Section: General Medicine

Original Research Article

CLINICO-DEMOGRAPHIC PROFILE AND PROGNOSIS OF COW DUNG POWDER INGESTION: A HOSPITAL-BASED PROSPECTIVE OBSERVATIONAL STUDY

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Received *: 11/03/2026*
Received in revised form *: 26/04/2026*
Accepted *: 14/05/2026*

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DOI: 10.70034/ijmedph.2026.2.317

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (2); 1894-1900

ABSTRACT

Background: Synthetic cow dung powder poisoning is an emerging toxicological emergency increasingly encountered in Southern India due to its easy availability and low cost. These products, commonly marketed under names such as *Sowbhagya* and *Mahalakshmi*, usually contain Auramine-O dye along with turmeric and dried cow dung powder. Auramine-O is the principal toxic component responsible for systemic toxicity, predominantly affecting the liver. Limited prospective studies are available regarding the clinical manifestations and prognosis of this poisoning.

Materials and Methods: This prospective observational study was conducted in the Department of General Medicine, Government General Hospital, Kurnool, from December 1st 2025 to May 31st 2026. A total of 100 patients with alleged history of synthetic cow dung powder ingestion were included. Detailed clinical history, examination findings, laboratory investigations, treatment details, and outcomes were recorded and analyzed.

Results: The majority of patients were young adults, and suicidal ingestion was the predominant mode of poisoning. Common presenting symptoms included nausea, vomiting, abdominal pain, and giddiness. Elevated serum AST and ALT levels were the most frequent biochemical abnormalities observed, while serum bilirubin remained within normal limits in many patients, indicating a predominantly hepatocellular pattern of injury. Bradycardia was observed in some patients. Patients with larger quantity ingestion and delayed hospital presentation showed increased severity of illness and prolonged hospital stay. Supportive treatment including N-acetylcysteine was associated with favorable outcomes in patients with hepatic dysfunction.

Conclusion: Synthetic cow dung powder poisoning predominantly produces hepatocellular injury secondary to Auramine-O toxicity. Elevated transaminases with relatively preserved serum bilirubin constitute an important biochemical pattern in this poisoning. Early recognition, close monitoring of liver function, and prompt supportive management significantly improve prognosis. Quantity consumed and delay in presentation are important predictors of outcome.

Keywords: Synthetic cow dung powder poisoning, Auramine-O, hepatotoxicity, prognosis, transaminitis, deliberate self-harm.

INTRODUCTION

Cow dung has traditionally been used in Indian households, particularly in rural and semi-urban areas, for sanitation and domestic cleaning because of its perceived antiseptic and germicidal properties.[1] With declining availability of natural cow dung and changing household practices,

synthetic alternatives have increasingly replaced traditional preparations. One such substitute is synthetic cow dung powder, commonly referred to as "Saani powder," "yellow powder," or "Manjal Saani powder" in South India.[2]

These products are widely available in local markets under trade names such as Sowbhagya and Mahalakshmi, usually in small 10-gram packets.[2] The commonly used constituents include Auramine-O dye, turmeric powder, and dried cow dung powder. Among these, Auramine-O is the principal toxic component responsible for systemic toxicity following ingestion.[6]

Despite legal restrictions and bans on Auramine-containing products, synthetic cow dung powder continues to be easily accessible due to its low cost and widespread availability.[2] In recent years, particularly in Andhra Pradesh, Karnataka, and adjoining regions, it has emerged as an important agent of deliberate self-harm.[3]

Auramine-O is a synthetic diarylmethane dye with known hepatotoxic, neurotoxic, and mutagenic properties.[6] Experimental studies have demonstrated oxidative stress, DNA damage, and hepatocellular injury associated with this compound.[5] Clinical manifestations following ingestion range from mild gastrointestinal symptoms such as nausea, vomiting, and abdominal pain to severe complications including hepatic dysfunction, cardiovascular abnormalities, altered sensorium, and multi-organ failure.[4,13]

