and continuous intravenous norepinephrine infusion (0.17–0.33 $\mu\text{g/kg/min}$). The patient regained consciousness and limb strength (grade 4); Babinski's signs became negative. ECG at 11:59 A.M. revealed monomorphic ventricular tachycardia at a rate of 180/min (Fig. 4), which spontaneously resolved to sinus rhythm after ~ 20 s. Amiodarone was administered to prevent recurrent ventricular arrhythmias: an intravenous bolus of 150 mg over 10–15 min, then continuous infusion at 1 mg/min for 24 h. The final diagnosis was malignant arrhythmia causing hemodynamic instability and coma.

To clarify the etiology, the patient's medical history was further reviewed. The patient had no prior heart disease, cardiac enzymes were normal, and no history of taking antibiotics, antihistamines, antidepressants, antifungal agents, or other QT-prolonging drugs was reported. A long history of traditional Chinese medicine use was identified on detailed questioning, including a new formula containing 10 g of Fuzhi (Aconitum) taken 10 days before admission. Fuzhi contains highly toxic diterpenoid

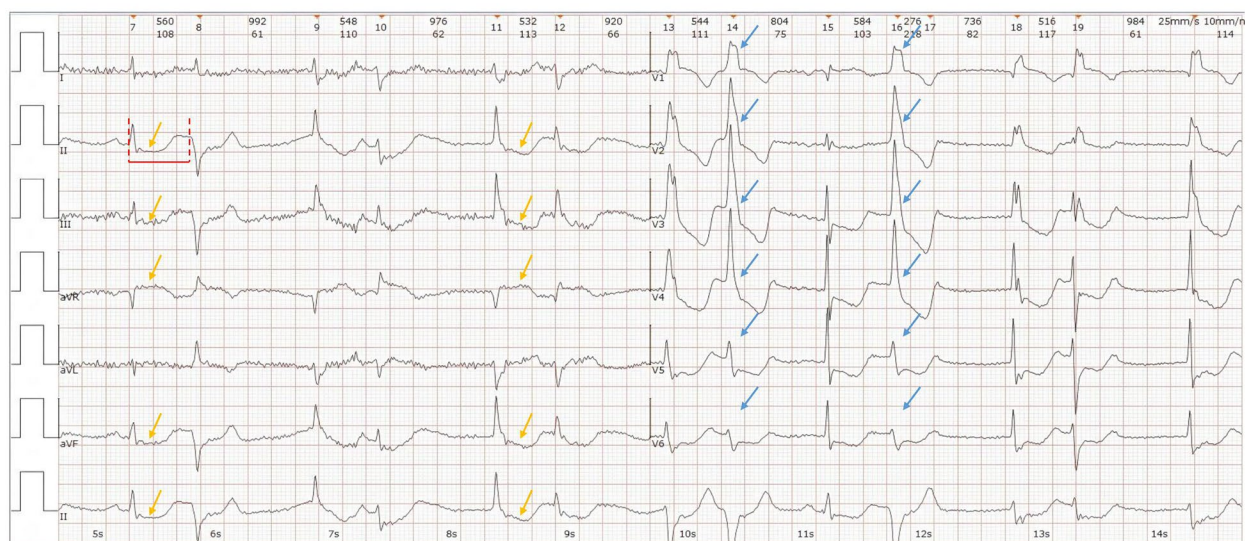


Fig. 3 Patient's ECG (11:05 A.M.). (1) Sinus rhythm; (2) multifocal frequent ventricular premature beats, some in couplets and others associated with accelerated ventricular escape rhythm; (3) ST-T segment changes; and (4) prolonged QT interval. Blue arrows indicate ventricular premature beats, yellow arrows indicate ST-T segment changes, and the red line indicates QT interval prolongation

