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CLINICAL SPECTRUM AND MANAGEMENT OF *CITRULLUS COLOCYNTHIS* (KUMATIKKAI) POISONING: A CASE SERIES FROM SOUTH INDIA

Sundaram R[1*], Vasanthi R[2], Krishnarajan P[3], Sugadev S[3], Prithviraj T[4], Revathi N[4], and Rajarajeswari S[5]

[1*]Professor of General Medicine, Madurai medical college, Madurai, Tamil Nadu.
[2]Assistant Professor of Microbiology, Madurai medical college, Madurai, Tamil Nadu.
[3]Assistant Professor of General Medicine, Madurai medical college, Madurai, Tamil Nadu.
[4]Junior Resident in General Medicine, Madurai medical college, Madurai, Tamil Nadu.
[5]Third professional year MBBS student, Madurai medical college, Madurai, Tamil Nadu.

***Corresponding Author:** Dr. R. Sundaram
*Professor of General Medicine, Madurai medical college, Madurai, Tamil Nadu.

Abstract

Background: *Citrullus colocynthis* (commonly known as Kumatikkai or Bitter Apple) is a toxic member of the Cucurbitaceae family, often mistaken for edible melons or gourds in rural India. Accidental or intentional ingestion of its fruit can result in a range of clinical manifestations, from mild gastrointestinal upset to severe hemorrhagic colitis and hepatic dysfunction.

Objective: To describe the clinical spectrum, diagnostic features, and management outcomes of *Citrullus colocynthis* poisoning based on a series of seven cases admitted to Government Rajaji Hospital, Madurai.

Methods: A retrospective case series of seven patients (ages 13–42) presenting with confirmed *C. colocynthis* ingestion between April and September 2025 was analyzed. Data on exposure type, quantity ingested, clinical presentation, laboratory/imaging findings, treatment, and outcomes were collected and reviewed.

Results: Of the seven cases, five were accidental ingestions and two were intentional. Common symptoms included nausea (100%), vomiting (71%), abdominal pain (57%), diarrhea (43%), and hematochezia (29%). Two patients developed hemorrhagic colitis confirmed by CECT imaging. Three patients exhibited transient hepatic dysfunction with elevated bilirubin levels, one peaking at 4.0 mg/dL. All patients received supportive management including IV fluids, antiemetics, proton pump inhibitors, and ursodeoxycholic acid where indicated. Antibiotics were administered in cases with radiological evidence of colitis. No fatalities occurred, and all patients recovered fully.

Conclusion: *Kumatikkai* poisoning presents with a broad clinical spectrum, ranging from mild gastrointestinal symptoms to colitis and hepatic dysfunction. While the prognosis is generally favorable with early supportive care, public awareness and accurate plant identification are crucial to prevent accidental ingestions. Clinicians should maintain a high index of suspicion in endemic areas to enable prompt diagnosis and management.

Keywords: *Citrullus colocynthis*, *Kumatikkai*, Plant poisoning, Herbal toxicity, Acute gastroenteritis

Introduction

Poisoning due to ingestion of plant-based substances continues to be a significant yet underrecognized cause of morbidity and mortality in many developing countries, including India. While industrial and pharmaceutical poisons often receive clinical attention, plant toxins especially those derived from locally available or traditionally used flora constitute a major portion of accidental and deliberate poisoning cases, particularly in rural settings. Among these, the Cucurbitaceae family is noteworthy for containing both edible and toxic members that are morphologically similar, often leading to misidentification and accidental ingestion [1]. Citrullus colocynthis, commonly referred to in South India as *Kumatikkai* or the "bitter apple," is one such plant. It is a perennial herbaceous vine native to arid and semi-arid regions of Asia, Africa, and the Middle East, and now naturalized in parts of southern India [2]. The plant bears small, spherical fruits that closely resemble edible melons or cucumbers, such as *Cucumis melo var. agrestis* (locally known as *Mithukankai*). This morphological similarity, combined with the practice of collecting and consuming wild fruits, often results in accidental ingestion of the toxic *Kumatikkai* fruit, especially among children and villagers unfamiliar with its toxic potential. Historically, *Citrullus colocynthis* has been used in traditional medicine for its purgative, anti-inflammatory, and hypoglycemic properties [3-4]. The fruit pulp and seeds contain potent glycosides such as colocynthin, colocynthitin, and cucurbitacins, which are responsible for its pharmacological and toxic effects [5]. However, these compounds act as irritant purgatives on the gastrointestinal mucosa, inducing severe vomiting, colic, and diarrhea that may progress to dehydration, hematochezia, and circulatory collapse in severe cases [6-7].