Hepatic involvement appears to be the predominant toxic manifestation in most patients. Elevated serum transaminases, particularly AST and ALT, are frequently observed, while serum bilirubin may remain normal in many cases, suggesting a predominantly hepatocellular pattern of injury.[8] Bradycardia and mucocutaneous yellow pigmentation have also been reported in some patients.[1]

Currently, available literature on synthetic cow dung powder poisoning is limited mainly to isolated case reports and small case series.[3,4] Prospective observational studies evaluating the clinical profile, biochemical abnormalities, treatment outcomes, and prognostic indicators are scarce. Hence, the present study was undertaken to evaluate the clinical manifestations and prognosis of patients presenting with synthetic cow dung powder ingestion in a tertiary care hospital setting.

Aim

To evaluate the clinical manifestations, biochemical abnormalities, and prognosis of patients with synthetic cow dung powder (Auramine-O) ingestion admitted to a tertiary care hospital.

Objectives

1. To assess the biochemical abnormalities associated with synthetic cow dung powder (Auramine-O) poisoning.
2. To evaluate the clinical course and outcomes of affected patients during hospital stay.
3. To determine the prognostic factors influencing outcome in synthetic cow dung powder poisoning.

MATERIALS AND METHODS

Study Design: This study was designed as a prospective observational study.

Study Setting: The study was conducted in the Department of General Medicine, Government General Hospital, Kurnool, attached to Kurnool Medical College, Andhra Pradesh. The hospital is a tertiary care referral center catering to patients from rural and urban areas of Rayalaseema region.

Study Duration: The study was conducted over a period of four months from December 1st 2025 to May 31st 2026

Study Population: The study population consisted of 100 patients admitted to the medical wards and Intensive Care Unit (ICU) of Government General Hospital, Kurnool, with alleged history of cow dung powder ingestion during the study period. Both male and female patients aged 18 years and above were included in the study.

Sample Size: A total of 100 patients with alleged history of synthetic cow dung powder ingestion who fulfilled the inclusion criteria were included in the study.

Inclusion Criteria

1. Patients aged 18 years and above.
2. Patients admitted with alleged history of synthetic cow dung powder ingestion.
3. Both male and female patients willing to provide valid informed consent.
4. Patients presenting within 72 hours of ingestion.

Exclusion Criteria

1. Patients with pre-existing chronic liver disease.
2. Patients with known chronic kidney disease or significant systemic illness.
3. Patients with co-ingestion of other poisons or substances.
4. Patients unwilling to provide valid informed consent.
5. Pregnant women and patients below 18 years of age were excluded from the study.

Methodology: After obtaining approval from the Institutional Ethics Committee and informed consent from patients or attendants, all eligible patients admitted with alleged history of synthetic cow dung powder ingestion were included in the study. Detailed history regarding age, gender, quantity consumed, trade name of poison, mode of poisoning, time of ingestion, and time interval between ingestion and hospital presentation was obtained. Address details including area/region of residence (e.g., Bethamcherla and surrounding areas) were also recorded to analyze the geographic distribution of cases. Clinical symptoms including nausea, vomiting, abdominal pain, giddiness, altered sensorium, jaundice, breathlessness, reduced urine output, and seizures were documented. Detailed

general physical examination and systemic examination were performed in all patients. Vital parameters such as pulse rate, blood pressure, respiratory rate, temperature, and oxygen saturation were monitored during hospital stay. Relevant laboratory investigations including complete blood count, liver function tests, renal function tests, serum electrolytes, random blood sugar, electrocardiogram, and arterial blood gas analysis where indicated were performed at admission. Serial monitoring of liver function tests, particularly AST, ALT, and serum bilirubin, was carried out on Day 1 and Day 3 to assess hepatic involvement.

All patients received supportive treatment including intravenous fluids, proton pump inhibitors, antiemetics, and symptomatic care. Gastric lavage was performed in patients presenting early after ingestion as part of initial decontamination measures N-acetylcysteine was administered in patients with elevated liver enzymes suggestive of hepatocellular injury. Ursodeoxycholic acid was used in selected patients with persistent hepatic dysfunction. Patients requiring intensive monitoring or ventilatory support were managed in the intensive care unit.

