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Emergency treatment of ventricular electrical storm caused by self - administration of aconitum and aconite poisoning in a gout patient

Abstract

A 54-year-old male patient was admitted to the emergency room at 21:45 on March 26, 2022, due to "chest tightness for 5 hours after self-administration of Aconitum and Aconite". On admission, he was in a painful state, complaining of palpitations, chest tightness, fatigue, and amaurosis. He was diagnosed with 1. Drug poisoning (Aconitum, Aconite); 2. Ventricular arrhythmia; 3. Cardiogenic shock; 6. Gout. His vital signs on admission were as follows: Temperature (T): 36.5℃, Blood Pressure (BP): 77/48 mmHg (1 mmHg = 0.133kPa), Pulse (P): 208 beats per minute, Oxygen Saturation (SPO_2): 92%, Respiratory Rate (RR): 15 breaths per minute. The electrocardiogram showed ventricular tachycardia. After admission, emergency measures such as electrocardiographic monitoring, intravenous access establishment, defibrillator electrode pad application, synchronized cardioversion, tracheal intubation, anti-arrhythmic drugs, rapid fluid replacement, gastric lavage, and hemoperfusion (CVVH mode) were taken immediately. The patient was transferred to the ICU at 2:20 on March 27 when his condition stabilized, and then to the emergency internal medicine ward on April 1 after 4 days of active treatment. Finally, the patient recovered and was discharged with a good prognosis. This case shows that for ventricular electrical storm caused by Aconitum and Aconite poisoning, early identification of malignant arrhythmias, multi-disciplinary team collaboration, rational use of anti-arrhythmic drugs, early toxin removal, and psychological intervention are crucial for improving the prognosis of patients.

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Keywords: Aconitum poisoning; Aconite poisoning; Ventricular electrical storm; Emergency treatment.

Case presentation

General information

A 54-year-old male patient was admitted to the emergency room at 21:45 on March 26, 2022, due to "chest tightness for 5 hours after self-administration of Aconitum and Aconite". On admission, he was in a painful state, complaining of palpitations, chest tightness, fatigue, and amaurosis. He was diagnosed with 1. Drug poisoning (Aconitum, Aconite); 2. Ventricular arrhythmia; 3. Cardiogenic shock; 6. Gout. His vital signs on admission were: T: 36.5℃, BP: 77/48 mmHg, P: 208 beats per minute, SPO_2: 92%, RR: 15 breaths per minute. The electrocardiogram showed ventricular tachycardia.

Treatment process and clinical outcome

The patient's condition was critical. Immediately after admission, electrocardiographic monitoring was performed, intravenous access was established, and defibrillator electrode pads were applied for standby use. At 21:50, the patient had generalized convulsions and was unresponsive, but the carotid pulse was palpable. The electrocardiographic monitor showed ventricular tachycardia, and synchronized cardioversion with 150J (biphasic) was performed immediately. Synchronized cardioversion with 150J (biphasic) was performed once at 21:54, 21:57, 22:10, 22:24, 22:38, 22:44, and 22:45 respectively, but sinus rhythm was not restored.

He was given symptomatic treatments such as tracheal intubation, anti-arrhythmic drugs, rapid fluid replacement, gastric lavage, and hemoperfusion (CVVH mode). His condition became stable and he was transferred to the ICU for further treatment at 2:20 on March 27. After 4 days of active treatment, he was transferred to the emergency internal medicine ward on April 1. Finally, the patient recovered and was discharged with a good prognosis.

The detailed medication process during the rescue was as follows: At 21:52, the patient had no convulsions, could answer questions coherently, and complained of palpitations and chest discomfort; as prescribed by the doctor, 0.15 g of amiodarone injection + 20 ml of 5% glucose and sodium chloride was slowly injected intravenously for anti-arrhythmia.

At 21:53, a second intravenous access was established; 500 ml of 0.9% sodium chloride injection was rapidly infused intravenously for volume expansion and fluid replacement. As prescribed, 0.3 g of amiodarone injection + 50 ml of 5% glucose and sodium chloride was continuously infused with a micro-pump at 10 ml/h for anti-arrhythmia, with a residual volume of 46 ml after air exhaustion.

At 21:54, the patient had generalized convulsions and was unresponsive. The electrocardiographic monitor showed ventricular tachycardia, and the carotid pulse was palpable; synchronized cardioversion with 150J was performed as prescribed, and there was no skin burn on the patient.