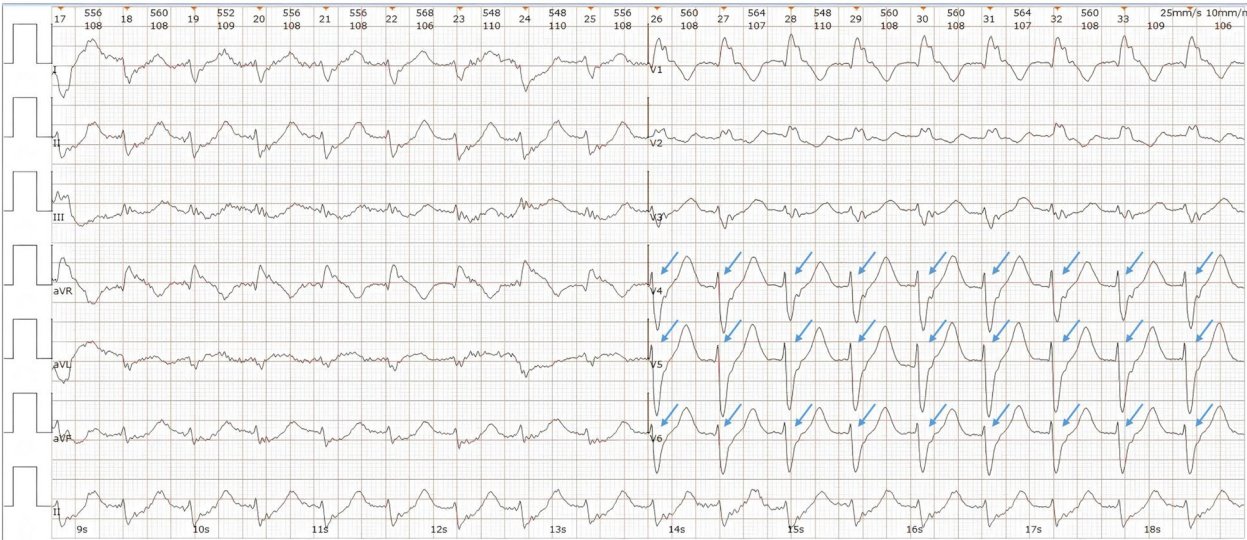


Fig. 4 Patient’s ECG (11:59 A.M.). The blue arrows indicate ventricular tachycardia

alkaloids-aconitine (Fig. 5, Table 3), hyaconitine, and monoacetylaconitine. Toxicological analysis revealed concentrations in the herbal decoction of 332, 936, and 1,760 ng/mL, respectively, all exceeding published toxicity thresholds (> 100, > 100, > 500 ng/mL). Even after 24 h of treatment, urine showed residual toxin (13.3, 32.1, and

65.2 ng/mL), indicating ongoing risk [12]. Combined hemoperfusion (HP) and continuous venovenous hemo-filtration (CVVH) were performed, with plasma toxin levels declining steadily within 24 h (Fig. 6), suggesting enhanced clearance. Considering clinical history, toxin concentrations, and simulated pharmacokinetics, the