Patients were followed throughout their hospital stay, and clinical outcomes including recovery, complications, duration of hospitalization, and mortality were recorded and analyzed.

Outcome Measures

The following outcome measures were assessed in all patients during the study period:

- Clinical manifestations at presentation
- Biochemical abnormalities, particularly hepatic involvement
- Requirement of ICU admission or ventilatory support
- Development of complications during hospital stay
- Duration of hospitalization
- Recovery or mortality at discharge
- Overall prognosis based on clinical and biochemical improvement

Ethical Approval: The study protocol was reviewed and approved by the Institutional Ethics Committee of Kurnool Medical College and Government General Hospital, Kurnool, Andhra Pradesh. The study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki. Written informed consent was obtained from all participants or their legally authorized attendants prior to inclusion in the study.

Statistical Analysis: The collected data were compiled in Microsoft Excel and analyzed using SPSS software version 25.0. Quantitative variables were expressed as mean ± standard deviation and qualitative variables were expressed as frequencies and percentages. Comparison of laboratory parameters between Day 1 and Day 3 was performed using paired t-test. Association between clinical variables and outcome was assessed using Chi-square test. A p-value of <0.05 was considered statistically significant.

RESULTS

A total of 100 patients with alleged history of synthetic cow dung powder ingestion admitted to Government General Hospital, Kurnool, during the study period from December 1st 2025 to May 31st 2026 were included in the study.

Table 1: Background Distribution of Study Population

Background / Area of Residence	Number of Patients	Percentage (%)
Rural	58	58%
Semi-urban	26	26%
Urban	16	16%
Total	100	100%

Note: Majority of patients belonged to rural background (58%), followed by semi-urban areas (26%), while urban patients constituted 16% of the study population. This finding suggests higher occurrence of synthetic cow dung powder ingestion among rural populations, possibly due to easier availability and accessibility of the substance in these regions.

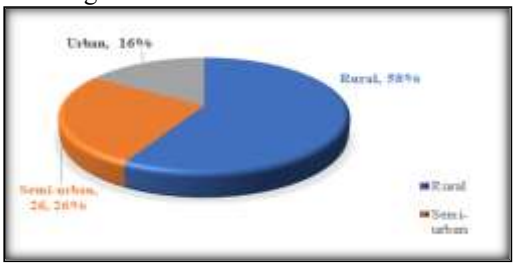

Figure 1: Background Distribution of Study Population (n = 100)

Note: Rural patients constituted the majority of the study population, followed by semi-urban and urban populations, indicating higher prevalence of synthetic cow dung powder ingestion in rural areas.

Gender Distribution: Among the study population, 34 patients were males and 66 patients were females, showing female predominance in the present study.

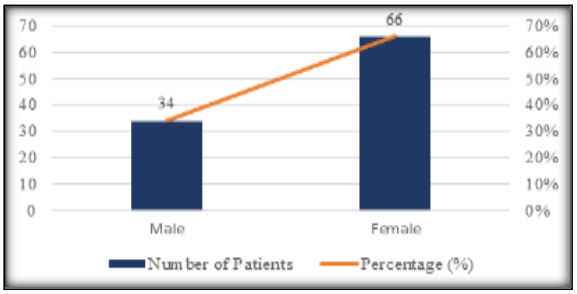

Figure 2: Gender Distribution of Study Population (n = 100)

Note: Female patients constituted the majority of the study population, accounting for 66% of cases, while male patients accounted for 34%. This finding indicates a female predominance in synthetic cow dung powder poisoning in the present study.

Clinical Presentation

Most patients belonged to the young and middle-aged age group, and suicidal ingestion was the predominant mode of poisoning. The commonly consumed preparations were locally available brands such as Sowbhagya and Mahalakshmi, usually marketed in 10-gram packets containing Auramine-O dye, turmeric, and dried cow dung powder.

