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Case Report

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Cuscuta reflexa Roxb. Poisoning: A Case Report

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ABSTRACT

Cuscuta reflexa (*C. reflexa* Roxb.), is an endogenous parasitic plant found in Nepal and has been used by various ethnic communities of Nepal. However, there has been cases of toxicity in humans which needs attention. Here we discuss a case of *C. reflexa* Roxb. accidental self-poisoning while being used for liver ailments that presented with gastrointestinal symptoms initially and local health facility and visited two health facilities before presenting to Tribhuvan University Teaching Hospital with multiple complaints. Appropriate risk assessment was done and the patient was managed conservatively at our center for three days following which he was discharged.

Keywords
Cuscuta reflexa, ethno-botany, herbal toxicity, human poisoning

INTRODUCTION

Nepal is rich in flora that have known ethno-botanical importance. Though there has been survey discussing the therapeutic uses of these plants, toxic potential has not been discussed much. Several rural communities in Nepal greatly depend on medicinal plants for their primary health care needs.[1-3] The ethno-medicinal practices of these communities has been documented time and often.[2-4] However, studies about their toxicity to humans is inadequate, if any exists. Here, we share a case of poisoning due to *Cuscuta reflexa* Roxb that presented to our health facility.

CASE PRESENTATION

A 58 year male from Makwanpur district of Nepal presented to Tribhuvan University Teaching Hospital (TUTH), Emergency Department (ED) in December 2015 with alleged history of deliberate self-ingestion of weed extract of "Akash beli" plant, as a measure for treatment of deranged liver function detected during regular health check-ups, following which he developed vomiting, multiple episodes, watery, non-billous, not blood stained. He was taken to local health facility and was managed conservatively following which he developed slurring of speech and dizziness for which he was referred to higher center with

differential diagnosis of hyponatremia, cerebro-vascular accident and was taken to another hospital, where he was managed conservatively and referred to other center followed by presentation at TUTH General Practice OPD with complains of unable to pass stool/flatus, restlessness, difficulty in breathing and was referred to TUTH ED.

At arrival to TUTH ED post nine hours of ingestion, he had spontaneous, regular breathing with 23 breaths/minute and oxygen saturation of 94% in room air; pulse rate was 86/min and blood pressure 80/50 mmHg; was conscious (Glasgow Coma Scale was 15/15), temperature was 98^0F and blood glucose was found to be 158 mg/dL with glucometer test; there was no active bleeding. Patient was received in red area of ED, O_2 via mask at 2L/min was administered, monitor was attached, peripheral venous access was established and was catheterized. Initially he received 500 mL of normal saline intravenously over 30 minutes, injections of Ondansetron 4 mg, Pantoprazole 40 mg, Hyoscine butyl bromide 20 mg, Tramadol 50 mg and Vitamin K 10 mg.

Risk assessment was done in terms of agent (whole plant extract), amount (approximately 10 mL, diluted in 350 mL of water), time since ingestion (9 hours back), current clinical status (stable, no active bleeding), expected clinical status (may develop bleeding disorders) and co-morbid conditions (no history of hypotension, diabetes, bleeding disorders).

Arterial blood gas analysis showed features of metabolic acidosis and electrolytes levels were Na^+ 122 mEq/L, K^+ 3.33 mEq/L, Cl^- 105 mEq/L, Ca^{++} 0.54 mmol/L, Mg^{++} 0.26 mmol/L. Twelve-lead ECG was done and showed sinus bradycardia (60/min). Complete blood count showed total leucocyte count (TLC) of 9900 with neutrophilic predominance (84%), haemoglobin 14.2 gm%, PCV 43.0%, RBC count of 4.51 million/mm3, platelets of 2,93,000/mm3. Blood biochemistry tests showed random blood sugar 115.2 mg/dL, blood urea 6.0 mmol/L, serum creatinine 95 Umol/L, Na^+ 139 mEq/L, K^+ 4.2 mEq/L, serum amylase 87 mmol/L, Ca^{++} 2.1 mmol/L, Phosphorous 4.2 mmol/L and serum lipase 70 mmol/L. His liver function test (LFT) showed total bilirubin 26 mmol/L, direct bilirubin 9 mmol/L, SGPT (AST) 35 mmol/L, SGOT (ALT) 51 mmol/L, alkaline phosphatase 63 mmol/L. His routine urine microscopy revealed trace amount of albumin, rest being normal.

Patient was observed in red area of ED overnight and was shifted to ED observation following day, where he was observed till 72 hrs post ingestion. During his stay, LFT was repeated and his bilirubin levels (both total and direct) was found to be decreasing. He was discharged after 72 hrs post ingestion and was stable at the time of discharge.

DISCUSSION

Ingestion of whole plant extract of *C. reflexa* Roxb. is known to cause uneasiness, vomiting, anorexia, shooting abdominal pain and purgation in livestock.[5] Whole plant or plant parts are known to affect multiple organ system, including gastro-intestinal system, which is consistent with the initial features seen in our patient.[6]

Young shoots of C. reflexa Roxb. have been used for treatment of jaundice by different ethnic communities of Nepal, which was also the indication for which our patient ingested the whole plant extract.[3] Cases of accidental plant poisoning in humans has been reported and is attributed to various causes.[6] In our case as well, the poisoning was accidental, which probably could be attributed to wrong plant part being used.

Being a parasitic plant, the phytoconstituents present in *C. reflexa* Roxb. varies greatly with the host plant and coumarin derivatives are one of them.[7] As coumarin derivatives are indirectly acting anticoagulants, their effects may become apparent by 24 hours post ingestion with peak effects seen until 2 to 3 days.[8] Thus, the patient was kept under strict observation for 24 hours and tests repeated.

There are 12 different pharmacological actions defined for *C. reflexa* Roxb., of which some were seen in our patients. Extract of *C. reflexa* Roxb. exhibits acetylcholine like activity rabbit's ileum and frog's rectus abdominis muscle and heart muscle; relaxant and spasmolytic effect on small intestine of rabbit and guinea pig; is known to cause bradycardia and hypotension.[7] These effects probably do explain the hypotension seen in our patient, the decreased gastrointestinal motility he developed later (demonstrated by complain of unable to pass stool/flatus) and a lower heart rate. Its extract is also known to show hypoglycemic effect, which was not evident in our patient, could be due to prior medical help received by him at other centers before presenting to our center.

Diagnosis of plant poisoning might not always be straight, and relies heavily on good clinical history combined with careful physical assessment and laboratory tests can be used to confirm the diagnosis if existent.[6,9] Identification of toxidrome and risk assessment of the poisoned patient are important aspect of management of a poisoning patient.9 We managed this case conservatively based on these principles.

The clinical features of the patient could have been distorted due to treatment at other health facilities prior to presenting to us, and also delay in presentation to our center. This could be a potential limitation for our understanding the toxic effects of the plant poisoning under study.

C. reflexa Roxb. can have therapeutic as well as

toxic effect when ingested and both aspects need to be studied. Thus, we would like to recommend further research on its adverse effects as evidences in this regard is minimal.

CONCLUSION

Toxicity due to ingestion of plant extract can present with involvement of multiple organ systems. Identification of toxidrome, appropriate risk assessment and conservative management of a patient presenting with features of toxicity plays a vital role in proper management of the patient.

CONSENT

Written informed consent was obtained from the patient for publication of this case report.

CONFLICT OF INTEREST

None declared.

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