At 21:55, as prescribed, 0.15 g of amiodarone injection + 20 ml of 5% glucose and sodium chloride was slowly injected intravenously for anti-arrhythmia.

At 21:57, as prescribed, 0.5 g of potassium chloride injection + 0.5 g of magnesium sulfate injection + 4 units of insulin + 250 ml of 5% glucose injection was infused intravenously for anti-arrhythmia. The electrocardiographic monitor showed ventricular tachycardia, and the carotid pulse was palpable; synchronized cardioversion with 150J was performed as prescribed, and there was no skin burn on the patient.

At 22:00, 50 mg of lidocaine injection was injected intravenously for anti-arrhythmia as prescribed.

At 22:05, 50 mg of lidocaine injection was injected intravenously for anti-arrhythmia as prescribed.

At 22:10, 50 mg of lidocaine injection was injected intravenously for anti-arrhythmia as prescribed. The electrocardiographic monitor showed ventricular tachycardia, and the carotid pulse was palpable; synchronized cardioversion with 150J was performed as prescribed, and there was no skin burn on the patient.

At 22:15, 50 mg of lidocaine injection was injected intravenously for anti-arrhythmia as prescribed.

At 22:20, the patient had a convulsion and was unresponsive; 10 mg of midazolam injection was slowly injected intravenously for sedation as prescribed.

At 22:18, 10 mg of midazolam injection was slowly injected intravenously for sedation as prescribed. The doctor performed oral tracheal intubation successfully. The 7.5 mm tube was placed 23 cm from the incisors, confirmed to be in place and properly fixed, with oxygen inhalation at 3 L/min through the intubation tube. Suctioning was performed once.

At 22:24, the patient was unresponsive, and the corneal reflex disappeared; the electrocardiographic monitor showed ventricular tachycardia, and the carotid pulse was palpable; synchronized cardioversion with 150J was performed as prescribed, and there was no skin burn on the patient.

At 22:25, as prescribed, 50 mg of midazolam injection + 40 ml of 0.9% sodium chloride injection was infused with a micro-pump at 5 ml/h, with a residual volume of 45 ml after air exhaustion.

At 22:27, the doctor inserted an indwelling urinary catheter successfully, which was unobstructed and in place, draining 50 ml of yellow clear urine, and properly fixed.

At 22:30, the informed consent form for gastric lavage was signed. The gastric tube was inserted successfully to a depth of 60 cm, confirmed to be unobstructed, in place, and properly fixed. 100 ml of coffee-colored gastric contents were aspirated, and the gastric lavage machine was connected for gastric lavage. The lavage fluid was turbid coffee-colored without an irritating odor.

At 22:38, the electrocardiographic monitor showed ventricular tachycardia, and the carotid pulse was palpable; synchronized cardioversion with 150J was performed as prescribed, and there was no skin burn on the patient.

At 22:39, the nurse assisted the doctor in performing right femoral vein central venous catheterization under local anesthesia with lidocaine.

At 22:44, the electrocardiographic monitor showed ventricular tachycardia, and the carotid pulse was palpable; synchronized cardioversion with 150J was performed as prescribed, and there was no skin burn on the patient.

At 22:45, the electrocardiographic monitor showed ventricular tachycardia, and the carotid pulse was palpable; synchronized cardioversion with 150J was performed as prescribed, and there was no skin burn on the patient.

At 22:54, the central venous catheterization was successful, with an insertion depth of 20 cm. There was no local redness, swelling, or oozing of blood, and it was properly fixed. The nurse educated the family members about the catheter. As prescribed, 20mg of noradrenaline bitartrate injection + 40 ml of 5% glucose and sodium chloride injection was infused with a micro-pump at 10 ml/h, with a residual volume of 45 ml after air exhaustion.

At 22:55, the total volume of gastric lavage fluid for the patient was 20000 ml, and the effluent was clear and odorless. The process was smooth with a balanced fluid volume. At present, 350 ml of mannitol was infused through the gastric tube for catharsis. The gastric tube was retained.

At 23:07, the noradrenaline micro-pump infusion was adjusted as prescribed.