The majority of patients presented to the hospital within 24 hours of ingestion. The most common presenting symptoms were nausea, vomiting, abdominal pain, and giddiness. A smaller proportion of patients presented with altered sensorium, bradycardia, breathlessness, yellowish discoloration of skin, nails, and soles, and jaundice.

Table 2: Common Presenting Symptoms

Symptoms	Number of Patients	Percentage (%)
Nausea	82	82%
Vomiting	78	78%
Abdominal pain	64	64%
Giddiness	48	48%
Bradycardia	16	16%
Altered sensorium	12	12%
Yellowish discoloration of skin, nails, and soles	12	12%
Breathlessness	10	10%
Jaundice	2	2%

Note: Nausea and vomiting were the most common presenting symptoms observed among patients with synthetic cow dung powder ingestion, followed by abdominal pain and giddiness. Cardiovascular and neurological manifestations such as bradycardia and altered sensorium were observed in severe cases. Yellowish discoloration of skin, nails, and soles was observed in some patients due to Auramine-O pigmentation.

Biochemical Abnormalities: Liver function abnormalities constituted the predominant biochemical abnormality in the study. Elevated serum AST and ALT levels were observed in the majority of patients, whereas serum bilirubin remained within normal limits in many cases, indicating a predominantly hepatocellular pattern of liver injury. Serial monitoring demonstrated further elevation of liver enzymes on Day 3 in some patients.

Table 3: Comparison of Laboratory Parameters on Day 1 and Day 3

Parameter	Day 1 Mean ± SD	Day 3 Mean ± SD
AST (U/L)	86 ± 42	132 ± 58
ALT (U/L)	92 ± 48	146 ± 64
Serum Bilirubin (mg/dL)	0.9 ± 0.3	1.1 ± 0.4
Blood Urea (mg/dL)	28 ± 10	34 ± 14
Serum Creatinine (mg/dL)	0.9 ± 0.2	1.2 ± 0.4

Note: Significant elevation of AST and ALT levels was observed on Day 3 compared to Day 1, indicating progressive hepatocellular injury. Serum bilirubin levels showed minimal elevation in most patients, suggesting predominantly hepatocellular rather than cholestatic liver involvement. Renal function parameters remained within normal limits in the majority of patients during hospital stay.

Clinical Outcome: Patients with larger quantity ingestion and delayed presentation to the hospital showed greater biochemical derangement, prolonged hospital stay, and poorer prognosis. Most patients improved with supportive treatment including intravenous fluids, antiemetics, proton pump inhibitors, and N-acetylcysteine. A small number of patients required ICU admission and intensive monitoring.

Table 4: Clinical Outcome of Patients

Outcome	Number of Patients	Percentage (%)
Recovered	90	90%
Improved	6	6%
ICU Admission	8	8%
Mortality	4	4%

Note: Most patients recovered with supportive treatment and close monitoring. Poor clinical outcomes, including ICU admission and mortality, were observed mainly in patients with delayed presentation, larger quantity ingestion, severe hepatocellular injury, bradycardia, and delayed hospital presentation.

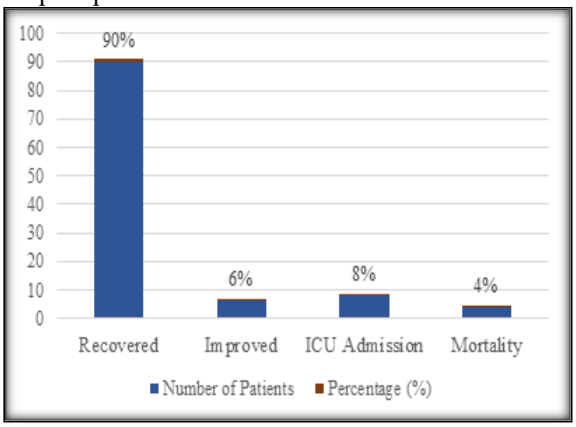

Figure 3: Clinical Outcome of Patients

Note: Majority of patients showed complete recovery with supportive management. ICU admission and mortality were predominantly observed in patients with delayed hospital presentation, larger quantity ingestion, severe hepatocellular injury, bradycardia, and associated renal dysfunction. ICU admission and mortality were predominantly observed in patients with delayed hospital presentation, larger quantity ingestion, severe hepatocellular injury, and bradycardia.

