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Single Case

Treatment of Pediatric Colchicine Poisoning with Single-Pass Albumin Dialysis: A Case Report

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Colchicine · Poisoning · Pediatric dialysis · Continuous kidney replacement therapy · Albumin dialysis

Abstract
Introduction: Colchicine has a narrow therapeutic index, and doses >0.5 mg/kg are considered toxic with a high mortality rate. ***Case Presentation:*** A previously healthy 15-year-old presents to the emergency department with abdominal pain and vomiting following intentional ingestion of colchicine (0.56 mg/kg) 12 h prior. By 24 h post-ingestion, they developed a multi-organ injury with hepatic dysfunction, coagulopathy, lactic acidemia, and pancytopenia, which prompted consideration of extracorporeal therapy (ECT). Considering the characteristics of colchicine, they were treated with continuous venovenous hemodiafiltration (CVVHDF) with single-pass albumin dialysis (SPAD) for 42 h. They were subsequently discharged from the intensive care unit 48 h after stopping CVVHDF with normal kidney function, resolved coagulopathy, and resolving pancytopenia and hepatic dysfunction. The rationale for CVVHDF with SPAD was based on the high protein binding, variably high volume of distribution, previous reports showing a sieving coefficient of 0.2 with CVVH, and the high mortality risk. We anticipated a high potential for rebound. Thus, continuous clearance would facilitate redistribution from the extravascular to intravascular space. SPAD was used to enhance the elimination of protein-bound fractions; the principle is that adding albumin to dialysate creates a protein-binding disequilibrium where the drug from the blood side may bind to albumin on the dialysate side. ***Conclusion:*** Colchicine ingestion of >0.5 mg/kg is highly toxic, and in addition to supportive management, continuous kidney replacement therapy with SPAD may be considered.

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Introduction

Colchicine is a tricyclic lipid-soluble alkaloid derived from *Colchicum autumnale* (autumn crocus). It is used to treat inflammatory conditions (e.g., crystal arthropathies, familial Mediterranean fever, and Behcet's disease). Colchicine has a narrow therapeutic index; doses >0.5 mg/kg are considered toxic and associated with a high mortality rate [1]. We present a pediatric case of colchicine poisoning treated with continuous kidney replacement therapy (CKRT) with single-pass albumin dialysis (SPAD).

Case Presentation

A 15-year-old previously healthy patient presents to the hospital after a suicide attempt by an intentional colchicine overdose. They had no pertinent family history. Chronologically, within 24 h they presented as follows:

- Hour 0: after an argument with a family member, they locked themselves in their room and intentionally ingested their parent's supply of 55 × 0.5 mg tabs of colchicine and 6 × 40 mg tabs of pantoprazole. They fell asleep following the ingestion.
- Hour 5: they woke with excruciating abdominal pain and vomiting (non-bilious and non-bloody).
- Hour 8: they presented to a local hospital. The intentional overdose was discovered, estimated at 0.56 mg/kg of colchicine. They had 15 vomits and started on intravenous 5% dextrose + 0.9% saline at 90 mL/h. Emergency transfer to a quaternary care center was done.
- Hour 12: they arrived at our quaternary care pediatric hospital and were immediately admitted to the pediatric intensive care unit (PICU).

Clinical Findings

On examination, their vital signs were within the normal range. They were euvolemic and had a Glasgow Coma Scale of 15. Their neurological, cardiorespiratory, and abdominal exams were unremarkable.

Timeline

Table 1 highlights relevant blood tests and electrocardiogram results at time intervals since the ingestion of colchicine. Of note, they developed leukocytosis, hepatic dysfunction, coagulopathy, and lactic acidemia throughout their course, with intervention instituted 24 h from ingestion.

Diagnostic Focus and Assessment

In acute colchicine poisoning, doses of <0.5 mg/kg are considered sub-toxic, doses of 0.5–0.8 mg/kg are highly toxic with a mortality rate of 10%, and doses of >0.8 mg/kg are typically lethal [1]. The effects of acute poisoning are described in three phases [1]: phase 1 (first 24 h) – primarily gastrointestinal effects (abdominal pain; vomiting; diarrhea, reflecting GI mucosal damage), possibly with leukocytosis and hypovolemia; phase 2 (1–7 days) – a spectrum of multi-organ dysfunction which may include respiratory failure, cardiovascular compromise, kidney failure, liver failure, central nervous system impairment, bone marrow suppression, and disseminated intravascular coagulation; metabolic derangements may include acidosis, multiple electrolyte abnormalities, and dysglycemia; and phase 3 (if survives; >7 days) – organ failure resolution, rebound leukocytosis, and alopecia [1].