Clinically, *Kumatikkai* poisoning manifests primarily as acute gastrointestinal irritation, with symptoms such as nausea, repeated vomiting, abdominal pain, and profuse watery or bloody diarrhea [8]. Imaging studies, including ultrasound or computed tomography, may show evidence of colitis or mucosal inflammation [9]. Fortunately, most cases are self-limiting with appropriate supportive management, including rehydration, proton pump inhibitors and antibiotics when necessary. The present case series reports seven cases of *Citrullus colocynthis* poisoning admitted to the Toxicology Ward of Government Rajaji Hospital, Madurai, between April and September 2025. These cases demonstrate the diverse clinical manifestations of *Kumatikkai* ingestion from mild hepatocellular dysfunction to hemorrhagic colitis and the effectiveness of supportive therapy. The objective of this report is to document the clinical spectrum of *Kumatikkai* poisoning, emphasize the diagnostic challenges it presents, and highlight the need for community-level preventive awareness regarding this toxic yet deceptively familiar plant.

Case Presentation

Case 1

A 42-year-old female from Madurai admitted in, Government Rajaji Hospital, with a clear history of ingestion of approximately two raw *Kumatikkai* fruits (Fig.1). The ingestion was accidental, as the patient mistook the fruit for an edible melon variant. About two hours following ingestion, she developed nausea, repeated non-bilious vomiting (approximately 8 episodes), and abdominal discomfort that gradually progressed to loose stools. Within 24 hours, she began to notice blood-stained stools, described as bright red, associated with abdominal cramping and tenesmus. There was no history of hematemesis, dizziness, syncope, or exposure to other toxins.

Fig: 1 *Citrullus colocynthis* (Kumatikkai)

The patient had no significant past medical history, no known comorbidities, and was not on any long-term medications. Menstrual history was normal with LMP on 14 April 2025. There was no history of chronic gastrointestinal disease, nor similar illness in the family.
On examination, the patient was conscious and well oriented, with stable vital parameters (pulse: 88/min, BP: 112/70 mmHg, temperature: afebrile). There was no pallor, icterus, or peripheral edema. Systemic examination was unremarkable except for the abdomen, which was soft with diffuse tenderness but without guarding or rigidity. Bowel sounds were hyperactive, and per-rectal examination showed no mass or polyp, with normal sphincter tone.
Laboratory investigations on admission revealed:

Parameter	**Admission**	**Day 1**	**Reference Range**
Total count (/µL)	10,600	—	4,000–11,000
Hemoglobin (g/dL)	13 → 10	—	12–15
Platelets (/µL)	3.56×10^5	—	$1.5–4.5 \times 10^5$
Urea (mg/dL)	22	—	10–40
Creatinine (mg/dL)	0.9	—	0.6–1.2
Na^+ / K^+ (mEq/L)	134 / 3.7	—	135–145 / 3.5–5.0
T. Bilirubin (mg/dL)	1.0	0.9	<1.2
SGOT / SGPT (U/L)	12 / 15	13 / 16	<40

Imaging:
- Ultrasound abdomen: Normal
- CECT abdomen: Features suggestive of colitis with minimal ascites

Diagnosis: *Citrullus colocynthis* (Kumatikkai) poisoning with acute colitis.
Treatment:
- IV fluids (normal saline)
- Inj. Piperacillin-tazobactam 4.5 g IV tds
- Inj. Pantoprazole 40 mg IV bd
- Inj. Tranexamic acid 500 mg IV tds
- Oral probiotics and ORS
- Soft oral diet

Outcome: The patient's symptoms resolved over four days, and she was discharged in stable condition.

Case 2
A 13-year-old female school student from Madurai was admitted after accidental ingestion of one whole *Kumatikkai* fruit while playing near her home garden. The child reportedly mistook it for an edible wild cucumber (*Mithukankai*). Within three hours, she developed nausea and two episodes of non-bilious vomiting, followed by mild abdominal discomfort but no diarrhea or hematochezia. There was no loss of consciousness, dizziness, or history of intake of any medication before admission. On examination, she was alert, afebrile, and vitals were stable (BP: 100/60 mmHg; HR: 90/min). There was no pallor, icterus, or signs of dehydration. Abdominal examination revealed mild diffuse tenderness, and other systemic examinations were normal.
Laboratory Parameters:

Parameter	**Day 1**	**Day 2**	**Day 3**	**Day 4**
Total count (/mm³)	8,900	6,800	—	—
RBC (/mm³)	5.16	4.31	—	—
Hb (g/dL)	10.1	11.4	—	—
Platelets (/mm³)	279,000	387,000	—	—
Urea (mg/dL)	23	28	21	14
Creatinine (mg/dL)	0.8	0.8	0.6	0.7
Total Bilirubin (mg/dL)	1.4	1.6	2.2	1.1
Direct Bilirubin (mg/dL)	—	0.6	0.2	—

Indirect Bilirubin (mg/dL)	—	1.6	0.9	—
SGOT (U/L)	31	20	19	18
SGPT (U/L)	33	22	18	16

Imaging:
- ECG and ultrasound abdomen: Normal
- Serum electrolytes: Within normal limits

Treatment:
- Antiemetics
- Oral Ursodeoxycholic acid (UDCA) 300 mg daily × 3 days
- Oral rehydration and dietary support

Outcome: The bilirubin levels normalized by Day 4, and the patient was discharged on 15 September 2025.

Case 3

A 16-year-old adolescent girl was brought to the Emergency Department following the intentional ingestion of approximately half a Kumatikkai fruit with the intention of deliberate self-harm. She presented four hours after ingestion with complaints of nausea, a single episode of vomiting, and mild upper abdominal pain. She denied consuming any other substances. On arrival, she was alert, oriented, and hemodynamically stable (BP: 108/68 mmHg, HR: 84/min). There was no pallor or icterus. Systemic examination was normal except for mild right upper quadrant tenderness. No signs of bleeding were observed.

Laboratory investigation:

Parameter	**Day 1**	**Day 2**	**Day 3**	**Day 4**
Total count (/mm³)	6,200	6,900	—	—
RBC (/mm³)	5.24	5.22	—	—
Hb (g/dL)	11.1	11.6	—	—
Platelets (/mm³)	346,000	346,000	—	—
Urea (mg/dL)	25	26	21	22
Creatinine (mg/dL)	0.9	0.8	0.92	0.8
Total Bilirubin (mg/dL)	1.9	2.6	4.0	2.1
Direct Bilirubin (mg/dL)	—	0.3	0.7	—
Indirect Bilirubin (mg/dL)	—	3.7	1.4	—
SGOT (U/L)	54	21	19	11
SGPT (U/L)	43	21	21	11

Imaging: Ultrasound, ECG, and electrolytes were within normal limits.

Treatment:
- IV fluids and antiemetics
- Tab. UDCA 300 mg for 3 days
- Observation for hepatic recovery

Outcome: Bilirubin levels declined to near-normal values, and the patient was discharged.

Case 4

A 23-year-old female presented to the at Government Rajaji Hospital, Madurai, with a history of accidental ingestion of one raw Kumattikkai (*Citrullus colocynthis*) fruit. She subsequently developed multiple episodes of vomiting (4–5, non-bilious, non-blood-stained) and 8–10 episodes of loose stools, which later became blood-stained, accompanied by diffuse abdominal pain. She denied any other systemic symptoms. There was no significant past medical history, comorbid illness, or drug intake. Her menstrual cycles were regular. On arrival, the patient was conscious, oriented, and hemodynamically stable (BP: 110/70 mmHg). There was no pallor, icterus, or dehydration. Systemic examination was normal except for diffuse abdominal tenderness. Per rectal examination showed normal sphincter tone and no palpable lesion.

Laboratory Parameters:

Parameter	Day 1
Total count (/µL)	10,600
Hb (g/dL)	15 → 12
Platelets (/µL)	2,56,000
Urea (mg/dL)	26
Creatinine (mg/dL)	0.7
Na^+/K^+ (mEq/L)	138/3.8
Viral markers	Negative

Imaging:

- CECT Abdomen & Pelvis: Findings suggestive of colitis with minimal ascites
- Ultrasound Abdomen: Normal
- ECG and Echocardiogram: Normal

Treatment:

• IV fluids and soft oral diet
• Inj. Piperacillin–Tazobactam 4.5 g IV tds
• Inj. Pantoprazole 40 mg IV bd
• Inj. Tranexamic acid 500 mg IV tds
• Probiotics and oral rehydration solution

Outcome:

The patient improved symptomatically, tolerated oral intake, and was discharged in stable condition after complete recovery.

