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A Rare Case of Aconite Poisoning in a Tertiary Care Center in Nepal: Clinical Presentation and Management

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Abstract:-

- ***Introduction:***

Aconite poisoning, caused by the toxin aconitine in the Aconitum genus (monkshood or wolfsbane), can be severe and requires supportive care as there is no specific antidote. Aconitine inhibits sodium channel inactivation, leading to dangerous cardiovascular and neurological symptoms.

- ***Case Presentation:***

A 46-year-old male ingested half of a suspected aconite seed, leading to abdominal cramping, persistent vomiting, tingling sensations, and weakness. Upon admission, he exhibited hypotension, tachycardia, and bilateral mydriasis. ECG showed ventricular premature contractions. Treatment included intravenous amiodarone, magnesium sulfate, calcium gluconate, and gastric lavage with activated charcoal. He was admitted to the ICU for intensive monitoring and treatment, including ongoing amiodarone administration. After improvement, he was transferred to the general ward and later discharged.

- ***Discussion:***

Aconite poisoning involves symptoms from aconitine's effect on sodium channels, including arrhythmias, hypotension, and neurological issues. Management focuses on supportive care, antiemetics, and monitoring. Advanced treatments like flecainide or amiodarone may restore normal heart rhythm, and severe cases might require a cardiac bypass or VA-ECMO.

- ***Conclusion:***

This case describes a rare aconite poisoning with severe neurological, cardiovascular, and gastrointestinal symptoms after ingestion of a suspected aconite seed. The patient was treated with decontamination, intravenous amiodarone, and ICU care, leading to recovery. Despite its traditional use, aconite poisoning often results from its application without proper regulation, highlighting the need for rapid diagnosis and management.

Keywords:- *Aconite Poisoning, aconitine, Ventricular Arrhythmias, Supportive Care, Antiarrhythmics, ICU Management.*

I. INTRODUCTION

Aconite poisoning is relatively rare, but it can be quite serious due to the potent effects of aconitine, which is found in plants of the Aconitum genus (often known as monkshood or wolfsbane). All varieties and all parts of the aconite plant are toxic, with the degree of toxicity varying according to the plant's developmental stage: it is least harmful when young, becomes more poisonous as the seeds mature, and reaches its highest toxicity during full bloom. Aconitine alkaloids are substances that inhibit the inactivation of sodium channels. (1) Previous research indicates that approximately 90% of individuals with aconite poisoning experience polymorphic ventricular arrhythmias, such as premature beats, rapid heartbeat, and ventricular flutter. (2) These arrhythmias are the primary cause of death in such cases. In addition to ventricular arrhythmias, patients with aconite poisoning often exhibit neurological symptoms, including sensory issues (paraesthesia and numbness in the face, perioral area, and limbs), motor problems (muscle weakness), and gastrointestinal symptoms such as nausea, vomiting, abdominal pain, and diarrhea(3). The management of accidental aconite ingestion is primarily supportive, as there is no specific antidote available(4). This article presents a case of aconite poisoning caused by ingesting a seed provided by an unidentified individual who claimed it had medical significance. The seed was later identified as aconite.

II. CASE PRESENTATION

A 46-year-old male, a non-smoker and alcohol user with no known comorbidities, presented to the emergency department following the ingestion of half of a seed suspected to be Aconitum (refer to Figure 1) approximately three hours before presentation. The seed, given by a local

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vendor, was supposed to alleviate abdominal discomfort and bloating. Approximately 30 minutes post-ingestion, the patient experienced severe abdominal cramping, accompanied by multiple episodes of non-projectile vomiting that contained recently ingested food particles but no blood or bile. The patient also reported perioral and generalized tingling in all four limbs. His condition progressively deteriorated, marked by generalized body weakness emerging approximately 90 minutes post-ingestion, and he complained of palpitations. There was no history of chest pain, loss of consciousness, abnormal body movements, or bowel and bladder incontinence. The patient denied any history of allergies, regular medications, substance abuse, or psychiatric illness. Family history was non-significant.

Upon admission to the emergency department, the patient appeared anxious and ill with persistent nausea. He was well-oriented, with a Glasgow Coma Scale score of 15/15. Vital signs were notable for a blood pressure of 100/50 mmHg, a heart rate of 120 beats per minute, a respiratory rate of 24 breaths per minute, and an oxygen saturation of 94% on room air. Physical examination revealed bilateral mydriasis with sluggish pupillary reflexes, diffuse abdominal tenderness without rebound tenderness or guarding, regular tachycardia without murmurs on cardiac auscultation, and an otherwise unremarkable pulmonary examination.