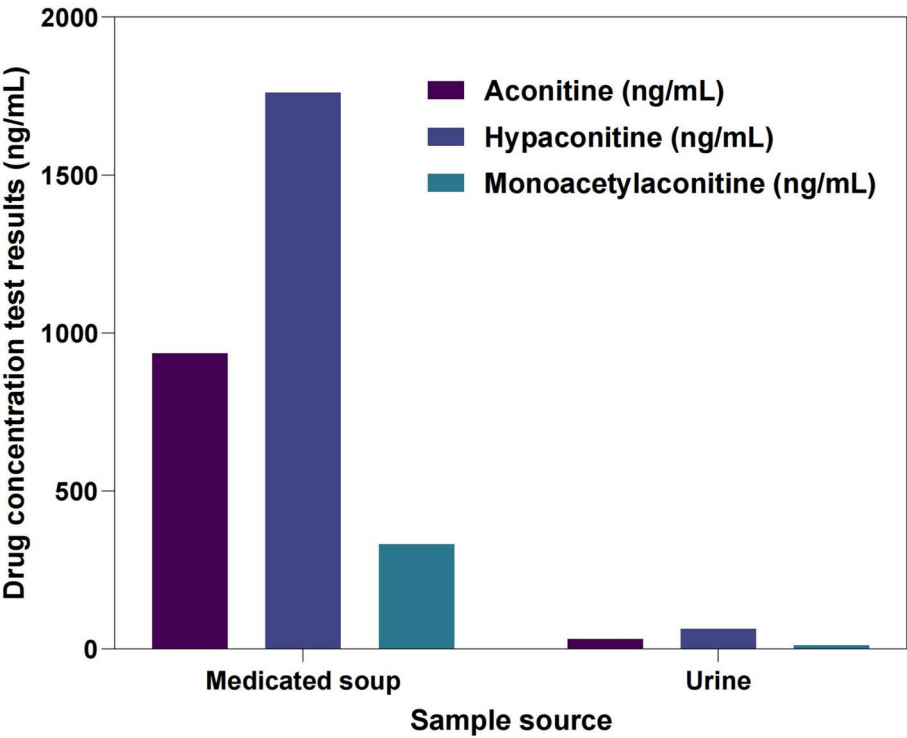


Fig. 5 Medication and urine test results

Table 3 Comparison of drug concentration testing results

Sample source	Pseudoaconitine (ng/mL)	New aconitine (ng/mL)	Aconitine (ng/mL)
Medicinal decoction	936	1760	332
Urine	32.1	65.2	13.3
Reference toxicity concentration	> 100	> 500	> 100

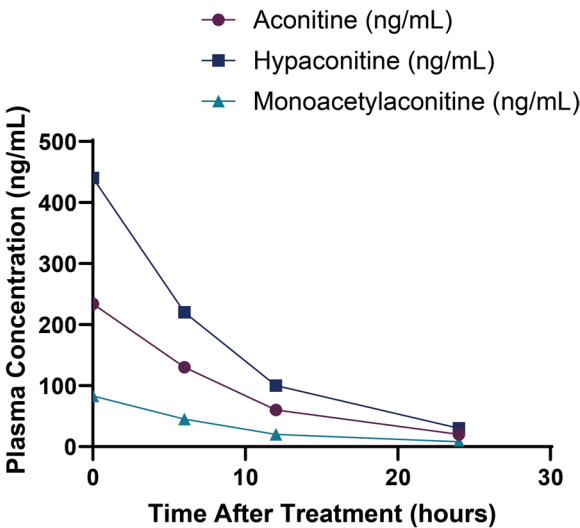


Fig. 6 Trends in plasma concentrations of three aconitine alkaloids following combined hemoperfusion (HP) and continuous venovenous hemofiltration (CVVH) therapy

diagnosis of acute Fuzhi (Aconitum) poisoning-induced malignant arrhythmia was confirmed.

The patient was transferred to the emergency intensive care unit (EICU), where follow-up ECG on the day of admission showed sinus rhythm (Fig. 7), with QT interval 480 ms and QTc 552 ms (Fig. 7). His condition stabilized the following day, allowing transfer to a general ward and discharge on day 6.

Long-term follow-up demonstrated favorable recovery. The patient was asymptomatic 1 month post-discharge, with normal ECG and LVEF of 59%. At 3 months, quality of Life improved, with the ability to perform Light work, sinus rhythm maintained, and LVEF increased to 61%. By 6 months, full recovery of daily activity was achieved, dynamic ECG showed no arrhythmias, and LVEF further improved to 63%. The patient continued oral amiodarone with regular blood level monitoring.

Discussion

Aconitum species, with approximately 350 species globally, are primarily distributed in Asia, followed by Europe and North America [9]. Due to their unique pharmacological properties in traditional Chinese medicine, aconitum plants are widely used in Asia, although less so in Europe and the United States [13, 14]. In China, most aconitum species are found in the Tibetan Plateau and surrounding regions, with fewer in the southeast and Taiwan, leading to regional disparities in clinical experience and prognosis for aconitine poisoning. Multi-system manifestations, including perioral numbness, nausea, vomiting, and chest tightness characterize acute

