Case Report

Acute Poisoning of *Aconitum*: A Case Report and Resuscitative Emergency Management with Amiodarone

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Abstract

Aconitine, a plant alkaloid is a usual constituent of various over-the-counter herbal remedies and is an uncommon cause of deliberate poisoning. Herein, we describe an unusual case of poisoning in a young male due to accidental ingestion of aconitine tincture. On presentation, his electrocardiogram documented ventricular arrhythmias for which amiodarone was administered promptly. This led to considerable improvement in heart rhythm. The patient recovered gradually with this management and achieved normal sinus rhythm. He was completely asymptomatic within 5 days of treatment and subsequently discharged. Our management with amiodarone narrates the importance of this drug in aconitine poisoning, which can prove to be fatal in the absence of any intervention or delay in diagnosis. Nonexistence of specific antidote also establishes amiodarone as a standard remedy.

Keywords: *Aconitine, amiodarone, idioventricular rhythm*

Introduction

Aconitum (family *Ranunculaceae*) genus of plants contains substantial amounts of the highly toxic aconitine and related alkaloids, which possess properties of potential cardiac, nervous, and gastrointestinal toxicity in humans.[1] It is a common ingredient of various Ayurvedic formulations and traditional Chinese analgesic medications. Certain parts of these flowering plants are extremely toxic, especially the roots and root tubers.

Human toxicity can result from accidental ingestion or application of an incorrect herbal formulation of *Aconitum* derivatives. Cardiotoxicity including fatal arrhythmias can prove lethal. Involvement of the gastrointestinal system initiates nausea and vomiting, which is exceedingly nonspecific and can conceal the actual diagnosis. The management of accidental ingestion is predominantly supportive since no specific antidote exists.[2]

Case Report

A 20-year-old male presented to the emergency department with accidental ingestion of 10–15 ml *Aconitum* tincture, lying in his father's homeopathy clinic where he worked as an apprentice. Within an hour of ingestion, he developed upper abdominal pain and discomfort accompanied by vomiting and retching. After a brief time, the patient started having palpitations and uneasiness. He had no significant history or comorbidity and no concomitant ingestion of any drug or alcohol. On presentation, the patient was conscious, oriented, well alert but restless. The pulse was irregular and thready, blood pressure was recorded to be 106/60 mm Hg, and 97% oxygen saturation by pulse oximetry with a normal respiratory rate and normal body temperature. The capillary glucose was 93 mg/dL. General physical and abdominal examination was unremarkable. Cardiovascular examination revealed irregular cardiac rhythm. Initial electrocardiography (ECG) of the patient suggested accelerated idioventricular rhythm with broad QRS complexes and atrioventricular dissociation [Figure 1]. Based on direct poison ingestion history and ECG evidence, initial management was

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started. The patient was given injectable amiodarone 150 mg over 10 min, followed by 1 mg/kg continuous infusion for 6 h and 0.5 mg/kg for the next 18 h. Sinus rhythm was achieved within 2 h of starting the infusion.

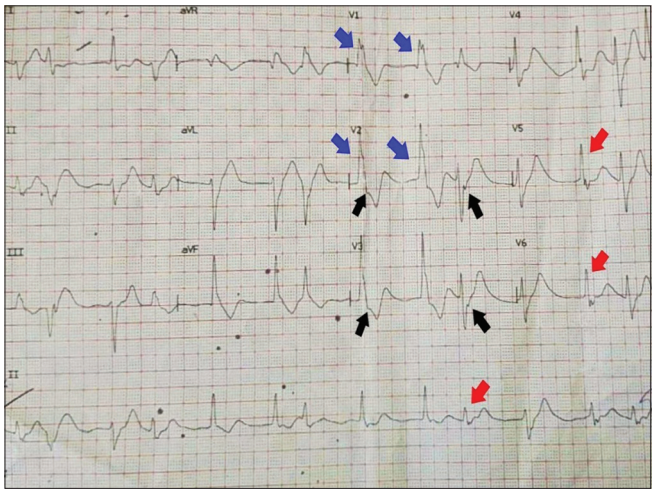

Figure 1: Initial electrocardiogram showing idio-ventricular rhythm with atrio-ventricular dissociation. "QRS" complexes are wide and notched in V1 and V2 (blue arrows) and "P" waves are inverted (black arrow). Fusion beats are also seen (red arrow)

After 24 h, oral amiodarone 200 mg twice a day was started and the patient was closely monitored for arrhythmias in the emergency department.

