Colchicine toxicity: Accidental Plant Poisoning with Colchicum Autumnale Resulting in Multiorgan Failure: A Case Report

Maryam IH Hanna [1*], Halder J Abozait [2]
[1]Azadi Teaching Hospital, Duhok, Kurdistan Region, Iraq
[2] Department of Internal Medicine, Mount Sinai Hospital, Chicago, IL, USA

* Corresponding Author: **Maryam IH Hanna**

Article Info

ISSN (online): 2582-8940
Volume: 07
Issue: 03
Received: 03-06-2026
Accepted: 02-07-2026
Published: 01-08-2026
Page No: 156-159

Abstract
Colchicine has a narrow therapeutic index, with no clear cut-off toxic dose and no age or gender predilection. Beyond its approved use for gout and familial Mediterranean fever, it occurs naturally in Colchicum autumnale (autumn crocus), a plant sometimes mistaken for wild garlic. Acute toxicity progresses through three phases: an early gastrointestinal phase within 24 hours, a peak phase between days 1–7 marked by metabolic acidosis, shock, myelosuppression, and multi-organ dysfunction, and a third phase from days 7–21 ending in either recovery or death. No specific antidote exists, and treatment relies on gastric decontamination, activated charcoal, and supportive care, with hemodialysis notably ineffective. We report a 35-year-old man who developed severe colchicine toxicity after accidentally ingesting autumn crocus mistaken for wild garlic, presenting with multi-organ involvement including acute kidney injury, coagulopathy, and severe thrombocytopenia. He was managed supportively and made a full recovery. The case reinforces the importance of considering plant-derived colchicine poisoning in patients with unexplained multi-organ failure.

DOI: https://doi.org/10.54660/IJMBHR.2026.7.3.156-159

Keywords: Colchicine toxicity, Plant poisoning, Multi-organ failure, Acute kidney injury, Supportive care

Introduction

Colchicine is a well-known drug used for its anti-inflammatory effect. It is FDA-approved for the treatment and prevention of acute gout flares, and for familial Mediterranean fever in patients 4 years and older. It has also been widely used off-label across oncology, immunology, cardiology, and dermatology [1]. Colchicine is an ancient drug, first mentioned as a plant extract in the Ebers Papyrus (an ancient Egyptian medical text, 1550 BCE), where it was used to reduce pain and swelling despite being well known as a poison. Despite this long history, the US Food and Drug Administration only approved it in 2009, after years of formal research. Colchicine is an alkaloid extracted mainly from the plant Colchicum autumnale (Autumn crocus) (Figure 1) [2, 3]. All parts of which contain colchicine, most heavily concentrated in the seeds and corms [4]. It remains the main active ingredient in the pharmaceutical drug. However, colchicine has a narrow therapeutic index, and the precise toxic dose remains unknown. A topic that becomes clinically important given how widely and casually the plant has historically been used.

Fig 1: Colchicum autumnale, commonly known as autumn crocus [4].

Case report

A previously healthy 35-year-old man with no significant past medical history. Accidentally ingested autumn crocus after mistaking it for wild garlic (Allium ursinum). Several hours later, he developed repeated episodes of vomiting. He did not seek medical advice until 2 days later, by which time this was also associated with frequent bowel motions and severe headache.

On admission, hypotension was noted, and he was initially treated as a case of gastroenteritis. Early on the second day, he developed abdominal pain and distention. His vital signs were: blood pressure 100/65 mmHg, pulse 82 bpm, oxygen saturation 97%, forehead temperature 36.7°C, and Glasgow Coma Scale 15. His urine bag showed 1100 cc of dark, concentrated urine. His abdomen was distended and tender. Laboratory investigations showed increased serum creatinine (1.43 mg/dL; RR: 0.6-1.2), elevated CRP (96 mg/L; RR: <6), deranged liver function tests, elevated D-dimer (11,817ng/mL; RR: <500), and isolated thrombocytopenia (platelet count 82 x 10*9/L; RR: 150-400 x 10*9/L).

An erect abdominal X-ray showed dilated bowel loops (Figure 2). Abdominal ultrasound showed multiple dilated bowel loops throughout the abdomen, with no other remarkable findings.

Paralytic ileus was assumed to be the cause of the intestinal obstruction as a result of the deranged metabolic state.

The patient was referred to the high dependency unit. Poisoning was suspected given the history of plant ingestion, but no specific toxin could be identified due to lack of test availability. As no specific antidote existed, management consisted primarily of supportive care, focused on hemodynamic stabilization, correction of electrolyte disturbances, symptomatic relief, and close monitoring for complications. The patient received 5 units of platelet transfusion to treat his isolated thrombocytopenia.