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Table 1. Summary of investigations (intervention administered at 24 h)

Laboratory values	Ref	Time since ingestion, h								
		12	24	36	48	72	96	108	132	180
Hemoglobin, g/L	112–151	147	132	122	**106**	**91**	**100**	**108**	120	119
Platelet, g/L	173–361	228	180	**151**	**76**	**19**	**21**	**52**	**83**	221
WBC, g/L	4.23–9.99	7.9	4.41	**15.69**	**12.26**	**2.99**	**3.19**	4.38	5.16	5.45
Lymphocyte, g/L	1.26–3.41	**5.2**	**3.69**	1.44	**0.27**	**0.76**	**0.7**	1.96	2.55	2.56
Neutrophils, g/L	1.45–6.75	2.0	3.9	6.43	**11.44**	1.97	2.01	**1.42**	**0.99**	1.8
pH, arterial	7.35–7.50	7.35	7.4	7.38	7.44	7.41	7.44	–	–	–
pCO_2, mm Hg	32–45	46	33	**28**	**30**	33	36	–	–	–
Bicarbonate, mmol/L	20–31	26	20	**16**	21	21	24	26	–	–
Base excess, mmol/L	−2 to 2	−1	**−4**	**−8**	**−3**	**−3**	0	1	–	–
INR	0.8–1.2	**1.3**	**2.4**	**2.1**	**1.6**	1.1	1.0	1.2	–	–
PTT, s	24–40	–	**48.9**	**47.4**	38.6	34.1	39.2	32.0	–	–
Fibrinogen, g/L	1.9–4.3	–	–	1.9	2.4	3.1	**4.9**	**5**	–	–
Lactate, mmol/L	≤2.4	1.4	**2.7**	**3.6**	1.5	0.5	0.6	0.6	0.8	–
Sodium, mmol/L	135–143	**144**	143	141	140	140	137	138	135	138
Potassium, mmol/L	3.7–5.0	3.9	4.1	**2.9**	**3.6**	4.1	4.2	4.2	**5.3**	4.0
Chloride, mmol/L	99–111	100	**112**	**115**	111	**112**	105	103	99	101
Creatinine, μmol/L	40–69	49	53	**31**	**22**	**24**	**29**	**32**	40	41
Urea, mmol/L	2.8–7.0	4.0	3.9	**0.7**	**0.7**	**0.7**	**1.5**	3.6	–	–
Albumin, g/L	35–52	51	51	32	26	24	30	39	–	–
Calcium, mmol/L	2.22–2.54	–	**2.14**	**1.91**	**1.94**	**2.01**	**2.16**	**2.21**	**2.43**	**–**
Phosphate, mmol/L	1.10–1.88	1.25	**0.98**	**1.00**	1.25	1.48	1.33	1.16	1.22	**–**
ALT, U/L	≤24	21	**27**	**29**	**28**	**29**	**40**	**36**	**37**	**28**
AST, U/L	<31	**92**	**158**	**191**	**204**	**148**	**114**	**46**	**34**	26
CK, U/L	50–240	–	–	**595**	**575**	**666**	**513**	95		24
Lipase, U/L	4–39	–	–	**88**	**–**	**–**	**168**	**561**	**191**	**731**
ECG parameters										
QRS, ms	<120	80	88	82	78	–	82	88	82	86
QTc, ms	<460	**460**	437	433	443	–	422	423	427	437

Items in bold are abnormal.

Our patient ingested a dose of 0.56 mg/kg which is highly toxic, requiring acute therapy. They were given activated charcoal (to bind colchicine; repeated doses may be of benefit due to enterohepatic recirculation), rifampin (enzyme inducer of CYP3A4 and P-gp; may increase colchicine elimination), and N-acetylcysteine (unclear if helpful but given with aim to mitigate oxidative stress and cell death by apoptosis) [2, 3]. Based on case report evidence, colchicine-specific Fab antibody fragments were considered but unavailable at our center [4]. Despite maximal medical therapy, our patient showed signs of evolving toxicity (GI symptoms, leukocytosis, hepatic dysfunction, coagulopathy, and lactic acidemia) which urgently prompted consideration for KRT.

