317. Plasmapheresis in managing copper sulphate poisoning

Aleksandra Babulovska, Lidija Petkovska, Andon Cibishev, Niko Bekjarovski, Zanina Pereska, Natasha Simonovska and Kristin Kostadinoski

University Clinic of Toxicology, SS Cyril and Methodius University, Skopje, Republic of North Macedonia

Objective: Plasmapheresis is a therapeutic intervention that involves extracorporeal removal, return, or exchange of blood plasma or components [1]. We report a case of copper toxicosis with clinical improvement after plasmapheresis (three cycles).
Case report: A 27-year-old previously healthy male was brought in after ingestion of an unknown amount of powdered copper sulfate, with suicidal intent. Epigastric pain and vomiting developed 20 minutes after ingestion and several hours later he developed hematemesis and melena. Laboratory analyses were insignificant except for total bilirubin 48.9 µmol/L (later 108.5 µmol/L), direct bilirubin 12.5 µmol/L (later 13 µmol/L), and indirect bilirubin 36 µmol/L (later 97 µmol/L). The methemoglobin level was 2% (later 0.4%), and serum copper was 1.2 mg/dL (later 0.65 mg/L). He had sinus bradycardia on electrocardiogram (EKG) and echocardiography was normal. Blood gas analyses were pH 7.39, pCO_2 4.17 kPa, pO_2 13.6 kPa, SpO_2 98%, HCO_3 20.6 mmol/L, and base excess −5.9 mmol/L. Gastroduodenal endoscopy revealed grade IIb gastritis and bulbous duodenum II according to the Zargar classification. Supportive treatment consisted of intravenous fluids, intravenously pantoprazole, sodium bicarbonate, dexamethasone, vitamin C, and furosemide. On the second day, hemoglobin (Hb) dropped to 97 g/L (later 66 g/L), with increased lactate dehydrogenase (LDH) 517 U/L (later 2430 U/L), aspartate aminotransferase (AST) 45 U/L (later 88 U/L), and blood urea nitrogen (BUN) 11.7 mmol/L. He had increased D-dimer 2179 ng/mL (later 13176 ng/mL) and he received low molecular weight heparin (LMWH). Due to the unavailability of chelating agents, plasmapheresis was indicated and performed in three cycles on the third, fifth, and ninth days. Anemia from hemolysis and bleeding was corrected with red cell concentrate transfusion. He developed acute renal failure with creatinine 257 µmol/L (later 423 µmol/L) with normal urine output 3000 mL/24 hours and estimated glomerular filtration rate (eGFR) 26.6 mL/min/1.73 m^2. Vigorous intravenous fluid administration was continued. He was discharged after 19 days with renal function still mildly impaired (creatinine 135 µmol/L) and referred to the Psychiatric Clinic.
Conclusion: Copper sulfate poisoning can lead to multi-organ failure and mortality. The essential treatment is supportive measures. Our case suggests that plasmapheresis can be used for treatment when chelating agents are unavailable.

Reference

[1] Schwartz J, Padmanabhan A, Aqui N, et al. Guidelines on the use of therapeutic apheresis in clinical practice-evidence-based approach from the Writing Committee of the American Society for Apheresis: the seventh special issue. J Clin Apher. 2016;31:149–162.

318. Sometimes it is the pump: a case of baclofen withdrawal secondary to intrathecal pump malfunction

Matthew K. Kolbeck[a,b] and Vincent J. Calleo[a,b]

[a]SUNY Upstate Medical University, Syracuse, NY, USA; [b]Upstate NY Poison Center, Syracuse, NY, USA

Objective: Baclofen, a gamma-aminobutyric acid (GABA)-B receptor agonist, may be administered orally or intrathecally. Intrathecal baclofen allows for higher concentrations in the spinal cord without concomitant oversedation [1]. Baclofen withdrawal may cause seizures, hallucinations, autonomic dysregulation, among other features [1–3]. Intrathecal baclofen withdrawal may be particularly challenging to diagnose and treat [3]. We report a typical case of intrathecal baclofen withdrawal highlighting a diagnostic dilemma identifying pump malfunction.
Case report: A 23-year-old patient with spastic quadraparesis managed with an intrathecal baclofen pump presented with sudden onset lower extremity spasms. Vital signs showed hyperthermia (38.9 °C), tachycardia (131 bpm), and tachypnea (21/min). He had a normal sensorium but increased tone and myoclonus in all extremities. Initial treatment with diazepam mildly improved symptoms. Pump interrogation showed no malfunction and radiographs demonstrated normal pump position without catheter discontinuity. His team pursued alternative differentials though toxicology advised further pump interrogation. Meanwhile, oral baclofen (30 mg Q4H) with adjunct diazepam was initiated as his baseline intrathecal dose was 549.5 µg/day. On hospital day three, his pump was accessed but fluid was not aspirated, suggesting catheter-pump interruption. On hospital day 10, he underwent revision of the pump by neurosurgery, and was discharged from the hospital on day 12 with return to baseline.
Conclusion: Typical diagnostic pathways for pump failure (e.g., interrogation, imaging) inadequately exclude pump malfunction and withdrawal may occur without overt signs of pump malfunction. Care must be taken to avoid premature closure on alternate diagnoses. Intrathecal baclofen withdrawal may also be challenging to manage despite large doses of oral baclofen and care must be taken to provide sufficient GABA agonists to prevent or treat complications of withdrawal while restoring pump function [1].

References

[1] Romito JW, Turner ER, Rosener JA, et al. Baclofen therapeutics, toxicity, and withdrawal: a narrative review. SAGE Open Med. 2021;9:20503121211022197.
[2] Al-Khodairy AT, Vuagnat H, Uebelhart D. Symptoms of recurrent intrathecal baclofen withdrawal resulting from drug delivery failure: a case report. Am J Phys Med Rehabil. 1999;78:272–277.
[3] Greenberg MI, Hendrickson RG. Baclofen withdrawal following removal of an intrathecal baclofen pump despite oral baclofen replacement. J Toxicol Clin Toxicol. 2003;41:83–85.

319. An audit of the use of hydroxocobalamin (Cyanokit®) for suspected cyanide poisoning in patients admitted to a UK burn centre

Mark Pucci[a,b], Mahroo Khalid[c], Ekta Punj[c], Jonathan Looms[b] and Peter Welby-Everard[b]

[a]National Poisons Information Service, Birmingham, United Kingdom; [b]Sandwell & West Birmingham NHS Trust, Birmingham, United Kingdom; [c]University Hospitals Birmingham NHS Foundation Trust, Birmingham, United Kingdom

Objective: The diagnosis of cyanide poisoning in smoke inhalation victims is challenging but should be based on a history of