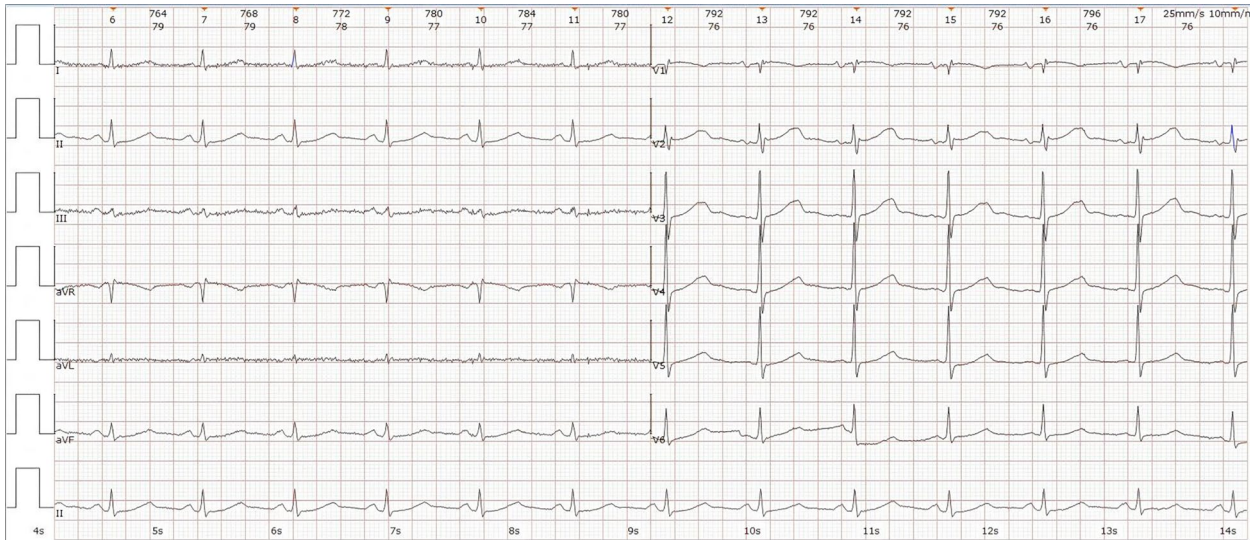


Fig. 7 Patient's ECG (4:30 PM). Sinus rhythm

aconitine poisoning. Severe cases may progress to coma, cardiogenic shock, and even death [9]. Mechanistically, aconitine exerts its toxicity mainly through persistent activation of voltage-gated sodium channels, which prolongs depolarization and disrupts the generation and conduction of action potentials, especially in cardiac and neural cells [10]. This pathophysiological process underlies cardiac arrhythmias and central nervous system hyperexcitability, such as seizures, frequently seen in poisoning cases.

The present patient developed Life-threatening arrhythmias and profound hypotension, consistent with previous reports indicating that approximately 5.5% of hospitalized patients with aconitine poisoning die, with refractory arrhythmias and cardiac arrest being the major causes [8]. A review of recent case reports (Table 4) shows that such presentations are common across age groups and often involve severe outcomes, such as cardiac arrest, shock, and even death, despite aggressive interventions. Sustained depolarization in cardiac cells has been consistently reported, leading to QT prolongation, impaired repolarization, and increased arrhythmogenic risk. In addition, aconitine's excessive activation of vagal nerve activity can cause sinus bradycardia and disrupt the autonomic balance, thereby increasing the risk of arrhythmias and hypotension [8]. Aconitine also induces vasodilation of vascular smooth muscle, decreasing peripheral resistance and contributing to hypotension, which may progress to shock when compounded by impaired cardiac function. The electrocardiographic manifestations in our patient, such as QT prolongation and ventricular arrhythmias, align with prior clinical and experimental observations [10].

Currently, no specific antidote exists for aconitine poisoning. Symptomatic support, reduction of toxin absorption, enhancement of elimination, and blood purification techniques such as hemoperfusion combined with continuous renal replacement therapy (CRRT) have been frequently utilized, especially in severe cases [12, 15, 16]. Early initiation of these measures may accelerate toxin clearance and prevent complications such as acute kidney injury. Antiarrhythmic agents, notably amiodarone and lidocaine, are frequently used to manage refractory ventricular arrhythmias, with some reports suggesting that mexiletine may be more effective than lidocaine, though its use remains limited [17, 18]. Combination therapy is generally more effective than monotherapy [10]. Electrical cardioversion and defibrillation remain vital for managing life-threatening arrhythmias, though restoration of sinus rhythm is not always achieved [9, 19]. In this patient, a transient pacemaker was not applied. The episodes of ventricular tachycardia were short-lasting and spontaneously terminated, and hemodynamic

stability was restored after norepinephrine infusion, amiodarone administration, and blood purification therapy. Therefore, the multidisciplinary team decided not to insert a pacemaker. Nevertheless, transient pacing has been reported as an effective acute intervention in drug-induced QT prolongation complicated by recurrent torsade de pointes or bradyarrhythmia, and it remains a valuable therapeutic option in refractory cases [20].

