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Severe suicidal poisoning with Auramine-o and amitriptyline

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Abstract
Auramine-o is yellow colored synthetic cow dung powder. In the rural area of Tamil Nadu, cow dung powder was commonly used as suicidal poison. It has a very high toxicity profile due to which even death can occur within hours of ingestion. Till now there is no specific antidote available for this poison. Amitriptyline, commonly prescribed antidepressant agent is very often involved in attempted suicides. Amitriptyline poisoning, which can be life-threatening, remains a significant clinical issue. The situation becomes harder when these two deadly poisons were taken together. Here we report a rare and severe poisoning case of 52 years old female who consumed yellow cow dung powder along with 100 Amitriptyline tablets as an attempt to suicide. The patient underwent gastric lavage thrice along with activated charcoal as a mean of gut decontamination. Supportive treatments for the clinical manifestations were given.

Keywords: cow dung, poison, Auramine-o, amitriptyline

Introduction
Auramine-o is yellow colored synthetic cow dung powder [1]. In the rural areas of Tamil Nadu, cow dung powder was commonly used as a suicidal poison [2]. It has a very high toxicity profile due to which even death can occur within hours of ingestion. This neurotoxic poison can cause CNS depression and severe hepatic damage [3]. Till now there is no specific antidote available for this poison [4].
Amitriptyline, commonly prescribed antidepressant agent is very often involved in attempted suicides. Its poisoning can be life-threatening, remains a significant clinical issue and necessitates primary therapeutic approaches of gastric irrigation and administration of activated charcoal recurrently [5].

Case report
A 52 -years-old woman ingested yellow sani powder with an attempt of committing suicide along with 'Amitriptyline 25 mg' 100 tablets at 7.30 pm in her residence. The patient was apparently normal until 8 pm. Patient relatives came to know the condition around 10.15 pm. She was first admitted to a private hospital in Tiruchengode with complaints of yellow discoloration all over the body and then referred to a multispecialty hospital. While shifting, she had multiple episodes of vomiting.
On receiving, the patient was unconscious, gasping and yellowish discoloration seen all over the body, the pulse rate was 65 beats/minute and blood pressure 70/40 mmHg. Both pupils were 2mm with sluggish reaction to light and soft bowel sound was present in the abdomen. Glasgow Coma Scale Score (GCS) was taken 3/15. In view of poor GCS, under aseptic precaution patient was intubated with 7.5 Endotracheal Tube fixed at 20cm length; BAE (Bronchial asthma exacerbation) was equal on both side. Ryle's tube inserted and stomach wash was done. Yellow colored gastric content aspirated. Bains circuit ventilation was started just before intubating. Cardiologist opinion was taken and Echo showed normal LV function with moderate Mitral Regurgitation. Complete haemogram report showed increase in total leucocyte count (TLC) count (12,700cells/cumm) and differential leucocyte count (Polymorph- 73 %, Monocyte -5 %, Basophil - 3%). On the first day, the patient was found to be hyperglycemic with a random blood sugar of 235 mg% and the presence of sugar (+) was seen in urine. The patient's total Creatinine Phosphokinase (CPK) level was elevated .The serum electrolytes level were normal on all days except for the serum potassium level reduction on third day 3.1(3.5-5.1mmol/L). So, syrup Potassium chloride 10ml (1-1-1) was given to the patient. The values of renal function test and arterial blood gas (ABG) analysis were normal. Patient's coagulation parameters were prolonged with prothrombin time.
The patient regained consciousness on the second day and continued the monitoring. In liver function test, all parameters were found to be normal except the values of Alkaline phosphatase (ALP) and Serum Glutamic Oxaloacetic Transaminase (SGOT) which was elevated consistently and Serum Glutamic Pyruvic Transaminase (SGPT) level elevated on last day.