Meantime, routine investigations, including complete blood count, urine analysis, renal and liver function tests, thyroid profile, serum calcium, and magnesium levels were carried out and they were within the normal limits [Table 1]. Cardiac biomarkers, including qualitative Troponin T was negative and creatine phosphokinase-MB was 12 IU/L (<25 IU/L). His chest X-ray was normal and there was no cardiomegaly. Arterial blood gas parameters and serum electrolytes were within the normal range. A 2-D echocardiogram did not reveal any abnormality. Since aconitine is a potent neurotoxin too, nerve conduction study was carried out; however, it did not show any abnormality. Repeat ECG [Figure 2] on the next day was suggestive of normal sinus rhythm and the patient was then shifted from the emergency department to the general ward for further monitoring. The laboratory investigations were repeated on the 3rd and 5th days. His symptoms markedly improved within 2 days. After 5 days of in-patient

Table 1: Laboratory investigations

Investigation	Value (day 1)	Day 3	Day 5	Normal range
Hemoglobin (g/dL)	14.5	14.2	14.3	13-16
Platelets ($\times 10^9$/L)	2.45	2.68	2.70	150-400
Total leukocyte count ($\times 10^9$/L)	7.1	6.3	6.4	4-12
Bilirubin (mg/dL)	0.7	0.6	0.7	0.2-1
ALP (U/L)	46	50	47	30-150
GGT (U/L)	33	28	30	<50
AST (U/L)	29	33	36	10-40
ALT (U/L)	32	37	35	10-40
Albumin (g/dL)	4.8	4.9	4.9	3.5-5.5
Globulin (g/dL)	3	2.9	2.8	2-3.5
Sodium (mmol/L)	138	140	139	135-145
Potassium (mmol/L)	4.2	4.0	4.1	3.5-5.5
Urea (mg/dL)	25	28	26	15-40
Creatinine (mg/dL)	0.8	0.9	0.7	<1.3
CRP (mg/L)	2	3	2	<5
Lactate (mmol/L)	0.7	0.6	0.6	0.5-1
CPKMB (IU/L)	12	14	11	25
T3 (ng/ml)	1.03	-	-	0.60-1.81
T4 (µg/ml)	7.92	-	-	3.2-12.6
TSH (µIU/mL)	3.74	-	-	0.35-5.50
Serum calcium (mg/dL)	9.7	9.5	9.6	8-10
Serum magnesium (mg/dL)	1.9	1.7	1.9	1.2-2.5
pH	7.39	7.42	7.41	7.35-7.45
HCO_3 (mmol/L)	23.5	24.6	24.0	23-29
PaO_2 (mm hg)	88	87	89	80-100
$PaCO_2$ (mm hg)	38.5	40.5	39.2	36-44

ALP: Alkaline phosphatase, GGT: Gamma glutamyl transferase, AST: Aspartate transaminase, ALT: Alanine transaminase, CRP: C-reactive protein, CPKMB: Creatine phosphokinase MB, T3: Triiodothyronine, T4: Tetraiodothyronine, TSH: Thyroid stimulating hormone, HCO_3: Serum bicarbonate, PaO_2: Partial pressure of oxygen in arterial blood, $PaCO_2$: Partial pressure of carbon dioxide in arterial blood

management, the patient was discharged on 200 mg of oral amiodarone, which was tapered over 2 weeks on subsequent visits.

A written patient consent is present, obtained from the patient at the time of the discharge. The patient was explained that his identity will not be revealed and the case information will be used for education purpose only.

Discussion

Aconitine and other *Aconitum* alkaloids are highly poisonous cardiotoxic and neurotoxic substances. It is commonly found in all parts of the plants of the *Aconitum* species (*aconite*) but most concentrated in roots and root tubers.[3] Three most toxic alkaloids in these plants are aconitine, mesaconitine, and 3-acetylaconitine. The presence of a benzoyl ester side chain at carbon 14 in these alkaloids makes them arrhythmogenic.[4] Aconitine alkaloid binds with a transmembrane region, which is known as neurotoxin receptor site 2, leading to persistent activation and opening of Na + channels.[5] This persistent open state leads to over-excitation and generation of automaticity in cardiac tissue. It is believed that processing during decoction preparation (Soaking and boiling) hydrolyzes the *aconite* alkaloids rendering them less toxic.