Case 5

A 15-year-old school boy was referred from Sivagangai Medical College to Government Rajaji Hospital, Madurai, with a history of accidental ingestion of approximately half a Kumattikkai (*Citrullus colocynthis*) fruit. The ingestion was unintentional. He presented with complaints of nausea but did not experience vomiting, diarrhea, or abdominal pain. On arrival, he was alert, oriented, and hemodynamically stable (BP: 112/70 mmHg, HR: 82/min). There was no pallor or icterus, and systemic examination was unremarkable.

Laboratory Parameters:

Parameter	**Day 1**	**Day 2**	**Day 3**
Total count (/µL)	7,200	—	—
Hb (g/dL)	12.8	—	—
Platelets (/µL)	3,38,000	—	—
Urea (mg/dL)	26	24	22
Creatinine (mg/dL)	0.8	0.8	0.7
Total Bilirubin (mg/dL)	2.1	1.6	1.1
SGOT (U/L)	34	22	17
SGPT (U/L)	31	20	15

Imaging:

• Ultrasound Abdomen and Pelvis: Normal
• ECG and Serum Electrolytes: Normal

Treatment:
•IV fluids and antiemetics
• Tab. Ursodeoxycholic Acid (UDCA) 300 mg once daily for 3 days
• Observation for hepatic function recovery
Outcome:
The patient remained asymptomatic, and liver parameters normalized by Day 3. He was discharged in stable condition.

Case 6
A 15-year-old male, a classmate of Case 2, was admitted on the same day with a history of accidental ingestion of approximately half a Kumattikkai (*Citrullus colocynthis*) fruit. He presented with a single episode of vomiting and mild nausea, but denied any diarrhea, abdominal pain, or bleeding manifestations. On examination, the patient was conscious, oriented, and hemodynamically stable (BP: 110/72 mmHg, HR: 84/min). There was no pallor, icterus, or signs of dehydration. Systemic examination was unremarkable.
Laboratory Parameters:

Parameter	Day 1	Day 2	Day 3
Total count (/µL)	7,400	—	—
Hb (g/dL)	12.5	—	—
Platelets (/µL)	3,40,000	—	—
Urea (mg/dL)	25	23	21
Creatinine (mg/dL)	0.8	0.8	0.7
Total Bilirubin (mg/dL)	2.3	1.7	1.0
SGOT (U/L)	35	23	18
SGPT (U/L)	30	21	15

Imaging:
- Ultrasound Abdomen and Pelvis: Normal
- ECG and Electrolytes: Normal

Treatment:
• IV fluids and antiemetics
• Tab. Ursodeoxycholic Acid (UDCA) 300 mg once daily for 3 days
• Observation for hepatic function recovery
Outcome:
Liver function improved over the next three days, and the patient was discharged in stable condition.

Case 7
A 15-year-old male was admitted in the Government Rajaji Hospital, Madurai, following the intentional ingestion of a Bitter Apple (*Citrullus colocynthis*) fruit at approximately 11:20 a.m., as an act of deliberate self-harm. At presentation, the patient was conscious and oriented. Vital signs revealed mild hemodynamic instability with BP: 100/60 mmHg, Pulse Rate: 86/min, and SpO_2: 92% on room air. He complained of nausea and headache but denied vomiting, abdominal pain, or bleeding. On physical examination, bilateral air entry was equal, and bowel sounds were soft. There was no pallor, icterus, or organomegaly. Cardiovascular and neurological examinations were unremarkable. An ECG performed on the day of admission showed sinus arrhythmia with borderline changes. Given the deliberate ingestion, a psychiatric consultation was promptly sought.
Laboratory Investigations: Initial investigations, including coagulation and liver function tests, were within normal limits, indicating no significant systemic toxicity at the time of evaluation:

Parameter	Result	Reference Range
Prothrombin Time (PT)	11.2 sec	(Control: 11 sec)
APTT	28.5 sec (Control: 28.2 sec)	—
Total Bilirubin	0.5 mg/dL	—

SGOT (AST)	19 IU/L	—
SGPT (ALT)	10 IU/L	—

A CECT Abdomen and Pelvis performed on 27 July 2025 revealed no evidence of free fluid or internal organ pathology. The liver, pancreas, spleen, and kidneys were reported as normal in size and echo texture.

Treatment:

• Intravenous fluids (Normal Saline at 15 mL/hour)
• Inj. Ondansetron (SOS)
• Inj. Pantoprazole 40 mg IV bd
• Inj. Vitamin B complex
• Supportive monitoring and psychiatric evaluation

Outcome:

The patient's clinical condition remained stable and improved over the hospital stay. He was discharged four days later, in a conscious and oriented state, with a documented stable condition.