Electrocardiogram (ECG) demonstrated persistent ventricular premature contractions with patterns of bigeminy, trigeminy, couplets, triplets, and quadruplets (refer to Figure 2). Initial treatment included intravenous administration of amiodarone 150 mg over 10 minutes, magnesium sulfate 2 grams over 30 minutes, and 10 mL of 10% calcium gluconate over 10 minutes. Gastric lavage with activated charcoal was performed following nasogastric tube placement and was maintained on free drainage due to persistent vomiting. Laboratory findings showed a white blood cell count of 9,800 cells/mm³, hemoglobin of 16.2 g/dL, platelet count of 280,000 cells/mm³, serum sodium of 130 mEq/L, serum potassium of 3.7 mEq/L (Table 1). Arterial blood gas analysis revealed a pH of 7.36, pCO_2 of 37.4 mmHg, bicarbonate level of 21.2 mEq/L (anion gap of 13.5), and a serum lactate level of 3.8 mmol/L (Table 3). Liver and renal function tests were unremarkable (Table 1). The peripheral blood smear revealed normal cell count and morphology. Serum amylase and lipase levels were within normal limits (Table 2). Urinalysis and electrolyte values were also normal. Additionally, thyroid-stimulating hormone (TSH) and anti-thyroid peroxidase (Anti-TPO) antibody levels were within normal ranges (Table 2).

Table 1 Showing Baseline Lab Findings

TEST	RESULTS	NORMAL RANGE
COMPLETE BLOOD COUNT:		
TLC	9800/cumm	4000-11000
DLC- NEUTROPHILS	73%	40-75
LYMPHOCYTES	25%	20-40
EOSINOPHILS	2%	1-6
HAEMOGLOBIN	16.2gm/dl	12-16
PLATELETS	280000mil/cumm	150000-450000
PCV	43.2%	33-55
RBC	4.83 mil/cumm	4.5-5.5
MCV	82.6fl	76-96
MCH	28.6pg	26-36
MCHC	36gm/dl	31-37
RBS	85mg/dl	70-140
RENAL FUNCTION TEST:		
SODIUM	135mmol/L	135-145
POTASSIUM	3.7mmol/L	3.5-5.5
UREA	30mg/dl	15-45
CREATININE	1.0mg/dl	0.4-1.4
LIVER FUNCTION TEST:		
TB	0.8mg/dl	0.6-1.3
DB	0.3mg/dl	0-0.25
AST	34IU/L	5-35
ALT	35IU/L	5-40
ALP	117IU/L	42-306
TOTAL PROTEIN	8.1g/dl	6.5-8.2
SERUM ALBUMIN	4.6g/dl	3.5-5.0

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Table 2 Showing Thyroid Function Test, Serum Amylase, Lipase and Serum Electrolytes

TEST	RESULTS	NORMAL VALUES
THYROID FUNCTION TEST:		
FT3	3.05pg/ml	1.84-4.04
FT4	1.46ng/dl	0.94-1.64
TSH	7.31 µIU/ml	0.3-5.1
Anti TPO Antibody	<5 IU/ml	<35
LIPASE	118U/L	23-300
AMYLASE	86.0U/L	30-110
SERUM MAGNESIUM	1.7mg/dl	1.6-2.3
SERUM CALCIUM (TOTAL)	9.7mg/dl	8.4-10.2

Table 3 Showing Arterial Blood Gas Analysis

TEST	RESULTS	NORMAL VALUES
pH	7.36	7.35-7.45
pCO2	37.4 mmHg	35-45
pO2	52 mmHg	80-100
cHCO3	21.2 mmol/L	22-26
LACTATE	3.8mmol/L	0.5-2.2
ANION GAP	13.5 mmol/L	12±2

The patient was admitted to the intensive care unit (ICU) for close monitoring and aggressive treatment. In the ICU, he received intravenous amiodarone as a 150 mg bolus over 10 minutes, followed by an infusion at 1 mg/min for 6 hours (total dose 360 mg), which was subsequently reduced to 0.5 mg/min for the next 18 hours (total dose 540 mg). Intravenous ondansetron was administered to manage persistent vomiting. A repeat ECG the following day revealed a normal sinus rhythm with bradycardia (refer to Figure 3). Strict fluid balance management was implemented, with regular monitoring of input and output. Neurological status was assessed frequently to monitor for symptom progression. After 3 days in the ICU, the patient was transferred to the general ward for continued monitoring and symptomatic treatment. He was discharged after clinical improvement and continues to attend regular follow-up appointments.

III. DISCUSSION

There are around 300 species of Aconitum plants worldwide, with the majority in East and South-eastern Asia and Central Europe, and to a lesser extent in Western North America and the Eastern United States. (5)It is also known as Monkshood, Blue Rocket, Wolf Bane, and Sweet Poison. It has been used for a long as a medicinal herb in traditional Chinese and Ayurvedic medicine because of its analgesic and sedative properties. (6)Aconite is also employed in Nepal as an Ayurvedic herbal practice for its pain-relieving and calming effects.

