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Combination of Multiple-Dose Activated Charcoal and Infusion Therapy That Led to Favorable Outcomes in Colchicine Poisoning: A Case Report

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Patient: Male, 20-year-old
Final Diagnosis: Colchicine poisoning
Symptoms: Diarrhea • drowsiness • dyspnea • nausea
Clinical Procedure: —
Specialty: Toxicology
Objective: Unknown etiology

Background: The therapeutic margin of colchicine is narrow, and toxicity occurs easily. No standard treatment exists because its toxicokinetics are poorly understood. Symptoms are usually gastrointestinal, and dehydration occurs easily. Few reports have monitored serum and urinary colchicine levels.

Case Report: A man in his 20s presented with nausea, vomiting, diarrhea, drowsiness, and dyspnea. He ingested 2 dried *Colchicum autumnale* bulbs (estimated colchicine: 6.24-15.6 mg) with an energy drink. Activated charcoal with laxative was administered at 27 hours post ingestion (h-PI), followed by multiple-dose activated charcoal (MDAC) every 6 hours (13 doses, 27-101 h-PI), high-volume fluid infusion (Ringer's acetate), and blood purification (hemodialysis [HD] at 48-52 h-PI; hemodiafiltration [HDF] at 71-75 and 98-102 h-PI). Serum colchicine was measured at 16 time points and urine at 11 time points. Serum colchicine at 27 h-PI was 50.3 ng/mL, and was temporally associated with a rapid decrease to 21.2 ng/mL at 35 h-PI, coinciding with initiation of infusion and activated charcoal. A secondary rise to 17.74 ng/mL occurred at 77 h-PI, approximately 2.4 hours after HDF1 completion. Urine colchicine was 110.0 ng/mL at 29 h-PI, then gradually decreased. Serum colchicine changed modestly (12.16 to 5.46 ng/mL) during HD.

Conclusions: In this case of severe colchicine poisoning, serial serum and urine concentration monitoring provided a time-resolved profile across concurrent interventions. The observed temporal associations support hypothesis generation regarding the potential roles of renal elimination and MDAC-mediated interruption of enterohepatic recirculation in colchicine clearance, while recognizing that concurrent therapies preclude attribution of effects to any single intervention.

Keywords: Charcoal • Colchicine
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Introduction

Colchicine is a lipophilic alkaloid used to treat gout flares. Its therapeutic margin is narrow, and peak plasma concentrations (Cmax) are reported to be 6.50 ± 1.03 ng/mL at 1.07 ± 0.55 hours (Tmax) after oral administration [1]. Toxicity occurs easily without the ingestion of markedly high doses. *Colchicum autumnale*, a lily plant, is widely available as an ornamental plant and has been associated with colchicine poisoning, which can lead to multiple organ failure and other serious complications [2,3]. After oral administration, colchicine is readily absorbed and undergoes extensive first-pass metabolism. It is primarily metabolized by the liver, undergoes significant enterohepatic recirculation, and is excreted by the kidneys [4]. Ferron et al detected a secondary peak within 6 hours of oral administration, indicating enterohepatic recirculation [1]. Colchicine-specific Fab fragments can be effective in treating colchicine poisoning; however, they are not readily available. No standard treatment is available, and patients are treated symptomatically [5,6]. Because colchicine undergoes enterohepatic recirculation, multiple-dose activated charcoal (MDAC) reportedly eliminates it. Renal excretion may also be important in patients with extremely high serum colchicine levels. However, few reports have simultaneously monitored serial serum and urine colchicine concentrations alongside a detailed treatment timeline, limiting understanding of the relative contributions of these pathways.