In recent years, additional therapies such as ginseng have been proposed to enhance detoxification, although the underlying mechanisms remain elucidated [21]. In cases of cardiac arrest unresponsive to standard interventions, prolonged cardiopulmonary resuscitation and extracorporeal life support (ECLS) such as ECMO may be lifesaving [22, 23]. VA-ECMO is particularly recommended for patients with severe circulatory collapse, because it provides respiratory and hemodynamic support. Aconitine is primarily eliminated through renal metabolism, and hypotension may further reduce renal perfusion, leading to acute kidney injury and metabolic acidosis [5, 24]. Elevated lactate and worsening acidosis can create a vicious cycle, aggravating organ dysfunction. Central nervous system depression, often exacerbated by acidosis, may progress from confusion and somnolence to coma, underscoring the importance of prompt recognition and correction of metabolic derangements.

Our patient benefitted from early multidisciplinary interventions, including blood purification, antiarrhythmic therapy, and supportive care, resulting in the resolution of acute symptoms and restoration of normal cardiac function. At 6 months of follow-up, the patient remained free of arrhythmia and exhibited stable cardiac performance. This favorable outcome demonstrates the potential effectiveness of an integrated treatment strategy, consistent with literature advocating for early, comprehensive management of severe aconitine poisoning. Nevertheless, timely diagnosis remains a challenge, and the effectiveness of different interventions requires validation through larger multicenter studies.

This case demonstrates that although multimodal treatment, including gastrointestinal decontamination, pharmacological intervention, blood purification, and cardiac pacing, can delay disease progression somewhat, rapid toxin distribution and irreversible organ damage may still limit therapeutic efficacy. Early in poisoning, aconitine can quickly affect the heart and nervous system, leading to severe arrhythmias, hypotension, and multiple organ dysfunction. To improve patient outcomes, future management should focus on early diagnosis and individualized intervention strategies, such as sensitive toxicological testing, specific antidotes, sodium channel blockers, and timely mechanical support. It should be noted that this report is based on a single case without

Table 4 Literature review summary

Journal	Authors	PMID	Years	Case presentation	Diagnosis	Treatment	Outcome
World J Clin Cases	Liao YP, Shen LH, Cai LH, Chen J, Shao HQ	36,483,800	2022	61-year-old male, severe arrhythmia and cardiogenic shock caused by aconitine poisoning	Aconitine poisoning	Advanced life support, heart transplantation	Recovered after heart transplantation
J Forensic Sci	Huang R, Pang Q, Zheng L, Duan R, Wang Y, Wang Z, Wang T	39,233,350	2024	Male, 50 mL homemade medicinal liquor ingestion leading to cyanosis, arrhythmia, and organ damage	Aconitine poisoning	Toxicological analysis, post-mortem examination	Forensic investigation completed
J Forensic Sci	Ya Y, Zhixiang Z, Chao L, Wei Z, Zhiyong W, Huaifeng C, Shaohua Z, Hongfei X	34,235,734	2021	65-year-old man, accidental death after ingesting Chinese medicinal wine containing aconitine	Aconitine poisoning	LC-MS/MS analysis, supportive treatment	Death by aconitine poisoning
J Nippon Med Sch	Chen X, Wu R, Jin H, Gao R, Yang B, Wang Q	26,568,394	2015	48-year-old male, ingestion of herbal medicinal wine, arrhythmia, and polycystic renal hemorrhage	Aconitine poisoning, arrhythmia	Blood purification, heparin treatment	Recovery with polycystic kidney hemorrhage resolution
J Med Case Rep	Mjølstad OC, Radtke M, Brodtkorb E, Edvardsen F, Brede WR, Aamo TO, Jacobsen D, Stokke MK, Helland A	38,129,927	2023	53-year-old male with recurrent cardiac arrest and neurological symptoms, later diagnosed with aconitine poisoning	Aconitine poisoning	Multidisciplinary treatment, hair analysis confirmation	Full recovery, no further episodes
Hum Exp Toxicol	Lin CC, Phua DH, Deng JF, Yang CC	20,937,638	2011	60-year-old male, chest tightness, elevated troponin I, and abnormal scintigraphy following aconitine ingestion	Aconitine intoxication	Supportive management	Recovery
Perfusion	Zhao Z, Fang Z	39,196,956	2024	Patient in shock, treated with ECMO and hemoperfusion after aconitine poisoning	Aconitine poisoning, shock	ECMO, hemoperfusion, anti-shock therapy	Full recovery after ECMO support
Clin Case Rep	Bonanno G, Ippolito M, Moscarelli A, Misseri G, Caradonna R, Accurso G, Cortegiani A, Giarratano A	32,274,038	2020	Accidental aconitine poisoning, presenting with ventricular tachycardia and cardiac arrest	Aconitine poisoning	Supportive care, no further details	Death
J Pharm Biomed Anal	Huang YF, He F, Cui H, Zhang YY, Yang HY, Liang ZS, Dai W, Cheng CS, Xie Y, Liu L, Liu ZQ, Zhou H	34,814,080	2022	Investigation of four toxic Aconitum alkaloids and their acute toxicity	Toxicological analysis	Animal model, systematic investigation	Understanding toxicity
Drug Test Anal	Bicker W, Monticelli F, Bauer A, Roeder G, Keller T	23,749,589	2013	3 cases of suicidal Aconitum napellus poisoning	Aconitine poisoning	Supportive care, including cardiopulmonary resuscitation and anti-arrhythmic drugs	Fatal; Aconitine as the cause of death