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Therapeutic Focus and Assessment

No studies compare the efficacy of KRT to conservative management in colchicine overdose. To determine the viability of KRT, we examined the characteristics of colchicine:

- Molecular weight: 399.4 g/mol = 399.4 Dalton – high flux membranes used for KRT easily clear drugs <1,500–2,000 Dalton.
- Protein binding: 40% – this is a high degree of protein binding and is a major determinant of the extent of removal by KRT as only the fraction of unbound drug can be removed [5].
- The volume of distribution (Vd): 2–12 L/kg – colchicine is lipid-soluble and rapidly distributed to peripheral tissue, where it binds to intracellular elements [6]. This leads to a high Vd, which limits removal by KRT as it is not present in significant concentrations in the vascular space. The high Vd also lends to high rates of rebound following clearance [5].
- Kidney clearance: 10–20% – the higher the endogenous kidney clearance, the higher the capacity to be removed by KRT.
- Molecular charge: neutral – colchicine is not affected by the Gibbs-Donnan effect or the negative charge of albumin, which can affect the membrane permeability of polycationic drugs [7].
- Membrane adsorption: minimal – as colchicine is a neutral compound, it will be minimally affected by membrane adsorption with anionic or cationic residues [8].

With the characteristics of colchicine in mind, the following KRT modalities were considered:

- Intermittent hemodialysis (IHD): IHD is favorable in the poisoned patient due to the speed at which a toxin can be removed and the ability to treat concomitant metabolic derangements. There are case reports describing the use of IHD for other indications in colchicine overdoses, i.e., oliguric renal failure and acidosis [9], but it is difficult to quantify the impact of IHD on colchicine clearance when started for other indications. With colchicine, the high Vd and the moderate percentage of protein binding limit the viability of IHD. Moreover, the possible rebound phenomenon would not be addressed by IHD.
- Peritoneal dialysis (PD): the use of PD is infrequent in poisoning when other forms of KRT are available because overall clearances are much lower with PD than other techniques.
- Plasma exchange (PLEX): as colchicine has a high degree of protein binding, PLEX, with the exchange of circulating proteins, theoretically stands as a potential treatment. There are cases in the literature of high-dose colchicine poisoning >0.5 mg/kg which was treated by PLEX [9–11]. However, more recent reports have provided conflicting evidence. In 2021, a case report demonstrated that a single dose of total PLEX removed only 0.01% of the ingested dose and was unlikely to have influenced the outcome [12]. The available evidence favors the lack of efficacy of PLEX, however, it is usually reserved for patients with dose ingestions >0.5 mg/kg which carries a higher rate of mortality that may skew outcomes [13, 14].
- CKRT: CKRT employs diffusive and convective clearance, but solute removal is lower per unit of time compared to IHD. However, the continuous and prolonged nature of KRT was seen as favorable as with the high Vd and protein binding, we anticipated that CKRT would allow a continuous redistribution of colchicine from the extravascular to intravascular space. There have been 2 reports of CKRT used for colchicine clearance with a case in 2018 calculating a sieving coefficient of 0.2 suggesting there is limited but partial clearance [9, 15].

Acknowledging the unclear potential for benefit, based on the previously demonstrated sieving coefficient of 0.2, high level of protein binding (unclear percentage due to patient condition), and variably high Vd in overdose settings, our patient was started on continuous venovenous hemodiafiltration (CVVHDF) with SPAD at 24 h post-ingestion. The CKRT prescription is detailed in Table 2.

Follow-Up and Outcomes

Twenty-four hours after CVVHDF with SPAD, our patient had improved lactic acidemia and coagulopathy but had progressive pancytopenia. They received 42 h of KRT, which was well tolerated with no circuit clotting but with anticipated hypokalemia, given their normal kidney function. Their urine output remained at 1.5–3.5 mL/kg/h while on KRT. In conjunction with the PICU and toxicology team, a decision was made to stop CKRT, as the only remaining signs of toxicity were pancytopenia and very mild hepatic transaminitis. They were transferred from PICU to the pediatric ward on day 5, 48 h after stopping KRT with baseline neurologic function, normal kidney function, resolved coagulopathy, and improved transaminitis.

Discussion

Colchicine is a tricyclic lipid-soluble alkaloid that binds free tubulin dimers, preventing microtubular polymerization; affected cells have impaired: mitosis, Golgi apparatus functions, endocytosis/exocytosis, cell morphology, and motility [1]. At toxic doses, colchicine arrests mitosis, thus inhibiting cell division. This occurs in multiple cell lines in the body. Its effect is most potent in homeostatic systems with the highest turnover rate, i.e., bone marrow, gastrointestinal tract, and hair follicles [1]. Colchicine reaches peak plasma concentration 0.5–3 h after administration, has a half-life of 20–40 h, and undergoes extensive hepatic first-pass metabolism with rapid distribution [16]. Significant poisoning (>0.5 mg/kg) carries a poor prognosis despite conservative therapies [1] and has pushed clinicians to seek viable options for extracorporeal therapy (ECT).

