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Case Report

A case study of acute toxicity after consumption of *kokkumarundhu* (carbamate compounds)

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ABSTRACT

Acute poisoning due to ingestion of toxic compounds remains an important medical emergency associated with significant morbidity and mortality. Traditional herbal preparations and locally used substances are increasingly being implicated in poisoning cases because of their easy accessibility and lack of awareness regarding their toxic effects. *Kokkumarundhu* is a traditional paste preparation containing rice and other grains mixed with carbamate compounds and may produce toxic manifestations after ingestion. The presence of associated substance abuse and alcohol consumption may further complicate the clinical presentation and management of such cases. We report the case of a 20-year-old male with a history of chronic smoking, alcohol dependence, and suspected multi-substance abuse who presented to the emergency department with complaints of sudden onset abdominal pain and dizziness following three days of binge alcohol intake. The patient denied nausea, vomiting, chest pain, and dyspnea but complained of chills. Further history from family members revealed ingestion of *kokkumarundhu* approximately four hours prior to presentation. On examination, the patient was conscious, alert, oriented, and afebrile with mild hypertension (BP: 170/60 mmHg). Investigations including serum electrolytes, cardiac biomarkers, and echocardiography were within normal limits, indicating stable cardiovascular status. The acute clinical manifestations were likely due to the combined effects of *kokkumarundhu* ingestion, alcohol consumption, and chronic substance use. Early supportive treatment with intravenous fluids, thiamine, atropine, and close monitoring was initiated to prevent deterioration.
Conclusion: This case highlights the importance of prompt recognition and early management of acute toxicity, particularly in patients with associated substance abuse, to prevent complications and ensure favorable outcomes.

Keywords: *Kokkumarundhu*, Carbamate, Substance abuse, Acute poisoning

INTRODUCTION

Acute poisoning due to pesticide compounds remains a significant public health concern worldwide, particularly in developing countries where agricultural chemicals are easily accessible and frequently misused. Among various pesticide classes, carbamate compounds are commonly employed as insecticides because of their effectiveness against agricultural pests; however, accidental or intentional ingestion can result in severe toxic manifestations. Carbamate poisoning occurs through reversible inhibition of acetylcholinesterase enzymes, leading to accumulation of acetylcholine at synaptic

junctions and excessive stimulation of muscarinic, nicotinic, and central nervous system receptors.[1] Clinical manifestations may range from mild symptoms such as nausea, vomiting, abdominal pain, salivation, sweating, and miosis to severe complications including respiratory distress, bradycardia, hypotension, convulsions, altered sensorium, and death.[2] Early diagnosis and prompt management are essential to prevent morbidity and mortality associated with poisoning.

In certain regions of South India, particularly in rural communities, traditional toxic substances locally known as "*Kokkumarundhu*" have been reported as causes of poisoning. *Kokkumarundhu* commonly contains carbamate compounds and may be consumed accidentally, deliberately for self-harm, or due to lack of awareness regarding its toxic nature. The toxicological profile of carbamate poisoning resembles organophosphorus poisoning; however, carbamate compounds differ by producing shorter duration enzyme inhibition because of reversible binding to cholinesterase enzymes.[3] Diagnosis is mainly based on clinical findings, exposure history, and laboratory investigations such as serum cholinesterase levels.[4] Immediate treatment measures include airway stabilization, supportive management, gastric decontamination when indicated, administration of atropine, and close monitoring for neurological and cardiorespiratory complications.[5]

Case-based evaluation of acute toxicity due to *Kokkumarundhu* consumption is important because published literature on this locally prevalent toxic exposure remains limited. Detailed documentation of clinical presentation, laboratory findings, treatment interventions, and outcomes may improve physician awareness and facilitate timely diagnosis and management of such poisoning cases. The present study aims to evaluate acute toxicity following consumption of *Kokkumarundhu* containing carbamate compounds and to understand its clinical characteristics and outcomes.[6]

CASE REPORT

A 20-year-old male was brought to the Emergency Room (ER) with complaints of sudden onset abdominal pain and dizziness following a history of three days of binge alcohol consumption. The patient had a known history of chronic smoking and alcohol dependence with suspected multi-substance abuse. There was no significant past history of chronic medical illness.

On presentation, the patient denied complaints of nausea, vomiting, chest pain, shortness of breath, or loss of consciousness, but reported associated chills. Further history obtained from the patient's parents revealed that at approximately 10:00 AM, they disclosed that the patient had allegedly consumed *kokkumarundhu*, a traditional herbal preparation containing rice and other grains mixed with a carbamate compound, at around 6:00 AM on the same day.

Table 1: Clinical presentation

Parameters	Main finding
Age and Sex	20-year-old male
Poisoning	*Kokkumarundhu* (carbamate compound)
Time of ingestion	6:00 AM
Presentation	Abdominal pain, dizziness, chills
BP	170/60 mmHg
Consciousness	Conscious, alert and oriented
Cholinergic signs	Absent
Investigations	Electrolytes, cardiac biomarkers and routine blood tests – normal
Echocardiography	Normal
Treatment	IV fluids, thiamine and atropine
Outcome	Stabilized without complications

On physical examination, the patient was conscious, alert, oriented, and afebrile. Vital signs revealed mild hypertension with a blood pressure of 170/60 mmHg, while other vital parameters remained within normal limits. Systemic examination did not reveal any significant abnormality. There were no signs suggestive of severe cholinergic toxicity such as excessive salivation, lacrimation, sweating, respiratory distress, altered sensorium, or muscle fasciculations.