Case Report

A man in his 20s was transferred to the emergency department (ED) with nausea, vomiting, diarrhea, drowsiness, and dyspnea. He had previously been treated for autism spectrum disorder and attention-deficit hyperactivity disorder but was taking no home medications at the time of admission. This was the patient's first episode of intentional overdose; no prior history of self-poisoning was documented. On arrival at the ED, he reported ingesting 2 dried *Colchicum autumnale* bulbs, with an estimated colchicine content of 6.24 to 15.6 mg, based on published colchicine concentrations of 0.08% to 0.2% by dry weight [7]. The actual bulb weight was not measured, and plant identification relied on the patient's verbal report alone. The bulbs were ingested with an energy drink of an unknown type and volume at 27 hours prior to admission (27 hours post ingestion [h-PI]). The patient's body weight was 56 kg (estimated intake: 0.111-0.279 mg/kg). Doses above 0.5 mg/kg have been associated with severe toxicity, and doses of approximately 0.8 mg/kg or greater have been reported to carry a high risk of fatal outcome, although considerable interindividual variability exists [4,8]. He was conscious (Glasgow Coma Scale: E4V5M6) and hemodynamically stable, with a blood pressure of 150/79 mm Hg, heart rate of 90 beats/min, respiratory rate of 20 breaths/min, body temperature of 36.7 °C, and SpO_2 of 99% in room air. Physical examination revealed facial flushing and ocular conjunctival hyperemia. Chest radiography, electrocardiography, and echocardiography revealed no abnormalities. Blood alcohol screening was performed on admission (result: below detectable limit); no multi-drug toxicology screen was performed. No significant irregularities were observed in the initial laboratory values (**Table 1**). Serial laboratory data including complete blood count, coagulation, hepatic function, lactate, renal function, and electrolytes across 7 time points are shown in **Table 1**. All values remained within normal limits throughout hospitalization or showed only minor transient fluctuations without clinical significance. Activated charcoal (1 g/kg) and sodium picosulphate hydrate (75 mg) were administered via nasogastric tube. The patient was admitted to the intensive care unit (ICU). MDAC (activated charcoal 0.5 g/kg every 6 h, 13 doses total) and blood purification therapy, consisting of 1 session of hemodialysis (HD; cellulose triacetate 0.9 m^2, blood flow rate [QB] 100 mL/min, dialysate flow rate [QD] 500 mL/min, 4.2 h) and 2 sessions of hemodiafiltration (HDF; polysulfone 1.5 m^2, QB 100 mL/min, QD 500 mL/min, 3.75 h), were administered for toxin removal. The 13-dose MDAC regimen was planned from admission based on clinical severity and the expected duration of enterohepatic recirculation. Serum colchicine concentrations were measured retrospectively by liquid chromatography–tandem mass spectrometry (LC-MS/MS) and were not available to the treating team during hospitalization. Serum and urine colchicine levels were measured using a commercially available drug and toxicant rapid screening system based on LC-MS/MS (Shimadzu Corporation, Kyoto, Japan) at the Division of Toxicology, Department of Pharmacobiology, Showa University Graduate School of Pharmacy. A case-specific analytical validation was not performed; the assay was not independently validated for the purposes of this report. Fluid resuscitation with Ringer's acetate was initiated at approximately 270 mL/h and subsequently titrated (60-200 mL/h) based on clinical assessment and fluid balance (**Table 2**). The complete intervention and monitoring timeline is presented in **Table 3**. Activated charcoal passage in stool was confirmed visually at approximately 32, 43, 55, and 70 h-PI. Safety during MDAC administration was monitored daily by clinical assessment, serial blood tests (complete blood count, renal function, electrolytes, and hepatic function), and chest and abdominal radiography. No adverse events related to MDAC, including bowel obstruction or electrolyte disturbances, were observed. Two days after ingestion, electrocardiography revealed slight QT prolongation (QTc, 451 ms) and sporadic premature ventricular contractions; however, no other specific symptoms were noted.

The serum colchicine level (**Figure 1**) was 50.3 ng/mL at 27 h-PI (on arrival). It decreased to 21.2 ng/mL at 35 h-PI, temporally associated with initiation of fluid infusion and activated

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Table 1. Laboratory data on admission and serial values during hospitalization (h-PI = hours post ingestion).

