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Cardiotoxic effects of poisoning from aconite root in a middle-aged man in Bhutan: a case report

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ABSTRACT

Introduction: Poisoning is a major global health concern and a preventable cause of death. Aconite, an alkaloid found in the aconitum genus frequently leads to poisoning in Asian countries including Bhutan owing to its use in traditional medicine and religious items. **Case presentation:** A 47-year-old male consumed a tincture of aconite plant root and experienced vomiting, blurred vision, paraesthesia, and light headedness. On presentation to the emergency department, his vitals were normal but on subsequent close monitoring, he had premature ventricular contractions and sinus bradycardia leading to hypotension and shock. He was admitted to the intensive care unit for continuous cardiac monitoring and supportive treatment including atropine and noradrenaline. He reported subjective improvement, his vital signs improved and ECG returned to normal, enabling discharge after 5 days of admission. **Conclusion:** There is no specific antidote to aconite toxicity so the treatment is mainly supportive. It is imperative to implement proactive measures such as public awareness programs and dedicated research efforts to mitigate the risk of inadvertent toxicity resulting from aconite use.

Keywords: Aconite; arrhythmia; cardiotoxicity; neurotoxicity; plant poisoning

INTRODUCTION

Poisoning represents a common preventable cause of morbidity and mortality worldwide. According to the World Health Organization, unintentional poisonings were responsible for about 84,000 deaths in 2019[1]. Aconite, also called Aconitum, is an alkaloid found within the Actinium genus. The plant is also referred to as monkshood, wolfsbane, or the queen of all poisons. Aconite poisoning is predominantly reported in Asian countries like China, India, and Japan due to its historical use in traditional medicine. Between 2001 and 2010, approximately 5000 instances of aconite poisoning were documented across these nations[2]. Bhutan also witnesses aconite poisoning given its centuries-old utilizations as both poison and an ingredient in traditional medicines. The clinical manifestations of aconite toxicity can vary widely and may lead to severe outcomes including fatality.

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We report a case of aconite poisoning in a lay monk who presented in June 2023 to our hospital in Gelephu, Bhutan.

CASE PRESENTATION

A 47-year-old male, who was previously well with no known comorbidities presented to the emergency department with vomiting, blurring of vision, paraesthesia, and light-headedness. He had ingested a tincture of aconite plant root (Figure 1) approximately 4 hours prior to hospital presentation. He was a lay monk and ingested the plant root to check if it was viable before offering it on the altar.

Figure 1. A small piece of the root of Aconite plant that had been collected from the forest for the purpose of adding it as an ingredient to religious ceremonial items (Photo shared by the victim)

On admission, his Glasgow Coma Scale score was E4V5M6 (15/15). He was diaphoretic, agitated, and anxious. His pulse rate was 70 beats per minute, blood pressure was 120/80 mmHg, and peripheral oxygen saturation was 100% on room air. Cardiovascular examination revealed normal heart sounds. The respiratory system examination showed bilateral vesicular breath sounds. The abdomen was soft and non-tender, with no organomegaly and normal bowel sounds. The nervous system examination revealed no focal neurological deficits.

On presentation, a 12-lead electrocardiogram (ECG) showed junctional rhythm with premature ventricular complexes. (Figure 2).

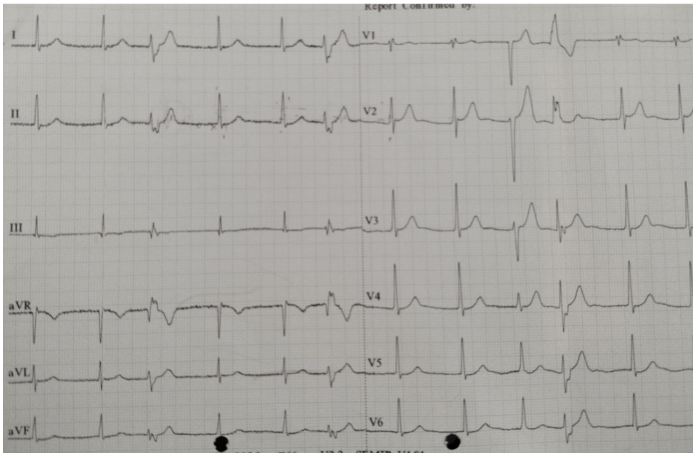

Figure 2. ECG at presentation: junctional rhythm with bidirectional premature ventricular complexes.

A comprehensive blood workup (Table 1) revealed values within the normal range.

Table 1. Summary of laboratory investigation findings of a 47-year-old make treated for cardiotoxicity following consumption of root of aconite plant.

