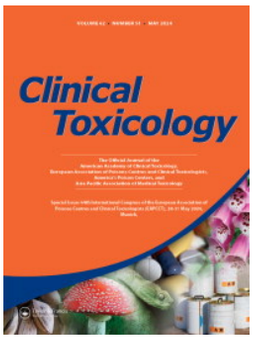

Clinical Toxicology

ISSN: (Print) (Online) Journal homepage: www.tandfonline.com/journals/ictx20

44th International Congress of the European Association of Poisons Centres and Clinical Toxicologists (EAPCCT), 28–31 May 2024, Munich, Germany

To cite this article: (2024) 44th International Congress of the European Association of Poisons Centres and Clinical Toxicologists (EAPCCT), 28–31 May 2024, Munich, Germany, Clinical Toxicology, 62:sup1, 1-135, DOI: 10.1080/15563650.2024.2333651

To link to this article: https://doi.org/10.1080/15563650.2024.2333651

which is highly cardiotoxic and binds to and reversibly inhibits the Na-K ATPase pump in myocardial cells [1]. This can be fatal and hence the tree is known as the "suicide tree". The clinical presentation and mechanism of toxicity is similar to digoxin. Clinical management is similar, with supportive treatment of bradycardia and hyperkalaemia. Administration of digoxin immune fab can be considered as well. We report a case to improve awareness about the potential toxic effects of *Cerbera odollam* ingestion.

Case report: A 37-year-old female presented to the Emergency Department (ED) four hours after ingestion of *Cerbera odollam*, "Pong Pong" seed with intent to self-harm. She broke through the husk, pounded two seeds and mixed it with honey and ice cream. Subsequently, she ingested 3 spoonful of this mixture. There was no co-ingestion with any other drug or substance. She initially had perioral and facial region numbness, as well as some lethargy, nausea and vomiting. Her initial vitals were: temperature 37 °C, blood pressure 102/70 mmHg, heart rate 54 beats/minute, respiratory rate 20 breaths/minute, SpO_2 100% (room air). The physical examination was unremarkable. Initial investigations showed digoxin concentration <0.4 μg/L, salicylate concentration <3 mg/L, paracetamol <5 mg/L, lactate 1.9 mmol/L. Blood gases were pH 7.559, pCO_2 24 mmHg, and bicarbonate 21 mmol/L. Haemoglobin was 10.6 g/dL and urea, creatinine, serum sodium, potassium, glucose and liver function tests were within normal range. The patient was diagnosed with mild cerberin poisoning. The toxicology service was consulted and advised activated charcoal administration and admission to short stay unit under the Accident and Emergency department for observation with continuous electrocardiogram (ECG) monitoring. Overnight, she had intermittently bradycardia with heart range ranging between 45 and 55 beats per minute. The heart rate improved to about 75 beats/minute by the morning (about 15 h post-ingestion) and the patient was discharged well after psychiatry review. She did not require any digoxin-specific antibody therapy.

Conclusion: *Cerbera odollam* fruit ingestion caused nausea, vomiting, perioral numbness and sinus bradycardia with hypotension in this patient but she did not require digoxin-specific antibody therapy. Physicians should be aware of the potential mortality and morbidity and possible treatment options in patients that ingest *Cerbera odollam* seeds.

Reference

[1] Menezes RG, Usman MS, Hussain SA, et al. *Cerbera odollam* toxicity: A review. J Forensic Leg Med. 2018;58:113–116.

46. Delayed treatment of severe hepatitis in unrecognized phalloides syndrome

Zanina Pereska[a], Dushan Petkovski[b], Natasha Simonovska[a] and Aleksandra Babulovska[a]

[a]University Clinic of Toxicology, Ss Cyril and Methodius University, Skopje, North Macedonia; [b]University Clinic of Cardiology, Ss Cyril and Methodius University, Skopje, North Macedonia

Objective: Delayed or insufficient antidote treatment of patients with mushroom poisoning is a challenge for every toxicologist [1]. We present a case of delayed recognition of severe phalloides syndrome treated with an additional acetylcysteine dose to the standard protocol with favorable outcome.

Case report: A middle-aged man was admitted to the Clinic on the 4th day after ingestion of mushrooms he had picked and eaten under the assumption that they were parasol mushrooms (*Macrolepiota procera*). He had a medical history of diabetes mellitus type 2, *coronary artery bypass graft* x3 (only aspirin) and hypertension. Profuse gastroenterocollitis developed 16 h after ingestion (over a weekend) and for the first 2 days he was treated at a local medical center for infectious enterocolitis as an outpatient. Laboratory analyzes on the third day showed an increased transaminase activity with a progressive increase the next day, after which he was brought to the clinic. He was alert, blood pressure 100/60 mmHg, an electrocardiogram (ECG) showed sinus rhythm, heart rate 100/min, pain under the ribs in the right upper quadrant, with yellow discoloration of the sclera. Laboratory analysis revealed platelets 129×10^i/L, gamma-glutamyl transferase (GGT) 199 U/L (later 608 U/L), alanine aminotransferase (ALT) 8389 U/L, aspartate aminotransferase (AST) 13804 U/l, lactate dehydrogenase (LDH) 7961 U/L, direct bilirubin 104 μmol/L, blood urea nitrogen (BUN) 17.1 mmol/L, creatinine 133.8 μmol/L, prothrombin time (PT) 29.3 s (11–14 s), and D-dimer 8183 ng/mL. Hepatitis viral marker negative. The patient was intensively rehydrated, treated with IV acetylcysteine (200 mg/kg over 4 h, followed by 100 mg/kg over 16 h, with additional 100 mg/kg dose), oral silymarin (100 mg 3×2) and supportive treatment. Abdominal ultrasound presented enlarged liver (not congested) with intensive steatosis. Progressive thrombocytopenia from day 2 of hospitalization was noted with a nadir of 58×10^i/L (150–450×10^i/L) on the 9th day of poisoning. He received dexamethasone and low molecular weight heparin (LMWH) and PT normalized on the 8th day of poisoning (14.2 s). The patient recovered after 10 days of treatment, with normalized transaminases after 3 months. The mushroom ingested was assumed to be *Amanita phalloides*, based on the clinical features, laboratory findings and recognition of the ingested mushroom with a look-alike poisonous mushroom from a mushroom atlas by the patient.

Conclusion: Data about the consumption of picked mushrooms should be seriously considered when determining the etiology of severe acute gastroenterocollitis. Delayed treatment of phalloides syndrome may worsen the clinical course and increase the potential risk of lethal outcome.

Reference

[1] Bonacini M, Shetler K, Yu I, et al. Features of patients with severe hepatitis due to mushroom poisoning and factors associated with outcome. Clin Gastroenterol Hepatol. 2017;15:776–779.

47. Coma and seizures induced by *Amanita pantherina* poisoning

Marco Cirronis, Andrea Giampreti, Georgios Eleftheriou, Mariapina Gallo, Maria Gioia Contessa, Lorella Faraoni, Raffaella Butera and Giuseppe Bacis

Bergamo Poison Center, ASST Papa Giovanni Hospital, Bergamo, Italy

Objective: *Amanita pantherina* poisoning is a rare, poorly described event. The clinical picture is usually associated with *A. muscaria*, but *A. pantherina* contains more muscimol and more commonly causes coma [1]. We describe a case of severe coma and seizures after *A. pantherina* ingestion.