Parameter	Admission (27 h-PI)	Day 2 6: 00 AM	Day 2 6: 00 PM	Day 3 6: 00 AM	Day 3 6: 00 PM	Day 4 6: 00 AM	Day 5 6: 00 AM
WBC (/μL)	10 800	13 900	12 100	14 000	14 400	13 100	11 300
Hb (g/dL)	15.4	14.8	13.8	14.1	14.5	14.6	14.4
Plt (×10⁴/μL)	31.7	30.9	27.7	26.6	28.3	27.9	26.2
PT (%)	90	86	79	88	90	86	91
APTT (s)	30.1	29.2	41.0	29.4	32.3	32.5	31.0
Fibrinogen (mg/dL)	230	—	—	289	356	425	444
D-dimer (μg/mL)	0.50	0.47	0.59	0.56	0.74	0.92	1.03
T-Bil (mg/dL)	1.1	1.1	1.1	1.2	1.1	0.8	0.7
AST (U/L)	24	20	16	16	17	14	15
ALT (U/L)	22	18	16	16	15	14	16
LDH (U/L)	184	145	130	138	175	145	120
Lactate (mmol/L)	0.87	0.73	0.78	0.65	0.77	0.63	0.67
Creatinine (mg/dL)	0.58	0.65	—	0.53	—	0.56	0.61
BUN (mg/dL)	9.9	11.6	—	2.8	—	2.5	6.0
eGFR (mL/min/1.73 m²)	149	131.5	—	164.5	—	154.8	141.0
Na (mEq/L)	136.3	140.3	138.5	136.3	136.7	138.1	138.8
K (mEq/L)	4.1	3.8	3.5	3.9	3.6	3.9	4.0
Cl (mEq/L)	102.6	101.4	104.2	102.1	103.0	105.1	105.7

Abbreviations: h-PI, hours post-ingestion; WBC, white blood cell count; Hb, hemoglobin; Plt, platelet; PT, prothrombin time; APTT, activated partial thromboplastin time; T-Bil, total bilirubin; AST, aspartate aminotransferase; ALT, alanine aminotransferase; LDH, lactate dehydrogenase; eGFR, estimated glomerular filtration rate. "—" indicates not measured at this time point.

Table 2. Fluid input/output balance by period.

Period	Fluid input (mL)	Urine output (mL)	Balance (mL)
Day 1, 5: 00 PM – Day 2, 6: 00 AM	850	1050	-200
Day 2, 6: 00 AM – Day 3, 6: 00 AM	3943	2580	+1363
Day 3, 6: 00 AM – Day 4, 6: 00 AM	1540	2600	-1060
Day 4, 6: 00 AM – Day 5, 6: 00 AM	~450*	920	-470

Fluid input = intravenous Ringer's acetate. Urine output = measured urinary drainage. Balance = input minus output. * Infusion largely discontinued during this period; residual maintenance volume estimated.

charcoal, and further to 12.16 ng/mL immediately before HD (48.0 h-PI; circuit line). After HD, the level was 5.46 ng/mL (52.2 h-PI; circuit line). Before HDF1, the level was 3.98 ng/mL (65.0 h-PI; peripheral vein). The next available sample, which was obtained approximately 2.4 hours after HDF1 completion, was 17.74 ng/mL (77.0 h-PI), representing a secondary rise. Before HDF2, the level was 2.54 ng/mL (89.0 h-PI); the subsequent sample at 113.0 h-PI was 5.24 ng/mL. No samples were drawn immediately before or after the HDF sessions via the extracorporeal circuit, limiting interpretation of HDF-specific effects. The urine colchicine level was 110.0 ng/mL at 29 h-PI (timed specimen), then gradually decreased through 89 h-PI. A spot urine at 117 h-PI confirmed further decline. Urine creatinine levels fell markedly between 41 and 53 h-PI (429.7 to

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Table 3. Intervention and monitoring timeline (h-PI = hours post ingestion).