Table 4 (continued)

Journal	Authors	PMID	Years	Case presentation	Diagnosis	Treatment	Outcome
Forensic Sci Med Pathol	Cho YS, Choi HW, Chun BJ, Moon JM, Na JY	31,802,365	2020	81-year-old woman ingested liquid from Aconitum root, presented with ventricular arrhythmias and cardiac arrest	Aconitine poisoning	CPR, anti-arrhythmic drugs	Fatal; Aconitine levels quantified in body fluids

a control group, and the limited sample size cannot fully reflect the clinical heterogeneity of aconitine poisoning or the generalizability of various treatment approaches. The effectiveness and safety of these interventions require validation in larger, multicenter prospective studies, and the underlying mechanisms and long-term outcomes warrant further investigation.

Conclusion

This study presents a case of acute aconitine poisoning and systematically reviews the pathophysiological mechanisms, clinical features, and comprehensive treatment strategies, including pharmacological therapy, blood purification, and electrical cardioversion. The findings emphasize the importance of early recognition and intervention, and demonstrate that a multimodal approach can significantly improve patient prognosis. Electrocardiographic abnormalities are highlighted as early diagnostic markers, supporting the development of standardized diagnostic and therapeutic pathways for aconitine poisoning (Graphic abstract).

However, as this report is based on a single case, the results may not fully reflect the clinical heterogeneity of aconitine poisoning, and the treatment protocol has not been validated in randomized controlled trials. Further research with larger, multicenter studies is necessary to confirm the effectiveness and safety of these strategies. In addition, in-depth investigation into the molecular mechanisms of aconitine toxicity will help identify precise diagnostic biomarkers and novel therapeutic targets, ultimately optimizing the clinical management of toxicological emergencies.

Abbreviations

AKI	Acute kidney injury
BE	Base excess
CRRT	Continuous renal replacement therapy
CNS	Central nervous system
CPR	Cardiopulmonary resuscitation
CVVH	Continuous venovenous hemofiltration
ECG	Electrocardiogram
ECLS	Extracorporeal life support
ECMO	Extracorporeal membrane oxygenation
EICU	Emergency Intensive Care Unit
HP	Hemoperfusion
LVEF	Left ventricular ejection fraction
PCB	Percutaneous cardiopulmonary bypass
VV-ECMO	Venovenous extracorporeal membrane oxygenation
VA-ECMO	Venoarterial extracorporeal membrane oxygenation
GCS	Glasgow Coma Scale

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None.

Author contributions

Cheng Yang and Yuan Suo conceived and designed the study. Cheng Yang, Ting Zou, and Qian-Hui Zang performed the experiments. Wei Cao and Yuan Suo analyzed the data. Cheng Yang and Yuan Suo wrote the manuscript. All authors reviewed and approved the final version of the manuscript.

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Availability of data and materials

All data can be provided as needed.

Declarations

Ethics approval and consent to participate

This study was approved by the Clinical Ethics Committee of The First Hospital of Jiaxing (No. 2024-LY-684). Clinical trial number: Not applicable.

Consent for publication

This retrospective case report was approved by the Clinical Ethics Committee of The First Hospital of Jiaxing (No. 2024-LY-684). Written informed consent for publication was not required as all patient data were anonymized, and no identifiable information was included, in compliance with institutional ethical guidelines for retrospective studies, where contact with the patient or family is impractical.

Competing interests

The authors declare no competing interests.

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