Discharge advice included taking:

- Tab. Ranitidine 150 mg (1-0-1 for 5 days).
- Cap. Preprobiotic (1-0-1 for 5 days).
- The patient was advised to follow up in the Outpatient Department (OPD) with new lab parameters.

Case No.	Age / Sex	Place of Admission	Type of Exposure	Amount Ingested	Clinical Presentation	Key Laboratory / Imaging Findings	Treatment Given	Outcome
1	42 / F	GRH, Madurai –	Accidental (mistaken for melon)	~2 whole fruits	Nausea, repeated vomiting, abdominal pain, loose stools, hematochezia	Hb: 13→10 g/dL; CECT: colitis with minimal ascites; normal LFTs	IV fluids, Piperacillin–Tazobactam, Pantoprazole, Tranexamic acid, probiotics, ORS	Recovered; discharged Day 5
2	13 / F	GRH, Madurai –	Accidental (mistaken identity while playing)	1 whole fruit	Nausea, vomiting ×2, mild abdominal discomfort	Total bilirubin peaked 2.2 mg/dL; normal ECG, USG	Antiemetics, UDCA 300 mg × 3 days, ORS, dietary support	Recovered; discharged Day 4
3	16 / F	GRH, Madurai –	Intention of deliberate self-harm	~½ fruit	Nausea, vomiting ×1, upper abdominal pain	Total bilirubin peaked 4.0 mg/dL; SGOT 54→11; normal USG, ECG	IV fluids, antiemetics, UDCA 300 mg × 3 days, observation	Recovered; discharged Day 3
4	23 / F	GRH, Madurai –	Accidental ingestion	1 whole fruit	Vomiting, diarrhea, hematochezia, diffuse abdominal pain	Hb: 15→12 g/dL; CECT: colitis + ascites; normal ECG	IV fluids, Piperacillin–Tazobactam, Pantoprazole, Tranexamic acid, probiotics, ORS	Recovered; discharged stable
5	15 / M	GRH, Madurai –	Accidental (schoolboy)	~½ fruit	Nausea only	Bilirubin 2.1→1.1 mg/dL; normal ECG, USG	IV fluids, antiemetics, UDCA 300 mg × 3 days	Recovered; discharged Day 3
6	15 / M	GRH, Madurai –	Accidental (classmate of Case 5)	~½ fruit	Vomiting ×1, mild nausea	Bilirubin 2.3→1.0 mg/dL;	IV fluids, antiemetics, UDCA 300 mg × 3 days	Recovered; discharged Day 3

						normal ECG, USG		
7	15 / M	GRH, Madurai –	Intentional (self-harm)	1 fruit (approx.)	Nausea, headache, mild hemodynamic instability	PT 11.2 s, INR 0.88; SGOT 19, SGPT 10; normal CECT abdomen	IV fluids, Ondansetron, Pantoprazole, Vit B complex, psychiatric evaluation	Recovered; discharged Day 4; psychiatric follow-up advised

Discussion

Citrullus colocynthis, commonly known as *Kumatikkai*, is a xerophytic perennial plant of the Cucurbitaceae family, traditionally found in arid and semi-arid regions. Its fruit, resembling a small melon with green and yellow mottling, is frequently mistaken for the edible *Mithukankai*, leading to accidental ingestions. Diagnosis of *C. colocynthis* poisoning is primarily clinical and relies heavily on a thorough dietary history. Patients often report the characteristic bitter taste of the fruit, followed by rapid onset of gastrointestinal symptoms. Laboratory investigations may reveal mild anemia, elevated liver enzymes, and hyperbilirubinemia, while imaging studies such as contrast-enhanced CT can demonstrate features of colitis and minimal ascites. These findings, in conjunction with a compatible history, are usually sufficient to establish the diagnosis and differentiate it from other acute gastrointestinal conditions. There is currently no specific antidote for *C. colocynthis* toxicity, and treatment remains entirely supportive. Early gastrointestinal decontamination may be beneficial in selected patients presenting soon after ingestion. Rehydration with intravenous fluids is critical to counteract fluid losses and maintain hemodynamic stability. Proton pump inhibitors are used to reduce mucosal injury, while broad-spectrum antibiotics may be indicated in cases with clinical or radiological evidence of colitis. Hepatoprotective agents such as ursodeoxycholic acid (UDCA) can support liver function, and tranexamic acid may be used to control gastrointestinal bleeding when present. In this series, all patients responded well to these interventions, with full clinical and biochemical recovery, highlighting the generally favorable prognosis with prompt and appropriate care.