Event	Day, clock time	h-PI	Notes
Ingestion and initial management			
Ingestion	Day 1, 1: 00 PM	0	2 dried *Colchicum autumnale* bulbs + energy drink (type/volume unknown)
Initial activated charcoal (1 g/kg) + laxative	Day 2, 4: 00 PM	27.0	Sodium picosulphate hydrate 75 mg; nasogastric tube
Infusion start (Ringer's acetate, ~ 270 mL/h)	Day 2, 4: 00 PM	27.0	Subsequently titrated to 60-200 mL/h
Activated charcoal stool passage confirmed	Day 2, 7: 37 PM	~32	Also confirmed at ~ 43, ~ 55, ~ 70 h-PI
MDAC (0.5 g/kg every 6 h; 13 doses total, 27-101 h-PI)			
Dose 1	Day 2, 4: 00 PM	27	Concurrent with initial AC
Dose 2	Day 3, 12: 00 AM	35	
Dose 3	Day 3, 6: 00 AM	41	
Dose 4	Day 3, 12: 00 PM	47	
Dose 5	Day 3, 6: 00 PM	53	
Dose 6	Day 4, 12: 00 AM	59	
Dose 7	Day 4, 6: 00 AM	65	
Dose 8	Day 4, 12: 00 PM	71	
Dose 9	Day 4, 6: 00 PM	77	Serum colchicine secondary rise (17.74 ng/mL) was sampled at this same time point; HDF1 had ended 2.4 h earlier (74.6 h-PI)
Dose 10	Day 5, 12: 00 AM	83	
Dose 11	Day 5, 6: 00 AM	89	
Dose 12	Day 5, 12: 00 PM	95	
Dose 13 (final)	Day 5, 6: 00 PM	101	
Blood purification therapy			
HD start	Day 3, 12: 59 PM	48.0	Indication: toxin removal; CTA membrane 0.9 m^2, QB 100 / QD 500 mL/min
HD end	Day 3, 5: 10 PM	52.2	Duration 4.2 h
HDF1 start	Day 4, 11: 51 AM	70.8	PS membrane 1.5 m^2, QB 100 / QD 500 mL/min
HDF1 end	Day 4, 3: 35 PM	74.6	Duration 3.75 h
HDF2 start	Day 5, 2: 54 PM	97.9	PS membrane 1.5 m^2, QB 100 / QD 500 mL/min
HDF2 end	Day 5, 7: 00 PM	102.0	Duration 4.1 h
Serum colchicine measurements (LC-MS/MS)			
50.3 ng/mL	Day 2, 4: 00 PM	27.0	On arrival; peripheral vein
21.2 ng/mL	Day 3, 12: 00 AM	35.0	Peripheral vein
12.16 ng/mL	Day 3, 12: 59 PM	48.0	Immediately before HD; circuit line
5.46 ng/mL	Day 3, 5: 10 PM	52.2	Immediately after HD; circuit line

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Table 3 continued. Intervention and monitoring timeline (h-PI = hours post ingestion).

Event	Day, clock time	h-PI	Notes
3.98 ng/mL	Day 4, 6: 00 AM	65.0	~6 h before HDF1 start; peripheral vein
17.74 ng/mL	Day 4, 6: 00 PM	77.0	~2.4 h after HDF1 end; peripheral vein Sampled at same time point as MDAC dose 9 (77 h-PI); represents secondary rise likely reflecting post-HDF1 redistribution and incomplete suppression of enterohepatic recirculation.
2.54 ng/mL	Day 5, 6: 00 AM	89.0	~9 h before HDF2 start; peripheral vein
5.24 ng/mL	Day 6, 6: 00 AM	113.0	~11 h after HDF2 end; peripheral vein
Urine colchicine measurements (LC-MS/MS)			
110.0 ng/mL	Day 2, 6: 00 PM	29.0	Timed urine; first collection post-admission
Subsequent samples	Days 3-5	35-89	Timed urine every 6 h; values shown in **Figure 1**
Final sample	Day 6, 10: 00 AM	117.0	Spot urine specimen

Abbreviations: h-PI, hours post ingestion; AC, activated charcoal; MDAC, multiple-dose activated charcoal; HD, hemodialysis; HDF, hemodiafiltration; CTA, cellulose triacetate; PS, polysulfone; LC-MS/MS, liquid chromatography–tandem mass spectrometry; QB, blood flow rate; QD, dialysate flow rate. Day 1, date of ingestion (1: 00 PM = 0 h-PI). Serum colchicine samples without a pre/post pair immediately adjacent to HDF sessions reflect the nearest available peripheral vein specimen.