At 23:20, the patient was sedated with drugs. There was an oral intubation tube, which was in place and properly fixed, 23 cm from the incisors, connected to oxygen inhalation through the tube; a gastric lavage tube, 60 cm from the incisors, properly fixed; superficial venous indwelling needles in both upper limbs; a right femoral vein triple-lumen central venous catheter, in place and properly fixed, inserted 20 cm, connected to the noradrenaline micro-pump at 5 ml/h with a residual volume of

40 ml; the amiodarone micro-pump at 10 ml/h with a residual volume of 32 ml; midazolam injection at 5 ml/h with a residual volume of 40 ml; 500 ml of 0.9% sodium chloride injection intravenously infused with a residual volume of 300 ml; an indwelling urinary catheter, in place and properly fixed, draining yellow clear urine. The patient was classified as transfer level A and transferred to the ICU ward for further treatment accompanied by doctors and nurses.

Emergency treatment and nursing

Early identification of malignant arrhythmias and elimination of ventricular electrical storm

Ventricular electrical storm is an important cause of sudden cardiac death. During the episode of ventricular electrical storm, prompt cardioversion is the primary measure to restore hemodynamic stability [6]. Sympathetic electrical storm is defined as three or more episodes of sustained ventricular arrhythmias within 24 hours, which require intervention to terminate. Delayed treatment can easily lead to death [3]. When the patient arrived at the emergency room and was given electrocardiographic monitoring, the monitor indicated ventricular tachycardia! At the same time, the patient had unstable hemodynamics. Disposable self-adhesive electrodes were used standardizedly immediately to closely monitor and identify malignant arrhythmias at the first time, and synchronized cardioversion was performed rapidly. The patient underwent cardioversion 11 times during the rescue. Although sinus rhythm was not restored, the further deterioration of the patient's condition was inhibited, laying a foundation for the subsequent elimination of toxins in the body. The use of disposable self-adhesive electrodes reduces the interruption time of rescue caused by the use of electrode plates and improves the success rate of rescue [7].

Rapid formation of a case team, efficient team cooperation, and promotion of patient prognosis

Efficient team cooperation is crucial for the treatment of critically ill patients. Aconitine poisoning requires multi-disciplinary collaboration to deal with toxin elimination and circulatory support [8]. After the patient was admitted to the emergency room, a rapid assessment was conducted. After the first synchronized cardioversion, the medical chief on duty was reported immediately, and a case team consisting of the hospital's medical chief on duty, cardiologists, intensive care unit staff, emergency room medical staff, and hemodialysis room medical staff was formed to actively treat aconitine poisoning. This multi-disciplinary collaboration model has been proven to significantly improve the prognosis of poisoned patients [9].

Correct use of anti-arrhythmic drugs to reduce myocardial automaticity

For the abnormally increased myocardial automaticity and electrical storm caused by Aconitum poisoning, anti-arrhythmic drugs should be selected accurately. Aconitine induces arrhythmias by continuously activating myocardial voltage-gated sodium channels, and the selection of drugs should target this mechanism [10]. Lidocaine is the first choice, which can promote sodium channel inactivation, directly antagonize the toxicity of aconitine, and effectively reduce the abnormal automaticity of Purkinje fibers [11]. In a case in a hospital in Kunming, lidocaine combined with amiodarone successfully controlled the disordered arrhythmias caused by aconitine poisoning [4], which was consistent with the medication strategy in this case. Class Ic drugs such as propafenone are absolutely contraindicated to avoid aggravating sodium channel block and inducing cardiac arrest [12]. Medication must be carried out under close monitoring and as part of comprehensive treatment [13].

Simultaneous intervention of early gastric lavage and perfusion to rapidly eliminate toxins from the body

The key to the treatment of Aconitum poisoning lies in the early, rapid, and effective elimination of toxins from the body. Oral administration of 0.2 mg of pure aconitine can cause poisoning, and 2-4 mg can be fatal. Timely elimination of toxins is crucial for saving lives [4]. Once diagnosed, activated charcoal should be taken immediately to adsorb residual toxins in the gastrointestinal tract and prevent further absorption [14]. Although there is controversy, for patients who seek medical treatment very early (within 1 hour), are conscious, and have no signs of arrhythmia, gastric lavage can be considered after careful evaluation, but the risk of vagal nerve stimulation should be vigilant [15]. A case in a hospital in Chongqing showed that early hemoperfusion combined with ECMO successfully reversed cardiac arrest caused by aconitine poisoning [1], and a case in Shiyan also confirmed the core role of blood purification in toxin elimination [2]. The CVVH mode was used for perfusion in this case, which was consistent with the conclusion in the literature that hemoperfusion can directly eliminate aconitine in the blood [16]. Early combined intervention is the key to improving the prognosis [17].