On day 3 of admission, he became dyspneic; respiratory examination revealed severe coarse crepitations over the right lung, predominantly at the right lower lung field. He developed pancytopenia, with his WBC count dropping to 4.5 x 10*9/L. A chest X-ray (Figure 3) was suggestive of pneumonia. The patient was in a state of multi-organ failure; notably, no intubation or vasopressor therapy was required. Cardiac evaluation with ECG, echocardiography, and serial troponin was unremarkable. Broad-spectrum antibiotics were started, comprising meropenem, metronidazole, and clindamycin, alongside aggressive fluid resuscitation and continued supportive therapy. Close monitoring of laboratory parameters was maintained (Table 1).

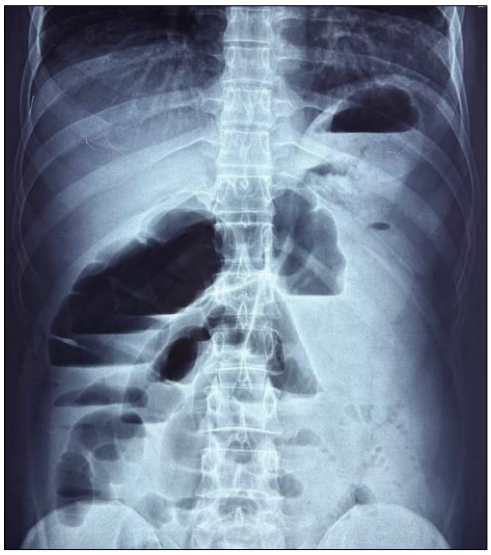

Fig 2: Erect abdominal X-ray.

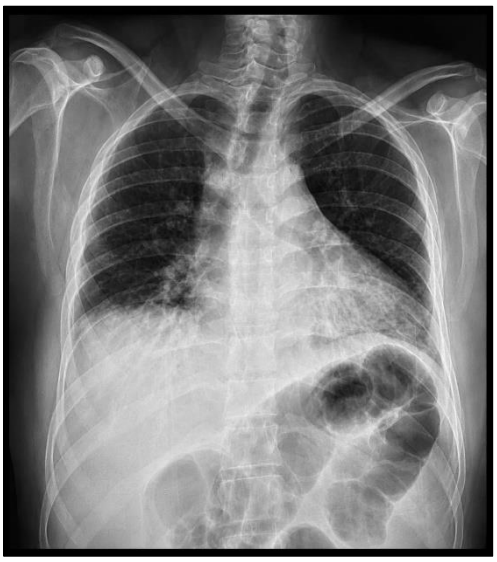

Fig 3: Chest X-ray.

Table 1: Laboratory values of the patient since admission

Test	Day 1	Day 2	Day 3	Day 4	Day 5	Reference Ranges
WBC Count	14.9	6.9	4.5	0.68	0.59	4-10 x 10*9/L
PLT Count	82	12	22	54	29	150-400 x 10*9/L
Urea nitrogen	118	123	76	68	65	10-50 mg/dL
Creatinine	1.43	1.42	0.82	1.3	0.87	0.6-1.2 mg/dL
Sodium	145	140	146	143	148	135–145 mmol/L
Potassium	4.30	3.41	3.39	3.29	3.27	5–5.0 mmol/L
AST	244	244	428	467	149	0.01-40 U/L
ALT	61	58	168	284	188	0.01-40 U/L
ALP	441	269	110	124	102	30-120 U/L
CRP	96	185	62	35	41	<6 mg/L

Abbreviations: WBC, white blood cell; PLT, platelet; AST, aspartate aminotransferase; ALT, alanine aminotransferase; ALP, alkaline phosphatase; CRP, C-reactive protein.

By day 7 of admission, his clinical condition had returned almost to baseline, with resolution of myelosuppression and normalization of liver and renal function, inflammatory markers, and coagulation profile, corresponding to day 9 post-ingestion. He was discharged to the medical ward, with close observation continued for a further week following full normalization. Overall, the patient progressed through the classical phases of colchicine toxicity, with full recovery to his baseline state.

Discussion

Microtubules — polymerized heterodimers of α- and β-tubulin — form a core part of the cytoskeleton, serving several fundamental cellular functions, including maintenance of cell shape, intracellular trafficking, mitotic spindle formation, formation of cilia and flagella, and positioning of organelles within the cell. Colchicine exerts its effect by binding to β-tubulin, disrupting microtubule polymerization. These mechanisms underlie both the therapeutic and toxic effects of colchicine. At low concentrations, colchicine prevents microtubule growth, and at higher concentrations, it may promote depolymerization [5]. Fundamentally, direct inhibition of neutrophil migration is considered the main mechanism of action on the immune system, though other independent anti-inflammatory effects are also seen.

