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Case Report

A case report: *Cleistanthus collinus* (Oduvan) poisoning

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ABSTRACT

Cleistanthus collinus-an extremely toxic plant has been reported to be used for deliberate self-harm in different parts of the country, notably South India. Toxins contained include Cleistanthin A and B arediphyllin derivatives and are known to cause neuromuscular blockade, type 2 respiratory failure, type 2 renal tubular acidosis, hypokalaemia, cardiac arrhythmias. We are presenting a case report where the poisoning was associated with type 2 respiratory failure, acute kidney injury, type 2 renal tubular acidosis, hypokalaemia, acute respiratory distress syndrome. The patient was managed with intravenous N-acetylcysteine, ventilatory support and multiple sessions of dialysis following which the patient after a prolonged stay of 29 days was discharged from the hospital.

Keywords: *Cleistanthus collinus*, Respiratory failure, Renal tubular acidosis, Hypokalemia, Arrhythmia

INTRODUCTION

Intentional self-harm accounts for approximately 1.5% of global deaths, ranking it as the tenth leading cause of mortality worldwide. Suicide claims an estimated 1 million lives each year, with a global annual rate of roughly 14.5 per 100,000 individuals.[1] The true incidence of poisoning may be higher than reported due to limitations in data collection and underreporting.[2] Southern India shows particularly high suicide rates, with one community-based study reporting a rate of 71.4 per 100,000 people.[3-5] Although plant-based poisoning is relatively rare on a global scale, it is a prevalent form of self-harm in the Indian subcontinent.[2,3,6-9] *Cleistanthus collinus* is a toxic shrub recognized by various regional names-Garari in Hindi, Oduvan in Tamil, Vadisein Telugu, and Nilapala in Malayalam. Historically, it has been used in cases of self-poisoning, homicide, and as an abortifacient (Table 1).[10] *Cleistanthus collinus* belongs to the Euphorbiaceae family, and every part of the plant is considered potentially toxic, with the leaves being the most harmful.[10] The primary toxic compounds are arylnaphthalene lignan lactones, particularly Diphyllin and its glycoside forms—Cleistanthin A, B, C, D, and Cleistanone.[11] Among these, Cleistanthin A and B, along with Diphyllin, are the main agents responsible for toxicity and were historically referred to collectively as "Oduvan".[12] Another compound, Collinusin, is also present in the leaves. The overall toxicity is largely attributed to Cleistanthin A and B. A hallmark clinical feature in affected individuals is distal renal tubular acidosis (RTA) characterized by a normal anion gap metabolic acidosis. Although this is commonly observed, some cases also show evidence of proximal tubular damage, suggesting more widespread tubular dysfunction.[10,13]

Given that the toxic components are glycosides, they are anticipated to exert effects on the cardiovascular system. Indeed, clinical cases have reported arrhythmias and other rhythm disturbances.[14] Patients frequently develop shock, thought to be due to peripheral vasodilation, which is often associated with increased mortality. Although rare, neuromuscular weakness has been reported and may be linked to the plant's toxic effects at the neuromuscular junction.[15] Human case studies suggest that fatalities typically occur between 3 to 7 days following ingestion. Mortality rates can exceed 40%, with poorer outcomes

observed in older adults or those with pre-existing health conditions, abnormal heart rates (either bradycardia or tachycardia), rapid breathing, low blood pressure, or fever.[10]

CASE REPORT

A 55-year-old lady, known hypertensive was referred from a hospital with an alleged history of consumption of 15 antihypertensive/sedative tablets 4 days before presentation. She was initially treated at the local hospital on an outpatient basis with the complains of giddiness. 2 days later she developed retrosternal discomfort, vomiting, generalized weakness and headache. She was admitted to the local hospital where she desaturated and was intubated. At the time of presentation to our hospital, patient was intubated on pressure control, HR-120/min, BP-124/60 mmHg on dopamine infusion. At the time of presentation GCS was E1VTM1 (Eye opening, Verbal response, Motor response) with mute plantar, deep tendon reflexes absent. Respiratory system examination revealed crepitations bilaterally. On admission her laboratory investigations (Table 2) showed high total leucocyte count (TLC) with neutrophilic predominance. Her C-reactive protein (CRP) was elevated. Renal function test revealed Acute kidney injury. Hypokalemia was secondary to type renal tubular acidosis. Liver function test revealed transaminitis. Cardiac injury markers and brain natriuretic peptide (BNP) were also elevated.

Arterial blood gases (Table 3) revealed severe respiratory and metabolic acidosis with normal lactate and normal anion gap. X-ray showed bilateral diffuse homogenous opacity with features suggestive of acute respiratory distress syndrome. Initial ECG showed sinus rhythm, normal axis, ST elevation in V1 aVR, Lead III-pathological Q wave, ST depression in lead I, aVL and V5. Initial ECG showed sinus rhythm, normal axis, ST elevation in V1 aVR, lead III-pathological Q wave, ST depression in lead I, aVL and V5.