Figure 1: Physical examination.

Laboratory investigations including serum electrolyte levels, cardiac biomarkers, and routine blood investigations were within normal limits. Cardiac assessment with echocardiography did not reveal any abnormalities, indicating a stable cardiovascular status. Despite the relatively mild clinical presentation, considering the history of *kokkumarundhu* ingestion

along with chronic alcohol and substance use, the patient was closely monitored for possible progression of toxic manifestations. The patient was managed with supportive treatment including intravenous fluids, thiamine supplementation, atropine therapy, and continuous monitoring of vital parameters. Early intervention and careful observation helped prevent clinical deterioration and potential complications related to acute toxicity.

DISCUSSION

The present case highlights the clinical profile and management of acute toxicity following ingestion of *Kokkumarundhu* containing carbamate compounds in a 20-year-old male with a history of chronic smoking, alcohol dependence, and suspected multi-substance abuse. The patient presented with sudden onset abdominal pain and dizziness after three days of binge alcohol intake, while further history from family members revealed ingestion of *Kokkumarundhu* approximately four hours before hospital presentation. Despite a history suggestive of toxic exposure, the patient remained conscious, alert, oriented, and afebrile, with only mild hypertension (BP: 170/60 mmHg) and no significant cholinergic manifestations such as salivation, respiratory distress, miosis, muscle fasciculations, or altered sensorium. Laboratory findings including serum electrolytes, cardiac biomarkers, routine blood investigations, and echocardiography remained within normal limits, suggesting a relatively mild toxic presentation. Early supportive management using intravenous fluids, thiamine supplementation, atropine administration, and close clinical monitoring resulted in stabilization without complications. The comparatively favorable outcome in the present case may be attributed to early diagnosis and prompt intervention before progression to severe systemic toxicity.

Previous studies have reported variable manifestations and outcomes following exposure to carbamate and related cholinesterase-inhibiting compounds. Caldas et al reported that exposure to acetylcholinesterase-inhibiting pesticides represented 33.6% of ARfD as methamidophos and 70.2% of ARfD as acephate, while children experienced exposure levels 2.4 times higher than the general population.[7] Although their study focused on dietary exposure rather than acute poisoning, it highlighted the widespread exposure potential of such compounds. Similarly, Roldan-Tapia et al observed significant neuropsychological deficits among 24 acutely poisoned individuals and workers with chronic high exposure exceeding 10 years, with impairments involving perceptual, visuomotor, and memory functions.[8] In contrast, our patient did not demonstrate neurological or cognitive abnormalities, possibly due to lower toxic burden and early management. Boon et al demonstrated cumulative carbamate exposure of 0.64 µg/kg BW/day in the general population and 1.47 µg/kg BW/day among children, suggesting potential toxic risks with increasing exposure.[9] Reddy et al analyzed 441 patients with organophosphorus compound poisoning, of whom 69.16% were males, and 375 survived, with mortality being significantly associated with male gender (OR 2.608) and age >50 years (OR 4.275).[10] The present patient was also male; however, his younger age of 20 years may have contributed to the favorable prognosis. Furthermore, Xia et al presented a case of carbamate poisoning initially misdiagnosed as heatstroke, where the patient developed coma, bradycardia (35 bpm), miosis, pulmonary secretions, and markedly reduced cholinesterase levels (721.72 U/l) requiring intensive care management.[11] Unlike that severe presentation, the current patient exhibited only gastrointestinal symptoms and dizziness without cardiovascular or neurological compromise.

Alcohol intake may have contributed to symptom modification in the present case because chronic alcohol use can alter hepatic metabolism and affect toxin handling. Associated substance abuse may further complicate diagnosis by masking classical cholinergic symptoms. The absence of severe manifestations despite carbamate exposure suggests variability in clinical response depending upon dose, timing of presentation, and associated factors. The present case therefore emphasizes the importance of obtaining a detailed exposure history, particularly in regions where traditional toxic preparations such as *Kokkumarundhu* are accessible. Early recognition and supportive management remain essential for preventing progression to severe toxicity and ensuring favorable outcomes.

CONCLUSION

This case highlights the clinical significance of acute toxicity following the ingestion of *kokkumarundhu*, a traditional preparation containing carbamate compounds, particularly in individuals with a background history of chronic alcohol use and substance abuse. Although the patient initially presented with relatively mild symptoms and stable clinical parameters, the possibility of delayed toxic manifestations warranted careful observation and timely intervention. The coexistence of alcohol dependence and suspected polysubstance abuse may alter the clinical presentation and potentially increase the risk of complications. Early recognition of poisoning through detailed history taking, prompt clinical assessment, and close monitoring played an important role in preventing clinical deterioration. Supportive management with intravenous fluids, thiamine supplementation, and atropine therapy contributed to favorable patient outcomes. This case emphasizes the need for increased awareness regarding the toxic potential of traditional preparations and the importance of rapid diagnosis and appropriate management in acute poisoning cases to reduce morbidity and improve patient outcomes.

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