Prognostic Factors

Poor prognosis was associated with large quantity ingestion, delayed hospital presentation, elevated liver enzymes, bradycardia, and altered sensorium.

Table 5: Prognostic Factors Associated with Poor Outcome

Prognostic Factors	Association with Poor Prognosis
Large quantity ingestion	Present
Delayed hospital presentation	Present
Elevated AST and ALT	Present
Bradycardia	Present
Altered sensorium	Present
Renal dysfunction	Present

Note: Large quantity ingestion, delayed hospital presentation, elevated liver enzymes, bradycardia, altered sensorium, and renal dysfunction were significantly associated with poor prognosis. Patients presenting early and receiving prompt supportive treatment showed better clinical outcomes and shorter duration of hospital stay.

DISCUSSION

Synthetic cow dung powder poisoning is increasingly emerging as an important toxicological emergency in Southern India due to the easy availability, low cost, and widespread accessibility of Auramine-O containing preparations.[1,2] Despite legal restrictions on Auramine-based compounds, these products continue to be commonly used in rural households and are increasingly implicated in deliberate self-harm.[2] The present study was undertaken to evaluate the clinical manifestations, biochemical abnormalities, and prognosis associated with synthetic cow dung powder ingestion in a tertiary care setting.

In the present study, the majority of patients belonged to the young and middle-aged age group, with female predominance accounting for 66% of cases. Similar demographic patterns were reported by Muruganathan et al,[4] and Sherfudeen KM,[3] where young females constituted the majority of Auramine-O poisoning cases. The higher incidence among females may be attributed to psychosocial stressors, impulsive deliberate self-harm behavior, and easy household accessibility of the poison.

The majority of patients in the present study belonged to rural areas (58%), followed by semi-urban (26%) and urban populations (16%). Similar rural predominance has been reported in previous studies on Auramine-O poisoning.[2,4] The higher incidence in rural populations may be attributed to easier availability, widespread domestic use, lower socioeconomic status, and increased accessibility of synthetic cow dung powder in rural households.

The most common presenting symptoms in the present study were nausea, vomiting, abdominal pain, and giddiness. Similar gastrointestinal manifestations have been reported in previous studies on Auramine-O poisoning.[1,4] These symptoms are likely due to direct irritation of the gastrointestinal mucosa and systemic toxic effects of Auramine compounds.[13] Persistent vomiting and abdominal pain were more commonly observed in patients with larger quantity ingestion, suggesting dose-related toxicity.

Gastric lavage was performed in patients presenting early after ingestion as part of initial decontamination measures. Early gastric lavage may reduce systemic absorption of Auramine-O and thereby limit toxicity. Similar supportive management approaches have been described in

earlier studies where prompt decontamination and early hospital presentation were associated with better clinical outcomes.[4,13] However, gastric lavage is most beneficial when performed within the early hours of ingestion and in carefully selected patients with protected airway reflexes.

Bradycardia was observed in a subset of patients and was associated with severe poisoning and delayed presentation. Similar cardiovascular manifestations were reported by Hisham et al.[1] The probable mechanisms include autonomic dysfunction, direct cardiotoxic effects, metabolic disturbances, and increased vagal stimulation secondary to persistent vomiting.[7]

Hepatic involvement was the predominant biochemical abnormality observed in the present study. Elevated AST and ALT levels were seen in most patients, whereas serum bilirubin remained relatively normal in many cases, suggesting predominantly hepatocellular rather than cholestatic liver injury. Similar findings were reported by Muruganathan et al,[4] and Lee WM,[8] who described transaminitis as the major manifestation of toxin-induced hepatic injury. Experimental studies by Subramanian et al,[5] demonstrated that Auramine-O induces oxidative stress and hepatocellular necrosis, leading to leakage of intracellular liver enzymes into circulation.