Although low-dose aconitine has shown good therapeutic potential in heart failure, myocardial infarction, neuro-inflammatory diseases, rheumatic diseases, and tumors, it has increased its global research in recent years. (7) However, its toxic effects are well-known and described. All parts of the plant are poisonous; entry routes are via skin and oral intake. Moreover, roots are highly toxic and are used more commonly than other parts. Its toxic principles are deterpene alkaloids known as aconitine, misaconitine, and hypaconitine. The expected fatal amounts are 2 mg of aconite, 5 mL of aconite tincture, and 1 gram of raw aconite plant. (8)

Various ways of alleged therapeutic or accidental exposure are documented. In traditional medicine, it is prepared by prolonged boiling for more than 2 hours to decrease toxic ingredients, and short-term boiling or excess intake is proposed for toxicity. (9) Accidental intake due to inability to differentiate it from edible wild herbs (Molopospermum Peloponnesian vs Aconitum napellus). (10)Online buying and use from herbal plant centers and increasing interest in herbal medicine also pose a threat in developed countries. (11)Its use as spice in restaurants, resulting in mass poisoning, is also documented. (12)

The clinical presentation of aconite poisoning typically involves a complex combination of neurological, cardiovascular, and gastrointestinal symptoms. Neurological manifestations often include paresthesia and numbness, particularly affecting the face and perioral region, and generalized muscle weakness. Cardiovascular involvement is prominent, presenting with symptoms such as hypotension, chest pain, palpitations, and a range of arrhythmias, including bradycardia, sinus tachycardia, ventricular ectopics, ventricular tachycardia, and, in severe cases, ventricular fibrillation. Gastrointestinal disturbances are also common, with affected individuals experiencing nausea, vomiting, abdominal pain, and diarrhea. (3) a notable sign of aconite poisoning is the hippus reaction, characterized by alternating contraction and dilation of the pupils. This spectrum of symptoms reflects aconite's potent and widespread effects on multiple organ systems.

Cardiotoxicity and Neurotoxicity are due to the persistent opening of voltage-sensitive sodium channels in excitable tissues, including the myocardium, nerves, and muscles. The effects of aconite are dose-dependent. At

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lower concentrations, it enhances pre-synaptic acetylcholine (ACh) release, which improves muscle contraction. However, aconite causes prolonged opening of neuronal voltage-gated sodium channels at higher concentrations, decreasing synaptic ACh release. (13)Cardiac arrhythmia is due to (a) delayed after-depolarization and early after-depolarization by prolonging sodium influx and (b) anticholinergic effects mediated by the vagus nerve. It also has hypotensive and bradycardiac actions due to activation of the ventromedial nucleus of the hypothalamus. Gastrointestinal features are due to strong ileum contractions through acetylcholine release from the postganglionic cholinergic nerves. (3)

The management of aconite poisoning is largely supportive, as no specific antidote is available. The first step is decontamination to reduce toxin absorption. Nausea and vomiting can be effectively controlled with antiemetic medications. While mild neurological symptoms typically resolve as the toxin levels decrease over time, seizures may necessitate the use of benzodiazepines. It is crucial to monitor and correct electrolyte imbalances to prevent metabolic complications. Immediate intervention for hypotension, ventricular arrhythmias, and seizures is critical and can be life-saving. (13)Advanced cardiopulmonary resuscitation, securing the airway with tracheal intubation, magnesium sulfate, defibrillation, and antiarrhythmics should be provided as needed. (10)Based on the evidence from systemic review, flecainide or amiodarone appear to be more associated with a return to sinus rhythm than lidocaine and cardioversion. (14) In refractory cases, cardiac bypass or VA-ECMO (Veno-arterial extracorporeal membrane oxygenation should also begin immediately. (15)

IV. CONCLUSION

This case highlights a rare instance of aconite poisoning, characterized by severe neurological, cardiovascular, and gastrointestinal symptoms following the ingestion of a suspected aconite seed. Despite the long-standing use of aconite as an effective herb in traditional medicine, poisoning often arises from confusion with other plants and its application without proper regulation. The patient's clinical presentation included persistent vomiting, paresthesia, hypotension, and significant arrhythmias, requiring immediate and intensive management. Effective treatment involved aggressive decontamination, intravenous amiodarone for arrhythmias, and supportive care in the ICU. The patient's condition improved with prompt intervention, highlighting the critical need for rapid diagnosis and management in rare and severe cases of aconite poisoning.

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