43.5 mg/dL), likely reflecting dilution from high-volume infusion. Urinary excretion rates could not be calculated without concurrent urine volumes. Renal function remained preserved throughout (creatinine 0.53-0.65 mg/dL; estimated glomerular filtration rate [eGFR] > 130 mL/min/1.73 m^2).

The patient was discharged on the fifth day of hospitalization after a psychiatric consultation, and was followed up as an outpatient. No obvious abnormalities were observed after hospitalization.

The CARE guidelines were followed in the preparation of this case report.

Discussion

Colchicum autumnale bulbs contain 0.08% to 0.2% colchicine [7]. Our patient ingested 2 dried bulbs. The estimated intake (0.111-0.279 mg/kg) falls within the range associated with severe toxicity, as doses of approximately 0.8 mg/kg or greater have been reported to carry a high risk of fatal outcome, with considerable inter-individual variability [4,8]. No standard treatment for colchicine poisoning exists, as its toxicokinetics are not well understood.

At 26 hours after ingestion, our patient's colchicine levels were much higher than those reported previously [9-11], suggesting that the amount of colchicine ingested was substantial. Furthermore, considering the Cmax and Tmax of colchicine, the colchicine levels were likely even higher 12 hours after ingestion, when the symptoms were most severe. Despite the high serum colchicine levels, the patient's outcome was favorable.

Activated charcoal significantly reduces the amount of colchicine released from the ingested bulb [12], and MDAC inhibits colchicine reabsorption via enterohepatic circulation. It is important to note that, given the 27-hour delay from ingestion to presentation, the drug had very likely undergone multiple cycles of enterohepatic recirculation prior to admission. The persistently elevated serum colchicine level on arrival (50.3 ng/mL) may in part reflect this recirculation-driven redistribution, rather than solely the initial absorbed dose. This observation further supports the plausibility of continued enterohepatic cycling at delayed time points and the potential benefit of MDAC even after the initial absorption phase. Although MDAC is typically considered most effective when initiated early after ingestion, its utility may extend beyond the initial absorption phase in drugs with significant enterohepatic recirculation. In our patient, MDAC was initiated at 27 h-PI—well beyond peak absorption—yet a secondary rise in serum colchicine was observed at 77 h-PI during ongoing MDAC administration. This secondary rise at 77 h-PI occurred approximately 2.4 hours after the completion of HDF1 (74.6 h-PI) and after the ninth dose of MDAC (administered at 77 h-PI; see **Table 3**), suggesting that HDF1 termination may have been followed by tissue redistribution before the next MDAC dose could suppress enterohepatic recirculation. The rebound likely reflects a combination