Laboratory investigations	Test results	Reference range
Complete blood count		
White cell count (*10^3/µL)	8.30	4.0-10.0
Lymphocyte (%)	26	20-40
Neutrophil (%)	69	40-60
Haemoglobin (g/dl)	14.7	14.0-18.4
Platelet (10^3/µL)	238	150-450
Liver function test		
AST (IU/L)	27	0-35
ALT (IU/L)	40	0-45
Total Bilirubin (mg/dL)	1.4	0.2-1.0
Direct Bilirubin (mg/dL)	0.3	0.0-0.2
Renal function test		
Urea (mg/dL)	28	15-45
Creatinine (mg/dL)	1.0	0.6-1.3
Electrolytes		
Sodium (mEq/L)	148	135-145
Potassium (mEq/L)	3.0	3.6-5.0
Chloride (mEq/L)	115	96-110
Coagulation studies		
PT (s)	15.7	13.6-17.5
INR	1.2	0.8-1.2
Venous blood gas		
pH	7.45	7.35-7.45
pCO2 (mmHg)	36.8	35-45
pO2 (mmHg)	37.9	95-100
HCO3 (mEq/L)	24.7	22-26

Considering the history of aconite ingestion followed by symptoms suggestive of cardiotoxic effects, the patient was admitted to the ICU for continuous ECG monitoring. During the course of monitoring at the ICU, he developed a syncopal episode during which his heart rate dropped to 40 beats per minute with a blood pressure of 80/60 mmHg. ECG at that point revealed sinus bradycardia (Figure 3). In response, two doses of intravenous atropine 0.6mg each were given, five minutes apart. Due to persistent hypotension despite a 1000ml bolus of crystalloids, noradrenaline infusion (0.1mcg/kg/min) was started and continued for about 12 hours to maintain mean arterial pressure > 65 mmHg. Although the choice of drug for chemical pacing is either adrenaline or dopamine, noradrenaline was the only agent available at the time. He had about 20 bouts of vomiting resulting in hypochloraemia and hypokalaemia.

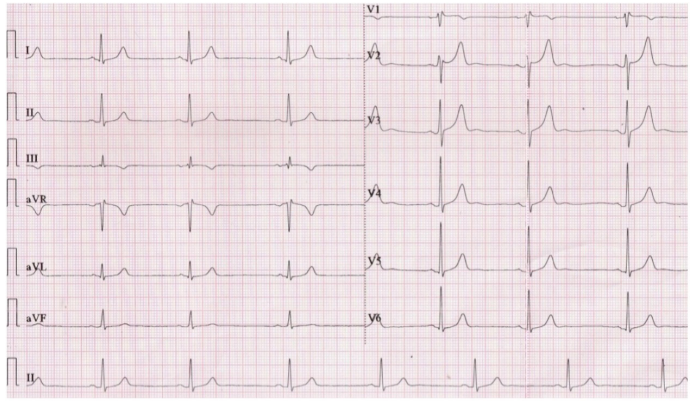

Figure 3. ECG showing sinus bradycardia

Forty-eight hours into ICU admission, vomiting had resolved and vital signs had returned to normal. Noradrenaline infusion was tapered off. Continuous monitoring of ECG revealed the presence of sinus arrhythmia. He was transferred to the Medicine Ward for observation and was discharged home 3 days later. At his one-week follow-up at the outpatient clinic, he was symptom free, his ECG had reverted to normal sinus rhythm (Figure 4) and 2D echocardiography showed normal cardiac function.

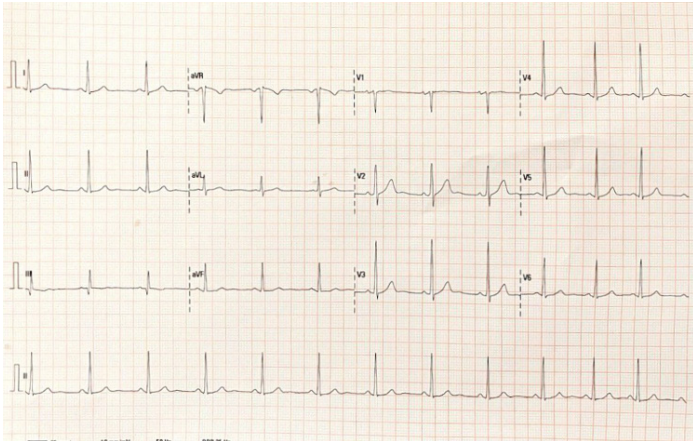

Figure 4. ECG on one-week follow-up: Normal sinus rhythm

DISCUSSION

The scientific name of aconite or monkshood is Aconitum. This group comprises more than 250 species of flowering plants that belong to the Renunculaceae family. Aconite is utilized in medicinal practices in Asian countries like China, Japan, India, and Bhutan. In Bhutan, it is referred to as Tsenduk, Menchen, or Bonga. Various parts of the aconite plant are used for different purposes, primarily in traditional medicine practices. It is used as a constituent in over 25 medicinal preparations of Bhutanese traditional medicines that are used for their analgesics and anti-inflammatory properties[3]. It is also used for other purposes such as poison in hunting wild animals and offerings during rituals. The toxicity of the extracts corresponds to its alkaloid content that is present in its roots, flowers, leaves, and stems. The most common route of exposure is oral ingestion.