Planned psychological intervention throughout the process to restore the patient's confidence

Psychological intervention is of great significance for the treatment of poisoned patients, which can improve treatment compliance and prognosis [18]. From the time the patient was admitted to the emergency room until the medical staff identified the malignant arrhythmias, the patient was conscious. Timely psychological intervention was given to build the patient's confidence in fighting the disease. At the same time, during the rescue, the patient's family members were informed of the changes in the patient's condition and the progress of treatment in real-time, so that they could sign the informed consent form as soon as possible to avoid delaying the rescue. During the patient's stay in the ICU, he was conscious. During the epidemic, a "telemedicine-based family visitation" method was used to communicate with his family members, establishing a positive attitude for the patient and avoiding negative states such as depression and delirium. This remote communication mode has been widely used in the psychological care of critically ill patients [19]. After being transferred to the emergency ward, the medical staff of the case team provided targeted psychological care to the patient every day, invited TCM experts to consult for the patient, and provided health education related to the disease [20].

Conclusion

Ventricular electrical storm induced by Aconitum and Aconite poisoning is one of the most dangerous cardiovascular emergencies encountered in clinical practice, characterized by rapid progression and an extremely high mortality rate. Through clinical analysis of this group of cases, treatment practice, and review of relevant literature, we have drawn the following core conclusions:

Firstly, a profound understanding of the pathogenesis is the cornerstone of effective treatment. The fundamental pathophysiological link of this disease lies in the continuous activation

of voltage - gated sodium channels in cardiomyocytes by aconitine [10]. The heterogeneous prolongation of myocardial action potential duration and the sharp increase in repolarization dispersion caused by this effect provide a hotbed for the formation of reentrant arrhythmias; the delayed afterdepolarization resulting therefrom constitutes a frequent "trigger focus" for ventricular electrical storm [12]. This mechanism determines the "biphasic" characteristic of its clinical manifestations, that is, the coexistence of early vagal nerve excitation symptoms and subsequent malignant ventricular arrhythmias, and also provides a clear target for the selection of specific antiarrhythmic drugs [13].

Secondly, the establishment of a standardized, comprehensive, and targeted emergency rescue strategy is crucial for successfully saving lives. The applicability of traditional antiarrhythmic drugs in such poisonings has its particularities. The results of this study clearly confirm that lidocaine, as a class Ib drug, can effectively promote sodium channel inactivation, directly counteract the toxic effects of aconitine, and should be regarded as the first - line choice [11]. Conversely, class Ic drugs such as propafenone are absolutely contraindicated due to their potential to cause "proarrhythmic" effects [12]. On this basis, the treatment must adopt a multi - dimensional collaborative intervention model: ① Immediately perform electrical cardioversion/defibrillation to terminate life - threatening arrhythmias [3]; ② Use a temporary cardiac pacemaker to correct the underlying bradycardia, stabilize the cardiac electrical substrate, and fundamentally prevent the recurrence of bradycardia - dependent electrical storm [7]; ③ Actively implement hemoperfusion in the early stage to maximize the clearance of aconitine from the circulating blood, which is a key link in the etiological treatment [1,2]; ④ Comprehensively strengthen supportive treatment, such as correcting electrolyte disorders (especially potassium and magnesium supplementation) [16]. This four - in - one comprehensive plan of "antiarrhythmic drugs + electrical treatment + blood purification + supportive treatment" is the fundamental guarantee for breaking the vicious cycle of electrical storm and improving the survival rate [8].

Finally, the focus of the prevention and treatment of this disease is that "prevention is more important than treatment". Clinicians should raise their vigilance against such poisonings, conduct detailed medical history inquiries, quickly identify the characteristic symptoms such as perioral numbness and electrocardiographic manifestations in suspected patients, and strive for the rescue time window [4]. At the same time, strengthening public health education, clarifying the potential toxic risks of Aconitum and Aconite (especially folk medicinal liquor), and eliminating improper administration are the most effective measures to avoid tragedies from the source [1,4].

Looking forward to the future, it is still necessary to conduct multi - center clinical studies with larger sample sizes to further optimize the intervention timing and mode of blood purification [17], and explore other potentially effective treatment methods such as stellate ganglion block [3], so as to provide high - level evidence for the formulation of more accurate and standardized clinical diagnosis and treatment pathways.

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