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# A Case report: Oduvan leaf poisoning

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**Abstract**
*Cleistanthus collinus*, a plant used for intentional self-harm have boosted the mortality rate over time. The toxic principles in the plant which do not have a challenger antidote are increasingly consumed by rural people with suicidal ideation. Mortality occurs due to arrhythmias, renal failure, shock and respiratory distress. Here, we report a case on a patient who consumed 6 leaves of Oduvan thalai.

**Keywords:** *Cleistanthus collinus*, Oduvan thalai

## Introduction
Oduvan *(Cleistanthus collinus)*, a toxic deciduous shrub belonging to family Euphorbaceae comprises 140 species native to the region between Africa and the Pacific islands [1]. It is deliberately used for self-harm in rural South India especially in Tamil Nadu and Pondicherry. This shrub named as *Oduvan* (Tamil), *kadise* (Kannada), *Vadisaku* (Telugu) and *Oduku* (Malayalam) account for about 40% mortality in consumed individuals. The method of ingestion of the plant for deliberate self-harm includes swallowing the crushed plant parts, chewing the leaves, consuming a paste/juice of the leaves or a decoction prepared by boiling the leaves in water [2]. The toxic principles in the leaf include *aryl naphthalene lignan lactones — Diphyllin* and its glycoside derivatives *Cleistanthin A* and *B. Lignan lactones* inhibit thiol enzymes, glutathione and AT Pases in tissues. Toxic effects of the plant demonstrates neuromuscular blockade with muscle weakness, distal renal tubular acidosis (dRTA) and type 2 respiratory failure with conflicting evidence of cardiac involvement. Hypokalemia and normal anion gap metabolic acidosis induces rhabdomyolysis resulting in myoglobin uric renal failure and neuromuscular weakness.

## Case report
A 38 year old male, was brought to the casualty with alleged history of consumption of *oduvan* leaf (6 leaves) at around 10.00 pm under the influence of alcohol. He was presented with the complaints of vomiting (multiple episodes) and giddiness and was disoriented. No signs of respiratory distress, loss of consciousness and seizure. He was an alcoholic and free of comorbidities. Patient was tachycardiac (pulse rate, 112 beats/min) during admission. Blood pressure was 130/80 mmHg and respiration rate 20 breaths/min. Patient was received by the hospital 2 hours 45 minutes after the ingestion of leaves. He underwent gastric lavage with 2 litres saline. Blood investigations revealed a gradual reduction in potassium and sodium levels leading to Hypokalemia (3.3 mmol/litre) and Hyponatremia (134 mmol/litre) respectively. Bicarbonate (18.7 mmol/litre) and pH (7.2) indicated hypoventilation accompanied by metabolic acidosis which was treated with sodium bicarbonate. Total leukocyte count was 10,400 cell/cumm.

Activated charcoal, 60g was given every 6 hours through ryles tube. Inj. Ondansetron, 4mg and Inj. Pantoprazole, 40mg were administered twice daily through IV route, which helped to overcome vomiting and gastric disturbances. Inj. Thiamine, 200 mg was given through IV route as stat in 100ml NS. Potassium correction was done giving syrup KCl, 10ml, PO thrice daily. Patient was discharged on 4$^{th}$ day with Syrup KCl and pantoprazole, devoid of symptoms on admission.

## Discussion
The initial presentation of the poisoning in our patient was restricted to GI symptoms (vomiting and abdominal discomfort) which gradually paved way to giddiness, systemic electrolyte disturbances (hypokalemia, hyponatremia) and changes in arterial blood gas report suggestive of metabolic acidosis. Symptomatic management was given to which the patient responded well.

It was Thomas *et al* in 1987, who first reported 32 cases of Cleistanthus collinus poisoning [3]. He stated that hypokalemic metabolic acidosis resultant to the renal tubular injury is integral to the clinical presentation. The same group conducted another study in 1991 to interpret the mechanism of Hypokalaemia and concluded it to be the renal potassium leak [4].

Toxic principles (*aryl naphthalene lignan lactones — Diphyllin* and its glycoside derivatives *Cleistanthin A* and *B*) in *Cleistanthus collinus* leads to renal tubular acidosis which results in hypokalemia and hyponatremia [5]. The common clinical manifestations shown by 56 patients with oduvan poisoning in the study conducted by Alladi *et al.* reflected hypokalemia, hyponatremia, leucocytosis, acute kidney injury and metabolic acidosis [6]. According to his study, the

mortality rate was high in patients who presented with all these symptoms.

APE Benjamin, in his case report introduces a 24 years old male who ingested around 40 – 50 leaves and faced hypokalemic metabolic acidosis, hypotension, acute respiratory distress syndrome, distributive shock, distal renal tubular acidosis and arrhythmia [7]. In our patient, the severity of the symptoms was less in comparison (consumed only 6 leaves).

The studies of V Shankar *et al*. [2] Shreya Sharma *et al.* [5] fortifies that the toxic effects of plant aggravated when the leaves were ingested as decoction. Mortality rate was high in these patients due to severe respiratory depression, cardiac arrhythmias and renal failure. Our patient consumed fresh leaves and the risk was comparatively low.

## Conclusion

Oduvan leaf poisoning which still remains without a proper antidote is the reason why it attracts the concern and curiosity of the medical care providers. Although the symptomatic management saves a few, severely toxified patients are challenging and add on to the mortality rate.

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