Aconite poisoning is primarily diagnosed on clinical grounds. It is suspected in patients with a history of consuming parts of the plant and exhibiting symptoms of toxicity. Aconite is a rapidly acting toxin, and individuals who have been poisoned by it typically exhibit a constellation of symptoms encompassing neurological, cardiovascular, and gastrointestinal manifestations[4]. Neurological symptoms such as paraesthesia, numbness, and burning sensations in the lips, tongue, and mouth are felt almost immediately after ingestion. These are followed by cardiovascular symptoms like palpitations, syncope, and dizziness. Gastrointestinal symptoms, including nausea, vomiting, abdominal pain, and diarrhea, may also occur. Additionally, individuals may experience visual blurring, yellow-red color vision distortion, and coordination difficulties. Severe cases may lead to refractory arrhythmias and refractory hypotension, potentially resulting in sudden death. Literature reports an in-hospital mortality rate of 5.5% with acute aonite poisoning[5]. Two cases of unintentional aconite poisoning with cardiotoxicity were reported in Bhutan following the consumption of aconite-containing traditional medicine[3].

C19-diterpenoid alkaloids, notably aconitine, mesaconitine, and hypaconitine, constitute the primary toxic components within aconite roots[4,6]. These potent alkaloids exert their toxicity by targeting voltage-sensitive sodium channels present in excitable tissues such as the myocardium, nerves, and muscles. This action leads to the prolonged activation of these channels, preventing their normal inactivation process. Consequently, there is a continuous influx of sodium, resulting in sustained depolarization that renders the tissue refractory to further excitation. Furthermore, aconite inhibits Sodium Potassium ATPase, prolonging the influx of sodium and sustaining depolarization. Activation of the Sodium Calcium Exchanger by aconite leads to an increase in cytoplasmic calcium levels, disrupting excitation-contraction coupling and precipitating arrhythmias[7].

Aconite's interaction with the human ether-a-go-go related gene channel (hERG) influences action potential duration; its blockade of hERG channels prolongs action potential duration, contributing to arrhythmias such as ventricular ectopics, ventricular tachycardia, torsades de pointes, ventricular fibrillation, and even death[7]. Aconite's propensity to induce irregular heart rhythms has also been linked to its anticholinergic effects, mediated through the vagus nerve.

Furthermore, aconite also acts on the ventromedial nucleus in the hypothalamus which regulates the autonomic nervous system causing bradycardia and hypotension[6].

Toxicological analysis of aconite can be performed to detect the presence of aconitine or its metabolites using techniques such as liquid chromatography mass-spectrometry and immunoassays[8]. However, it is not available in Bhutan and diagnosis is mainly based on clinical grounds.

There is no specific antidote to aconite poisoning and management is mostly supportive. Hypotension can be managed with the administration of intravenous fluids and vasopressors. Various arrhythmias necessitate tailored management approaches. Bradycardia is typically addressed with atropine. For ventricular arrhythmias linked to aconite poisoning, amiodarone and flecainide as preferred first-line antiarrhythmic agents[9,10]. In cases of polymorphic ventricular tachycardia, magnesium sulphate is recognized as a potential treatment option[11]. In cases of ventricular arrhythmias that are refractory to initial intervention including cardioversion and anti-arrhythmic drugs, systemic circulation can be maintained using percutaneous cardiopulmonary bypass like portable extracorporeal membrane oxygenation devices or ventricular-assist devices[11].

CONCLUSIONS

This case highlights the prevalence of utilization of aconite within distinct demographic groups, including monks, traditional medicine practitioners, and local hunters. It is imperative to augment public awareness through prominent mass media channels. Furthermore, there is an essential requirement to promote research on the uses of aconite and foster collaborative partnerships with the Faculty of Traditional Medicine at Khesar Gyalpo University of Medical Sciences of Bhutan, an institution dedicated to the training of traditional medicine practitioners in the nation.

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AUTHORS CONTRIBUTION

Following authors have made substantial contributions to the manuscript as under:

KS: Concept, design, data collection and analysis, manuscript writing and review.

TD: Data analysis, manuscript editing and review

VR: Data analysis, manuscript editing and review

KC: manuscript editing and review

UC: manuscript editing and review

CG: manuscript editing and review

NBR: manuscript editing and review

Author agree to be accountable for all respects of the work in ensuring that questions related to the accuracy and integrity of any part of the work are appropriately investigated and resolved.

CONFLICT OF INTEREST

None

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