Cardiotoxicity with Yellow Cow Dung Poisoning

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Abstract
Cow dung powder coloring agent poisoning is common in Southern Tamil Nadu. Both yellow and green varieties are common. Yellow cow dung poisoning usually produces central nervous system (CNS) and hepatic involvement as well as gastrointestinal problems. Though cardiac issues like arrhythmias are seen, toxic myocarditis and cardiac failure are not common. We present a case of a 42-year-old lady with yellow cow dung poisoning who developed toxic myocarditis and cardiac failure with complete recovery over a period of time.

Introduction
Cow dung has widely been used in Southern India since ancient times due to its germicidal properties. Nowadays it has been replaced by synthetic compounds popularly known as saani powder. It is available in two colors—yellow and green. Yellow powder primarily contains auramine O—diarylmethane dye and green powder malachite green—triphenylmethane dye. Cow dung poisoning has commonly been reported in Coimbatore, Erode, and Tirupur districts of Tamil Nadu.1

Case Description
This 42-year-old lady, with no known comorbidities, hailing from Coimbatore, presented with an alleged history of consumption of yellow cow dung (one teaspoon mixed with banana) poisoning around 3.00 pm at her residence. She had three episodes of vomiting on the way to the hospital. On arrival in the emergency room, she was conscious, oriented, and hemodynamically stable. Systemic examination was unremarkable. Blood investigations revealed normal counts, sugars, renal, thyroid, and liver parameters. Electrocardiogram (ECG) was normal. She was treated with intravenous fluids and other supportive measures. The following day she developed asymptomatic bradycardia and hypotension. ECG showed sinus bradycardia. A cardiologist’s opinion was obtained, and a bedside echocardiogram (ECHO) (on 2nd day) revealed adequate left ventricle (LV) function with no regional wall motion abnormalities. Since hypotension and bradycardia persisted despite the fluid challenge, dopamine infusion was initiated. Serum electrolytes were normal. As she continued to remain bradycardic and hypotensive, after ruling out dyselectrolytemia, ECG and ECHO were repeated (on 3rd day) which showed new onset ST segment- (ECG term) depression in anterolateral leads and global hypokinesia of LV with moderate LV dysfunction and elevated high-sensitive troponin I (1650) and N-terminal pro-brain natriuretic peptide (8670) levels. Toxic myocarditis secondary to yellow cow dung poisoning was suspected. Serial liver and renal parameters were normal. Her cardiac status gradually improved, bradycardia and hypotension improved, and was weaned off inotropes. Repeat ECHO (done on the 10th day) showed adequate LV function and no regional wall motion abnormalities. The patient remained clinically stable and was discharged home.

Discussion
Auramine O is a diarylmethane dye used as a fluorescent stain which is known to induce in vivo deoxyribonucleic acid damage to liver, kidney, and bone marrow cells.2 Easy availability of this product locally, makes it a common household poison in regions in and around Coimbatore. There are very few articles in the literature regarding this common household poisoning, hence, the mechanism of action, clinical presentation, and cause of death are not clearly documented in many textbooks of medicine.

There is no specific antidote for these dyes. Deaths can occur within hours of ingestion due to cerebral edema, resulting in convulsions, coma respiratory, cardiac arrest, and death, or after 2–3 days due to the direct action of dye on the liver causing centrilobular necrosis, resulting in hepatitis and fulminant hepatic failure.3

The common clinical symptoms following auramine poisoning are staining all over the body, especially hands, face, and tongue; nausea, vomiting, abdominal pain, cramps; diarrhea, confusion, and irritability. It is a gastrointestinal tract irritant causing mucosal damage, epigastric pain, and discomfort. It is a neurotoxic poison causing CNS depression leading to altered mentation, coma, seizures, and a low Glasgow Coma Scale.4 Tachypnoea and respiratory distress needing mechanical ventilation have been recorded earlier. Tachycardia, metabolic acidosis, and hyperglycemia were also observed with auramine poisoning.5 Ventricular dysrhythmias, such as monomorphic ventricular tachycardia, were noticed, particularly in patients with underlying cardiac diseases. Murugananthan et al. in their study noticed 55.1% of patients developed hypotension during their course of treatment which responded well to fluids and dopamine infusion.6

Though tachycardia, hypotension, and arrhythmias mainly ventricular tachyarrhythmia have been recorded, no cases of bradycardia or toxic myocarditis have been reported with auramine O poisoning so far. Our patient had no recorded cardiac abnormalities at the time of admission and had complete recovery of cardiac dysfunction at the time of discharge points out in favor of the unusual cardiotoxicity of this molecule.

Conclusion
The treatment of poisoning caused by an uncommon compound is always challenging and the situation becomes graver when the patient develops an unexpected complication and does not respond properly to treatment. Further studies are necessary to elucidate this fatal poison in a broader aspect and this article will serve as a guide for future research.

References
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