Clinical symptoms depend on variable doses of different preparations. Severe poisoning can be caused by 1 g of the cured plant, 0.2 mg of aconitine, and 5 ml of the tincture.[6] Cardiovascular, nervous, and gastrointestinal organ systems are the predominantly affected ones, the latter being the earliest affected system [Table 2].[7,8] Detection of aconitine in serum can be useful in situations where antecedent history is obscured. Various chromatographic techniques, particularly liquid chromatography, in conjunction with mass spectrometry can be utilized; however, they are usually unavailable in most laboratories, are time-consuming often delaying the diagnoses.

Management of poisoning primarily includes supportive therapy as no specific antidote is available. Decontamination is the first step in the line of management. Literature has described that damaged epidermis can absorb the alkaloids.[9] Gastric lavage with activated charcoal can be beneficial, but less useful in late presentation. Hemodialysis or extracorporeal removal techniques are less valuable in cardiotoxic plant poisoning.[10] Nausea, vomiting can be easily managed with antiemetic agents. Minor neurological symptoms improve as toxin concentration decrease with time, but seizures may require the incorporation of benzodiazepines. Special sight should be given to

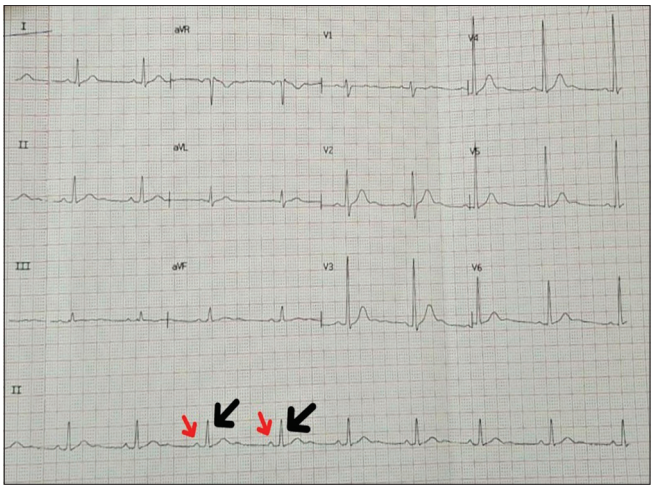

Figure 2: Subsequent electrocardiogram with normal rate and rhythm (Normal "P" wave-red arrow, "QRS" complex-black arrow)

Table 2: Signs and symptoms of various affected systems by aconitine

System	Signs/symptoms
Gastro-intestinal	Nausea, hyper-salivation, vomiting
Nervous	Numbness, ataxia, perioral paresthesias, dysarthria, limb weakness, decreased consciousness, seizures and coma
Cardiovascular	Brady-arrhythmias, hypotension, ventricular ectopic and tachycardia or fibrillation, prolonged QT interval, bundle branch blocks, myocardial depression
Metabolic	Metabolic acidosis, hyperglycemia, hypokalemia

electrolyte imbalance to avoid metabolic complications. Prompt attention to hypotension, ventricular arrhythmias, and seizures can be life-saving. Ventricular arrhythmias or cardiogenic shock refractory to first-line treatment may require an early use of cardiopulmonary bypass.

Ventricular dysrhythmias such as ventricular ectopy, ventricular tachycardia, torsades de pointes, and ventricular fibrillation are mostly observed within the first 24-h. Different rhythm abnormalities require specific management such as atropine can be given in cases with symptomatic bradycardia. For more common complications such as ventricular arrhythmias, cardioversion is less effective, and different agents have been used in the literature. Coulson *et al*. described 65 patients of *aconite* poisoning, showing the higher effectivity of amiodarone and flecainide than lidocaine, or cardioversion in achieving sinus rhythm.[10] Mexiletine, procainamide, and magnesium sulfate may also prove useful occasionally.[10] Likewise, our patient improved with amiodarone and subsequently achieved normal sinus rhythm, which reemphasizes the effectiveness of the amiodarone in arrhythmias precipitated by aconitine poisoning.

Conclusion

Aconitine alkaloid has widespread usage in the herbal medications prepared locally in Asia. There is a low margin of safety between therapeutic and toxic doses of aconitine. Though aconitine poisoning is relatively rare, it is notorious in causing fatal cardiac arrhythmias. Other organ system involvement is also common but is not usually life-threatening. Cardiac involvement requires prompt identification of rhythm disorder and urgent intervention to decrease mortality. This case highlights the importance of such early recognition and appropriate anti-arrhythmic agent use, such as amiodarone, to achieve normal sinus rhythm. Appropriate regulations regarding the sale and over-the-counter availability of aconitine should be in place to avoid loss of lives.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that their name and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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