Treatment

The initial working diagnosis of multiple drug overdose followed by aspiration pneumonia leading to sepsis was considered and the patient was started on antibiotics-intravenous piperacillin/tazobactam and clindamycin. As the patient was in severe acidosis with nil urine output, renal hemodialysis was initiated. Meanwhile, patient bystanders found some crushed leaves of Cleistanthus, while cleaning the house, thus leading to the suspicion of Oduvan poisoning. There is no known antidote for Oduvan poisoning. Based on the previous studies and case reports the patient was started on IV Bronac (N-Acetyl cysteine) which was continued till the normalization of hepatic functions. There was a gradual improvement in her sensorium. There was also a improvement of blood pressure following which inotropic supports were gradually tapered and then stopped. She was extubated on 10th day of admission. The renal functions continued to be deranged with elevated creatinine and persistent metabolic acidosis, thus regular sessions of hemodialysis were continued. As in the previous studies the patient was managed symptomatically. For the acute kidney injury (AKI) the patient underwent 12 sessions of hemodialysis. For the type 2 respiratory failure she was intubated as she went into ARDS she was continued on ventilatory supports with IV Antibiotics being continued. For the cardiac failure she was continued on ionotropic supports. Based on the previous studies she was given intravenous N- Acetyl cystine150 mg/kg over 1 hour followed by 50 mg/kg over 4 hours than 100 mg/kg over 16 hours, after this was continued 600 mg intravenous every 8 hourly till the hepatic functions normalized.

Table 1: Common name of *C. collinus*.

Bengali	Karlajuri
Bihar	Pasu
Hindi	Garari
Kannada	Badedarige, Bodadaraga, Kadagargari
Malayalam	Nilappala, Odugu
Marathi	Garari
Oriya	Korodo
Sanskrit	Indrayava, Kaudigam, Kutaja, Nandi
Tamil	Nilaippalai, Odaichi, Odan, Odishi, Oduvan
Telugu	Kadise, Korshe, Korsi, Vadise

Table 2: Laboratory investigation.

Labs	Value	Ref range
TLC	19,000	4,000-10,000
Neutrophil	88%	50-70%
CRP	360	<5
Creatinine	5.17	0.6-1.2
Sodium	135.8	135-145
Potassium	2.0	3.6-5.5
T. Bilirubin	0.4	0.1-1.2
SGOT/AST	179	10-40
SGPT/ALT	85	10-35
ALP	152	30-120
Troponin I	0.22	0.0-0.04
CK-MB	30.3	5-25
BNP	113.2	<100

Table 3: Arterial blood gas analysis.

Labs	Value	Ref range
pH	7.07	7.35-7.45
pO_2	137	>90
pCO_2	64.8	35-45
SO_2	98.2	>95
HCO_3	14.7	18-24
Lactate	0.6	0.5-2.2
Anion APG	7.8	8-12

DISCUSSION

Cleistanthus collinus, a highly poisonous shrub. It belongs to the family Euphorbiaceae. All parts of the plant are potentially toxic. Leaves being the most toxic ones. Toxins most implicated are Diphyllin and its glycoside derivatives Cleistanthin A, Cleistanthin B, C, D and Cleistanone.[10,11] The toxic active principles in the leaves are arylnaphthalene lignan lactones-Diphyllin and its glycoside derivatives Cleistanthin A and Cleistanthin B; and Collinusin. Diphyllin, Cleistanthin A and B were collectively known as "Oduvan" in the past.[12] In addition, the lignans Cleistanthin C, Cleistanthin D and Cleistanone, are present.[11] The toxicity of the plant has been attributed primarily to Cleistanthin A and B. Distal renal tubular acidosis with normal anion gap acidosis appears to be a consistent feature in all patients although proximal tubular injury with global tubular dysfunction has also been seen.[10,13] The toxic principles, being glycosides, would be expected to cause cardiac effects. Clinical reports have shown cardiac rhythm abnormalities.[14] Shock often seen which is associated with mortality, hypothesized that it's due to peripheral vasodilation. Neuromuscular weakness has been rarely documented which have shown a consistent effect of *C. collinus* toxins on the neuromuscular junction.[15] Studies in humans have revealed death to take place in 3-7 days of consumption with mortality of 40% and more increased in older age group, underlying chronic disease, tachycardia or bradycardias group, underlying chronic disease, tachycardia or bradycardias, tachypnea, fever, hypotension.[10]

CONCLUSION

Oduvan consumption continues to be a preferred mode for deliberate self-harm. The renal tubule is a key site affected during injury. In cases of distal renal tubular acidosis, patients often develop hypokalemia and metabolic acidosis, both of which must be addressed as part of effective treatment for this type of poisoning. As there are no known antidotes for the same, management continues to be symptomatic with early recognition of the poisoning various signs and symptoms with initiation of the appropriate management.

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