Serial monitoring of liver function tests demonstrated progressive elevation of AST and ALT levels from Day 1 to Day 3 in some patients, indicating ongoing hepatocellular injury during the early phase of poisoning. Similar biochemical progression has been reported in toxin-induced hepatitis and drug-induced liver injury.[8,12] Renal dysfunction was observed only in a small proportion of patients and was mainly associated with severe poisoning, dehydration, or systemic toxicity.[13]

N-acetylcysteine (NAC) was administered in patients with elevated liver enzymes because of its antioxidant and hepatoprotective properties. Most patients who received early NAC therapy showed favorable clinical and biochemical improvement. Similar beneficial effects of NAC in toxic hepatocellular injury have been reported by Prescott et al,[9] NAC acts by replenishing intracellular glutathione stores, reducing oxidative stress, and improving hepatic microcirculation.[14]

Most patients in the present study recovered with supportive management including intravenous fluids, antiemetics, proton pump inhibitors, gastric lavage, NAC therapy, and close monitoring of liver function tests. Similar favorable outcomes with conservative treatment were reported in earlier studies on Auramine poisoning.[3,4] Poor prognosis was associated with delayed hospital presentation, large quantity ingestion, severe transaminitis, altered sensorium, bradycardia, and renal dysfunction. Comparable prognostic indicators have been described in previous toxicology studies involving hepatotoxic compounds.[7,13]

The mortality rate in the present study was 4%, which is comparable with earlier reports on Auramine poisoning.[1,4] Mortality was predominantly observed in patients presenting late with severe hepatic dysfunction and systemic complications. Early recognition, prompt supportive management, and serial monitoring of liver function tests are therefore essential in improving patient outcomes.

The findings of the present study are broadly comparable with previously published studies on Auramine-O poisoning, which identified hepatotoxicity as the principal systemic manifestation.[4,5] However, prospective observational studies on synthetic cow dung powder poisoning remain limited. The present study contributes additional prospective clinical and biochemical data regarding this increasingly encountered poisoning pattern in Southern India.

The major limitations of the present study were its single-center design, relatively limited sample size, lack of toxicological confirmation of serum Auramine-O levels, and absence of long-term follow-up. Further multicentric studies with larger sample sizes are required to better understand the pathophysiology, prognostic indicators, and optimal management strategies for synthetic cow dung powder poisoning.

CONCLUSION

Synthetic cow dung powder poisoning is an emerging toxicological emergency increasingly encountered in Southern India due to the easy availability and low cost of Auramine-O containing preparations. Gastrointestinal symptoms such as nausea, vomiting, abdominal pain, and giddiness were the most common clinical manifestations observed in the present study. Hepatic involvement was the predominant systemic abnormality, characterized mainly by elevated serum AST and ALT levels with relatively preserved serum bilirubin levels, suggesting a predominantly hepatocellular pattern of liver injury.

Patients with larger quantity ingestion, delayed hospital presentation, bradycardia, altered sensorium, and renal dysfunction had poorer prognosis and prolonged hospital stay. Early recognition, prompt supportive management, serial monitoring of liver function tests, and timely administration of N-acetylcysteine were associated with favorable clinical outcomes in most patients. Further multicentric studies are required to better understand the long-term outcomes and optimal management strategies for synthetic cow dung powder poisoning.

Limitations: This was a single-center study with a relatively small sample size, limiting generalizability. Toxicological confirmation of serum Auramine-O levels could not be performed due to limited laboratory facilities.

Histopathological assessment and long-term follow-up were not feasible. Variability in the composition of synthetic cow dung powder preparations may have influenced clinical severity. Further multicentric studies with larger sample sizes are required to validate these findings.

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