Table 1: Clinical evaluation of patient with Auramine-o and Amitriptyline poisoning

Parameters	Day 1	Day 2	Day 3	Day 4	Day 5
Bilirubin Direct (mg/dl)	0.3	0.2	0.3	0.2	0.2
Bilirubin Indirect (mg/dl)	0.6	0.6	0.6	0.6	0.7
Albumin/Globulin Ratio	1.6	1.4	1.5	1.4	1.3
Alkaline Phosphatase (U/L)	97	128↑	135↑	122↑	127↑
Serum Glutamic Oxaloacetic Transaminase (U/L)	19	73↑	71↑	68↑	129↑
Serum Glutamic Pyruvic Transaminase (U/L)	15	44	55	51	91↑
Creatinine Phosphokinase(U/L)	235	479	---	---	---
Prothrombin Time	16.1 sec↑	15.7sec↑	19.6sec↑	17.3sec↑	---
INR	1.19	1.21	1.57	1.31	---
APTT	---	28.9sec↑	33.6sec↑	31.7sec↑	---

INR- International Normalized Ratio, APTT- Activated Partial Thromboplastin Time

Gastroenterologist opinion was taken and supportive treatment of Ursodeoxycholic acid (UDCA) and Silymarin were added. After 36 hours the patient was extubated and alerted with the resolution of hypotension and bradycardia. The patient was treated with IV fluids, Cefoperazone + Sulbactam antibiotic, Activated charcoal, Inj. Hydrocortisone, Inj. Vitamin K, Nebulization with Levosalbutamol and Budesonide with other supportive care. Psychiatric opinion obtained. The patient was hemodynamically stable on sixth day and discharged.

Discussion

Auramine-o (C 17H12 N3.HCL) is manufactured industrially from N, N – dimethylaniline, and formaldehyde. It can cause centrilobular necrosis of liver which is either dose-related or toxic metabolite related hepatotoxicity [2]. The clinical features of its poisoning started after 15 to 30 minutes of ingestion includes burning in stomach, abdominal disturbances, increased salivation, nausea, vomiting, a difference in heart rate, and neurological features include convulsion, coma and even death. Yellowish discoloration of patient's skin due to deposition of powder in the different part of the body was same as observed in Khaja Mohideen *et al* study [3]. The patient had features of toxic hepatitis from day two of poison intake which was evident from the elevating SGPT, SGOT and coagulation parameters. Supportive treatment of Ursodeoxycholic acid and Silymarin was given under the instruction of Gastroenterologist. UDCA have multiple hepatoprotective activities like choleretic effect, cytoprotective, antiapoptotic and immunomodulatory properties that were evident from Angulo P study [6]. Silymarin has the effect of liver parenchymal regeneration, antioxidant property and protects hepatocyte membrane.

The patient has elevated prothrombin time, so injection Vitamin K was given as treatment. The direct central nervous system (CNS) effect of Auramine-o as a neurotoxic poison was evident from the GCS score of 3/15. So that the patient was intubated and proper ventilation was given. The patient underwent gastric lavage thrice along with activated charcoal as a mean of gut decontamination.

For Amitriptyline, serum levels of 50-200 ng/ml are considered to be therapeutic, but >1000 ng/ml was a toxic concentration. Amitriptyline mainly acts by inhibiting the uptake of amines by nerve terminals, but also has antimuscarinic and antihistamine properties. Due to the Amitriptyline poisoning, there was an elevation in CPK level as a result of serum enzyme disturbances similar to Shannon M study [7] and hypotension occur as a result of myocardial depression owing to sodium channel blocking properties as well as receptor antagonism was observed. The patient went bradycardia which differs from the cases observed by Hisham *et al.* [2] where the patient's had tachycardia. Hyperglycemia and respiratory distress were seen in this patient.

Elevation in the total leukocyte count 12,700 cells/cumm was seen. Leucocytosis was a common feature for both auramine-o poisoning and Amitriptyline poisoning [5, 8]. In contrast to the metabolic acidosis observed by Hisham *et al*, the ABG analysis was normal in this case [2].

Even though there is no specific antidote for auramine-o, N-acetyl cysteine (NAC) was given as a hope of survival because of its proven anti-oxidative effect same like in studies done by Avinash *et al.* [1].

Conclusion

The treatment of poisoning from these type of fatal compound will be a challenge. Other than supportive treatment for the clinical manifestation, there is no specific antidote was available for Auramine-o. So it is better to develop a standard treatment protocol in order to reduce the mortality and morbidity due to the toxic exposure of this lethal poison.

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