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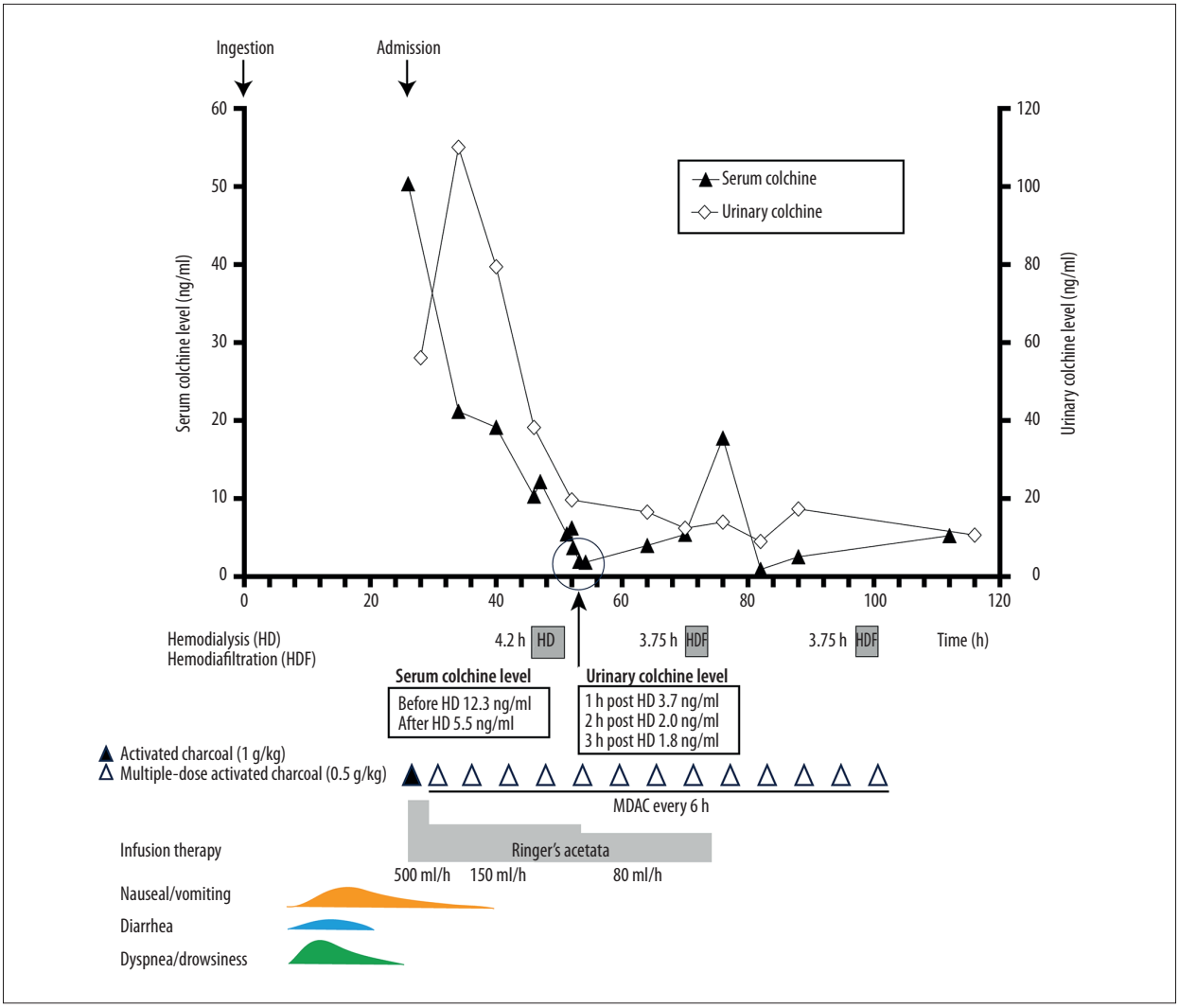

Figure 1. Serial serum and urine colchicine concentrations (ng/mL) plotted against hours post ingestion (h-PI). Key interventions are annotated on the time axis: activated charcoal and laxative (27 h-PI), multiple-dose activated charcoal doses (27-101 h-PI, every 6 h), infusion rate changes, HD (48.0-52.2 h-PI), HDF1 (70.8-74.6 h-PI), and HDF2 (97.9-102.0 h-PI). Timed urine specimens were used for all points except the final specimen at 117 h-PI (spot urine). Pre-HD serum samples were obtained via circuit line; post-HDF samples were peripheral vein obtained 2.4 h (HDF1) and 11 h (HDF2) after session completion.

of post-HDF redistribution from peripheral tissues and incomplete inhibition of enterohepatic recirculation at this time point. This secondary rise could also reflect redistribution from tissues (given the large volume of distribution of 5-8 L/kg [13]), delayed absorption, or incomplete suppression of enterohepatic recirculation. While the mechanism cannot be confirmed from concentration data alone, the pattern is consistent with continued enterohepatic cycling at delayed time points, supporting a plausible rationale for late MDAC use in this setting.

Colchicine is characterized by high tissue binding and a relatively large distribution volume, and it is metabolized mainly in the liver. Therefore, blood purification therapies, such as HD, HDF, and hemoperfusion, have previously been considered ineffective against colchicine toxicity based on their physical properties and kinetics. Pérez Marín et al [14] described a patient with colchicine poisoning who had undergone renal replacement therapy alongside the monitoring of serum colchicine levels. In their study, renal replacement therapy did not contribute to colchicine clearance. Conversely, Lu et al and Ramezani et al [15,16] reported the effectiveness of blood purification therapy for colchicine intoxication. However, the indications for blood purification therapy in the treatment of colchicine poisoning remain controversial. Mullins et al [17] reported the ineffectiveness of HD in diphenhydramine poisoning, which shares kinetic features with colchicine. In our