We considered PLEX and CVVHDF with SPAD as potential treatment options. PLEX is a viable treatment, especially in patients with hepatotoxicity and coagulopathy, but has had conflicting case report data as to its efficacy in colchicine poisoning [9–14]. Given the high degree of protein binding and Vd of colchicine, we hypothesized that a standard 4-h session of PLEX would be effective in removing circulating toxins but would lack the benefit of continuous removal of colchicine molecules which are mobilized from tissue due to rebound. Our approach to CVVHDF with SPAD was guided by a desire to maximize solute clearance while providing the added potential to remove protein-bound toxins. Our center can use heparin or citrate for anticoagulation. We selected ACD-A citrate to regionally anticoagulate the circuit without affecting the patient's coagulopathy. However, our citrate solution is run in the prefilter "pre-blood pump" location within the machine, preventing us from increasing the pre-dilution fluid replacement rate. In the dialysis solution pump position of the machine, we performed SPAD for the theoretically enhanced elimination of protein-bound fractions. The principle for its use is that adding albumin to the dialysate creates a protein-binding disequilibrium where the drug from the blood side may bind to the albumin on the dialysate side. We could not access a molecular adsorbent recirculating system or extracorporeal liver-assisted device. With SPAD, we achieved a 4.2% or 42 g/L concentration by adding a 1-L bag of 25% albumin to a 5-L bag of dialysate. Albumin concentration of 3–4% with SPAD effectively removes water-soluble and protein-bound toxins. This is shown in a study calculating clearance rates of bilirubin and bile acids according to the first-order kinetic rate of log

Table 2. CKRT prescription

Access	Left femoral vein – 11.5 Fr non-tunneled catheter
Modality	CVVHDF
Anticoagulation	Citrate (ACD-A) – due to coagulopathy
Filter	HF1000
Blood flow rate	100 mL/min
Dose	3,000 mL/1.73 m^2/h
Pre-blood pump fluid rate (ACD-A)	150 mL/h
Post-filter replacement fluid	1,000 mL/h
Replacement fluid composition	*Prism0CAL B22*
	Sodium: 142 mmol/L
	Bicarbonate: 22 mmol/L
	Additives
	Potassium chloride: 4 mmol/L
	Sodium phosphate: 1.5 mmol/L
Dialysate flow rate	1,600 mL/h
Dialysate composition	*Prism0CAL B22*
	Sodium: 142 mmol/L
	Bicarbonate: 22 mmol/L
	Additives
	25% albumin (concentration of 4.2% or 42 g/L by adding a 1 L bag of 25% albumin to a 5-L bag of dialysate)

graphs of concentrations versus time at varying albumin concentrations [17]. In the post-filter replacement solution position of the machine, we ran a higher-than-usual replacement solution to maximize solute clearance (higher rates of SPAD would be a feasibility issue), being mindful of the potential to increase clotting risk. Utilizing a citrate anticoagulation system, hypocalcemia was anticipated and circumvented by a continuous calcium chloride infusion separate from the circuit, titrated to maintain systemic normocalcemia. Hypophosphatemia due to solute clearance was addressed by the addition of sodium phosphate (1.5 mmol/L) to post-filter replacement fluid. The typical cessation for KRT in poisonings would be guided by plasma levels and assessment of rebound effect to determine KRT duration. Colchicine may be measured by liquid chromatography-mass spectrometry, HPLC, and radioimmunoassay. These methods were not readily available and thought to be of limited use as there is no established correlation between plasma levels and disease severity [1]. At our center, we did not have access to validated methods of colchicine measurement in the dialysate to calculate sieving coefficients. As such, the cessation of KRT was driven by the improvement of patient biochemical markers.

In summary, although it is difficult to provide definitive evidence of colchicine removal by ECT, we offer that it is worth considering this therapeutic approach, especially for severe overdoses (>0.5 mg/kg) given the high mortality rate and limited treatment options. Further research is needed into the viability and optimal mode of ECT in the treatment of colchicine poisoning. The CARE Checklist for this case report has been completed and is attached as online supplementary material (for all online suppl. material, see https://doi.org/10.1159/000543020).

Statement of Ethics

This case report is exempt from requiring ethics approval from the SickKids Research Ethics Board. Written informed consent was obtained from the patient's parent or legal guardian for the publication of the details of their medical case and any accompanying images.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

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Data Availability Statement

All data generated or analyzed during this study are included in this article and its online supplementary material files. Further inquiries can be directed to the corresponding author.

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