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Case Report

CLEISTANTHUS COLLINUS POISONING - A CASE REPORT

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ABSTRACT

Cleistanthus collinus is a toxic shrub most commonly encountered plant poison in southern India. Ingestion of the leaves or its decoction causes hypokalemia, renal failure, shock, respiratory distress and cardiac arrhythmias leading to mortality in most cases.Cleistanthin A and Cleistanthin B are reported to be toxic substances responsible for poisoning. We report a case of healthy female who ingested the leaves of this plant in suicidal intension. Vomiting, hypokalaemia, decreased urine output, tachycardia are the findings developed in this patient. She received supportive care and recovered within 5 days.

INTRODUCTION

Cleistanthus collinus, a plant belonging to the family "Phyllanthaceae", is a toxic shrub, meant for self-abuse exclusive to the southern Indian states of Tamil Nadu and Pondicherry, because of its easy availability and free access to the plant. It is popularly known in tamil as oduvanthalai.[1] All parts of the plant are potentially toxic even though leaves are commonly used for poisoning. The method of consumption of the plant for intentional self-harm includes swallowing the crushed plant parts, chewing the leaves, consuming a paste/juice of the leaves or a decoction formulated by boiling the leaves in water.[2] This plant is commonly used as suicidal, homicidal and for procuring criminal abortion.[3]

Case Report

A 40 years old female came to the emergency department after ingestion of 1-2 leaves of 'oduvan' at her residency with the complaints of having abdominal pain, dizziness, slurred speech and dry mouth. Samples of the plant were brought by her relatives. Soap water was given by their family members as a first aid therapy. She also had one episode of vomiting. Gastric lavage had been done after hospitalization. She had a past medical history of gastric ulceration and was on medications for the same.

On examination she was conscious, well oriented and afebrile. Her Pulse rate was 110/min, blood pressure 120/70 mmHg, respiratory rate was 26/min. Laboratory investigations revealed that her total leucocyte count was elevated 13900cells/cumm with elevated neutrophils 11120 cells/cumm and decreased lymphocytes as 15%. Serum sodium was 135 mmol/L, Serum potassium 3.1 mmol/L, serum chloride 108 mmol/L, other renal and hepatic parameters were found to be normal.

ABG showed pCO2 was 22.7 mmHg and her pH was 7.325 which indicate respiratory acidosis. Oxygen supplementation 2L per min were given to correct her respiratory acidosis. Hypokalaemia was corrected using syrup potassium chloride 10 ml enterally thrice daily through Ryle's tube following which ECG normalized. After 48 hours of hospitalization, she becomes hypotensive with decreased urine output.

Table 1 Estimated serum potassium

	Day 1 (3 a. m.)	Day 1 (11 a. m.)	Day 1 (7 p. m.)	Day 2 (7.40 a.m.)	Day 3 (5.20 p.m.)	Day 4 (6.20 a.m.)
Serum K^+ (mmol/L)	4.4	4.0	3.5	3.2	3.9	4.3

Blood pressure and decreased urinary output were promptly corrected when adequate fluids were given. Gastric lavage was

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done followed by activated charcoal with the dose of 1gm/kg and continued multi dose activated charcoal 60g every 6 hours for 48 hours were given.

Her serum potassium, blood pressure, urine output, pulse rate were normalized by the fifth day of admission. Patient conditions were improved and hence discharged.

DISCUSSION

Suicide rates in South India are high, as evidenced by an overall suicide rate of 71.4 per 100,000 people in a community-based study.[2]

Mortality rate with C. collinus is about 40% and death usually arisesin 3-7 days (majority of deaths occurring on the third day) of consumption. Higher mortality rates seen in patients who had ingested larger number of leaves (>60) and boiled leaf extract.[4] The toxic constituents in the leaf include aryl naphthalene lignan lactones -Diphyllin and its glycoside derivatives Cleistanthin A and B.

Cleistanthin A arrests cells growth by inhibiting DNA synthesis and celldivision, causing DNA strand breaks and hence DNA damage. It also triggers cell apoptosis. In addition,it inhibits membrane metalloprotein-9, which decreases proliferating cell viability. Cleistanthin B is clastogenic and induces micronuclei formation and chromosomal aberrations. It inhibits cellular proliferation by causing a G1 phase arrest and develops cellular apoptosis.[5]

Clinical manifestations of C. collinus poisoning include vomiting, nausea, abdominal pain, diarrhea, chest pain, dyspnea, tachycardia, hypotension, visual disturbances, muscle cramps, weakness, altered sensorium, altered speech, hypokalaemia, leukocytosis, respiratory failure, renal failure, elevated hepatic transaminases, metabolic acidosis and alkaline urinary pH.Kaliuresis appears to be the main mechanism underlying this abnormality, though vomiting and dehydration may have a role.[6]

SPE Benjamin *et al*. study showed that his patient had abdominal pain, dizziness, vomiting on admission. He developed tachycardia, oliguria and hypokalaemia after 48 hours of hospitalization.[7]

In our present case, patient had tachycardia on admission which later normalized. She also had slurred speech, hypotension, abdominal pain, dizziness, vomiting and dry mouth (dehydration). She had hypokalaemia and respiratory acidosis after 48 hours of admission.

Management of poisoning mainly comprise of stabilizing the airway, breathing and circulation. Mechanical ventilation, gastric lavage and activated charcoal may be required dependson the patient's condition and the method of ingestion.[5]

Our patient was treated with activated charcoal with the dose of 1gm/kg and continued multi dose activated charcoal 60g every 6 hours for 48 hours. Hypotension and decreased urine output was managed by giving 1000ml bolus dose of Ringer lactate followed by maintenance dose of 150ml/hr of ringer lactate. Hypokalaemia was treated with syrup potassium chloride. Vomiting was managed with injection ondansetron

N-acetylcysteine, L-cysteine and melatonin have been recommended as antidotes for the management of C. collinus toxicity. The renal tubule appears to be the primetarget of injury and distal renal tubular acidosis results in hypokalemia and metabolic acidosis. By identifying and correcting these factors and initiating early and aggressive supportive management, there is likely to reduce mortality in patients with *C. collinus* poisoning.[2]

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