Case Report

Cleistanthus Collinus Poisoning

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Abstract

Cleistanthus collinus is an extremely toxic plant poison. Cleistanthin A and B, the toxins of Cleistanthus collinus, are diphyllin glycosides which produce cardiac arrhythmias, urinary potassium wasting, hypoxia, metabolic acidosis and hypotension. We report ARDS, distal renal tubular acidosis and distributive shock secondary to inappropriate vasodilatation in a case following ingestion of its leaves.

INTRODUCTION

Cleistanthus collinus poisoning usually occurs following intentional ingestion of the leaves with mortality as high as 30%, usually occurs 3-7 days after ingestion. Thomas K, et al,1 identified hypokalemia due to renal potassium wasting and cardiac dysrrhythmias. Subrahmanyam DKS, et al,2 identified other clinical features of severe poisoning (Table 2). S Easwarappa, et al,3 have described neuromuscular weakness.

CASE REPORT

A 24 years male was referred from a rural hospital 2 days after ingestion of 40-50 leaves of ‘oduvan’ with history of abdominal pain, vomiting and breathlessness. Gastric lavage had been done. Samples of the plant were brought with him. It was identified as Cleistanthus collinus at the Presidency College and Forensic Sciences Department, Chennai.

On examination, he was conscious, oriented, afebrile and dyspnoeic. Blood pressure was 110/70 mmHg, pulse rate 128/min and respiratory rate 33/min. He had epigastric tenderness. Fine crepitations were heard bilaterally. Examination of the cardiovascular and nervous system was unremarkable.

Total WBC count was 9800 cells/cumm with predominant neutrophils. Serum potassium was 2.5 mEq/L, sodium 144 mEq/L, chloride 112 mEq/L, bicarbonate 17 mEq/L, total calcium 6.7 mg/dL, urea 37 mg/dL, creatinine 1.4 mg/dL, total bilirubin 0.8 mg/dL, direct bilirubin 0.4 mg/dL, SGOT 112 U/L, SGPT 90 U/L, SAP 1341 U/L, creatinine kinase (CK) total 883 U/L and CK-MB 123 U/L. Ultrasonogram of abdomen and echocardiogram were normal. ECG showed prominent U waves. ABG (breathing room air): pH 7.290, pCO2 34.0 mmHg, pO2 89.8 mmHg and HCO3 17.9 mmol/L.

Later, as he became hypoxic despite oxygen supplementation, ventilator support was given. Peak airway pressure was 48 cmsH2O. PaO2/FiO2 ratio: 142. Central venous pressure (CVP) was low. A chest X-ray showed bilateral peripheral alveolar infiltrates (Fig. 1). ARDS was diagnosed and managed with PEEP and prone ventilation.

There was increased urinary potassium loss (Table 1). This in combination with hyperchloremic metabolic acidosis and normal serum anion gap suggested renal tubular acidosis (RTA). Positive urinary anion gap

Fig. 1: Chest X-ray of the patient showing ARDS. A right internal jugular catheter and endotracheal tube are seen in position.
despite low serum bicarbonate confirmed RTA. Acidosis was corrected using IV sodium bicarbonate followed by Shohl’s solution and potassium citrate by Ryle’s tube. Normal bicarbonate excretion following this and urinary pH of 6.5 established distal (Type 1) renal tubular acidosis (dRTA). Hypokalemia was corrected using IV KCl and potassium citrate enterally (Table 1) following which ECG normalised.

About 48 hrs after hospitalisation, he became hypotensive with tachycardia, low CVP, oliguria and increased urinary osmolality suggestive of toxin induced vasodilatation. Blood pressure and urinary output were corrected promptly when adequate fluids were given.

We used N-acetyl cysteine 150 mg/Kg over 1 hr followed by 50 mg/Kg over 4 hrs and 100mg/Kg over the next 16 hrs as glutathione depletion and benefit from –SH containing compounds has been reported in animal studies. Cardiac and liver enzymes normalised in a week. Distal RTA improved over 2 weeks. He was off the ventilator on the 10th day and lung shadows disappeared after 3 weeks.

**DISCUSSION**

Hypokalemic metabolic acidosis, hypotension and hypoxia have been described in this poisoning. We evaluated further and identified dRTA, distributive shock and ARDS respectively in this patient (Table 2). Specific interventions against these entities were initiated.

Hypokalemia was corrected by estimating urinary losses and compensating for it. ARDS, dRTA and distributive shock were also meticulously corrected. Arrhythmias were either offset by these measures or probably occur at higher blood levels of the toxin with a permissive role for hypokalemia similar to digitoxicity. Though we cannot categorically attribute this patient’s survival to any of these measures or to N-acetyl cysteine, these deserve evaluation in further trials.

Though the pathogenesis of *Cleistanthus collinus* poisoning is well established, progress in our
understanding of the pathophysiology could have therapeutic and prognostic implications.

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REFERENCES


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Announcement

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