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patient, the pre- and post-HD serum colchicine samples (12.16 and 5.46 ng/mL, respectively) were both obtained from the dialysis circuit rather than peripheral veins. Circuit-line sampling may not accurately reflect systemic drug concentrations, as the blood in the extracorporeal circuit may differ from peripheral circulation; therefore, these values cannot be used to reliably evaluate the elimination efficacy of HD. For HDF1 and HDF2, immediately pre- and post-session samples were unavailable; post-session values likely reflect redistribution from tissues rather than HDF-specific clearance. This case does not establish extracorporeal removal effectiveness for colchicine.

Colchicine renal clearance accounts for approximately 10% to 20% of total excretion under normal conditions [8]; however, Ben-Chetrit et al noted the potential importance of renal clearance at high serum levels [18]. In our patient, a marked temporal association was observed between the initiation of high-volume fluid infusion and the rapid decrease in serum colchicine, coinciding with high urine colchicine concentrations (110.0 ng/mL at 29 h-PI). Renal function remained preserved throughout (eGFR > 130 mL/min/1.73 m^2). However, renal clearance cannot be quantified from urine concentration data alone without concurrent urine volume measurements; urine creatinine was available at only 2 time points and showed a marked dilutional effect. The observed temporal association between fluid infusion and serum colchicine decrease may reflect increased renal excretion, redistribution, or other concurrent interventions; causality cannot be established from this case.

The serum colchicine level in our patient was elevated again 76 hours after ingestion. The volume of distribution of colchicine was relatively high (5-8 L/kg), suggesting redistribution from the tissues [13]. However, this increase in colchicine levels could not be explained solely by the volume of distribution. Clinical research is warranted to clarify the mechanism by which this delayed increase occurs.

The advantage of this report is the close serial monitoring of serum and urine colchicine levels across a detailed intervention timeline (16 serum and 11 urine time points), enabling hypothesis generation regarding the relative contributions of renal elimination and MDAC to colchicine kinetics. Further prospective studies with standardized sampling protocols are required to clarify the mechanisms and treatment of colchicine poisoning.

Limitations

This case has several important limitations. First, multiple interventions (MDAC, high-volume fluid therapy, HD, and HDF) were administered concurrently, precluding attribution of observed concentration changes to any single therapy. Second, renal clearance cannot be quantified from the available data, as urine volume was not measured at individual colchicine sampling intervals, and urine creatinine was available at only 2 time points. Urine colchicine concentrations were neither corrected for urine volume nor normalized by urine creatinine; therefore, these data reflect concentration only and should not be interpreted as reflecting excretion rate or renal clearance. Third, no serum samples were drawn immediately before HDF1/HDF2 start or immediately after HDF end from the circuit line; post-HDF values likely reflect a mixed signal of HDF effects and post-session redistribution. Fourth, plant identification relied solely on the patient's verbal report, and the dose estimate was based on published colchicine content ranges without weighing the actual material. Fifth, no comprehensive multi-drug toxicology screen was performed (only alcohol screening, which was below the detection limit), and the energy drink's composition and quantity were unknown; these factors cannot be excluded as potential contributors to the observed kinetics. Sixth, colchicine concentrations were measured using a commercially available LC-MS/MS screening platform (Shimadzu Corp); a case-specific analytical validation was not performed, and the assay was not independently validated for the purposes of this report. These limitations should be considered when interpreting the findings, which are presented as hypothesis-generating observations rather than definitive conclusions.

Conclusions

The most common symptoms of colchicine poisoning are gastrointestinal, including nausea, vomiting, diarrhea, and dehydration. In this case, serial monitoring of serum and urine colchicine over a detailed treatment timeline revealed temporal associations between fluid therapy, MDAC administration, and concentration changes. These findings support the hypothesis that promotion of renal excretion through fluid therapy and inhibition of colchicine reabsorption from the enterohepatic circulation using MDAC may contribute to colchicine elimination, while acknowledging that concurrent interventions and the absence of quantitative clearance data preclude definitive conclusions.

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Informed Consent

The patient provided informed consent for the publication of his case history.

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Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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