There is no clear evidence of a cut-off toxic level, with no age or gender predilection [6]. Clinical features of acute toxicity typically progress through three phases. Phase 1 (within 24 hours post-ingestion) presents with vomiting, abdominal pain, and diarrhea. Phase 2 (1 to 7 days) represents peak toxicity, characterized by metabolic acidosis, shock, myelosuppression, rhabdomyolysis, and multi-organ dysfunction involving the kidneys, liver, and respiratory system. Phase 3 (7 to 21 days) is characterized by either recovery or death. These phases may overlap and can be nonspecific [7].

Acute colchicine toxicity carries a high mortality rate. Treatment focuses on ameliorating the toxic effects of colchicine. Management should begin with gastric lavage, ideally performed within the first 1–2 hours post-ingestion. Activated charcoal is another effective method, particularly when administered within the first hour in cases of high dose ingestion. Evidence has shown that 5 g of activated charcoal can effectively bind up to 90% of approximately 10 mg of colchicine [8]. Diuresis has also proven effective in eliminating the drug, as the kidneys contribute to approximately 20% of drug elimination. However, hemodialysis was, somewhat surprisingly, not effective in treatment [9]. Fluid resuscitation is fundamental, with treatment aimed at replacing gastrointestinal fluid losses adequately to maintain renal perfusion.

The exact volume of fluid required is not yet clearly established; however, the goal is to restore fluid losses and maintain adequate tissue perfusion.

Vital signs should be monitored regularly to prevent complications such as pulmonary edema, hypothermia, acid-base imbalance, hyperkalemia, hypocalcemia, and coagulopathy [8]. Bone marrow suppression is a fatal complication of acute colchicine toxicity. Prophylactic platelet transfusion is indicated when the platelet count falls below 20×10^9/L to prevent spontaneous bleeding as seen in our case [10]. Leukopenia and immune dysfunction can be treated with recombinant human granulocyte-colony stimulating factor (G-CSF), and prophylactic antimicrobial therapy — in the form of antibiotics and antifungals — may be used when appropriate [8].

As no specific antidote is currently available, some studies have investigated colchicine-specific antigen-binding fragments (Fabs); however, these have not yet been clinically validated in current guidelines.

Conclusion

Colchicine toxicity from plant misidentification is rare but potentially fatal, and should be considered in unexplained multi-organ failure. With no specific antidote available, early recognition and supportive care remain the cornerstone of management, as illustrated by this patient's full recovery.

References

1. Dasgeb B, Kornreich D, McGuinn K, Okon L, Brownell I, Sackett DL. Colchicine: an ancient drug with novel applications. Br J Dermatol. 2018;178(2):350-356.
2. National Institute of Diabetes and Digestive and Kidney Diseases. LiverTox: Clinical and Research Information on Drug-Induced Liver Injury [Internet]. Bethesda (MD): National Institute of Diabetes and Digestive and Kidney Diseases; 2012-. Colchicine. Updated 2017 Oct 26.
3. Evans WC. Trease and Evans' Pharmacognosy. 16th ed. Edinburgh: Saunders/Elsevier; 2009.
4. Gardenia. Colchicum (Autumn Crocus) [Internet]. Available from: https://www.gardenia.net/genus/colchicum-autumn-crocus
5. Leung YY, Yao Hui LL, Kraus VB. Colchicine—update on mechanisms of action and therapeutic uses. Semin Arthritis Rheum. 2015;45(3):341-350.
6. Sadiq NM, Robinson KJ, Terrell JM. Colchicine. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026-. Updated 2025 Jan 19.
7. Parekh S, Merry TJ, Tripathi V, Reddy P. A case of colchicine overdose and toxicity. Cureus. 2025;17(6).
8. Wu J, Liu Z. Progress in the management of acute colchicine poisoning in adults. Intern Emerg Med. 2022;17(7):2069-2081.
9. Sadiq NM, Robinson KJ, Terrell JM. Colchicine. StatPearls.
10. Yuan S, Otrock ZK. Platelet transfusion: an update on indications and guidelines. Clin Lab Med.

How to Cite This Article

Hanna MIH, Abozait HJ. Colchicine toxicity: accidental plant poisoning with *Colchicum autumnale* resulting in multiorgan failure: a case report. Int J Med All Body Health Res. 2026;7(3):156-159.
doi:10.54660/IJMBHR.2026.7.3.156-159.