The clinical manifestations observed in this case series align closely with previously documented reports of *Citrullus colocynthis* toxicity. Notably, Case 1 and 4 in our series mirrors the presentation described by Espinosa et al. [10], where a pediatric patient developed hematochezia and colitis following accidental ingestion of bitter apple fruit, requiring supportive management and close monitoring for gastrointestinal bleeding. Similarly, our adult patient developed hemorrhagic colitis within 24 hours of ingesting two raw fruits, with CECT imaging confirming mucosal inflammation and minimal ascites. The similarity in symptom onset and bleeding profile reinforces the irritant and cytotoxic role of colocynthin, a potent glycoside known to cause gastrointestinal mucosal damage and fluid–electrolyte imbalance. In both cases, recovery was complete with conservative therapy, emphasizing the importance of early recognition and prompt supportive management in preventing complications. In contrast, Cases 2 and 3 exhibited milder gastrointestinal symptoms but demonstrated transient hepatic dysfunction, characterized by elevated serum bilirubin and mild transaminase derangement. This pattern partially parallels the findings of Rezvani et al. [11], where hepatic involvement occurred following ingestion of a colocynth decoction; however, their patient developed hypotension and multisystem features indicative of more severe toxicity. The hepatic stress in our patients is likely attributable to cucurbitacins and related triterpenoid compounds, which undergo hepatic metabolism and can induce reversible hepatocellular injury. Of particular note, the adolescent in Case 3, who intentionally ingested half a fruit, exhibited the highest bilirubin level (4.0 mg/dL) in our cohort, supporting a dose–response relationship between the quantity ingested and hepatic involvement. Although liver enzymes were only modestly elevated, this pattern underscores that hepatic toxicity may be an underrecognized aspect of colocynth poisoning, especially in non-hemorrhagic presentations.

Cases 5 and 6, both involving school-aged boys with accidental half-fruit ingestion, showed only mild hyperbilirubinemia without systemic features, highlighting the self-limiting nature of low-dose

exposure. Conversely, Case 7, an intentional ingestion by a 15-year-old male, presented with nausea, headache, and transient hemodynamic instability but no biochemical abnormalities, further illustrating the wide individual variability in clinical response despite similar exposure.

Our findings also remain consistent with the more severe toxicity profile reported by Alemseged et al. [6], who documented a case of multiorgan dysfunction—including gastrointestinal hemorrhage, hepatotoxicity, and acute kidney injury following colocynth ingestion in an adult. None of our patients progressed to renal or cardiovascular compromise; however, these reports collectively suggest that systemic toxicity may develop with larger doses, delayed presentation, or concomitant dehydration. Importantly, all seven patients in our series recovered completely with supportive therapy, which included intravenous fluids, proton pump inhibitors, antibiotics where indicated, and ursodeoxycholic acid for hepatic protection. The absence of mortality, despite varied clinical presentations, highlights the effectiveness of early hospital-based management and the typically reversible nature of colocynth-induced organ injury. Public health measures must prioritize strategies that prevent accidental ingestion of *Citrullus colocynthis* by raising awareness and improving identification at the community level. A key step is differentiating toxic *Kumatikkai* from similar-looking edible gourds, which can be achieved through the use of illustrated posters, community education drives, and integration into agricultural extension programs. Additionally, traditional healers and practitioners should be specifically educated about the dangers of using unprocessed *C. colocynthis* pulp in herbal remedies, as even small amounts can be toxic. To enhance surveillance and response, the establishment of regional toxic plant registries is also essential. These registries would support the early identification, documentation, and reporting of plant-related poisoning cases, thereby aiding both clinical management and public health interventions.

Conclusion

This case series emphasizes that *Kumatikkai* (*Citrullus colocynthis*) poisoning can manifest with a wide clinical spectrum**,** from mild nausea to hemorrhagic colitis and hepatic dysfunction. Although recovery is usually complete with supportive therapy, early recognition and hydration management are critical to prevent complications. Given its similarity to benign cucurbits, *Kumatikkai* poisoning is likely underreported. Increased clinical vigilance and awareness can prevent avoidable morbidity.

Conflict of interest

The authors report no conflicts of interest in this work.

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