



**Posters submitted at the Middle East and North Africa
Clinical Toxicology Association Annual Conference and
Scientific Meeting**

March 28-31, 2021

INGESTION OF UNKNOWN SEEDS CAN CAUSE FATALITY: THE CASE SERIES OF RICIN TOXICITY

Vildan Ozer¹, Sukriye Tasci Karagol², Huseyin Cetin Ketenci³, Aynur Sahin¹, Ziad Kazzi⁴

1 – MD, Karadeniz Technical University Faculty of Medicine, Department of Emergency Medicine, Trabzon, Turkey

2 – MD, Karadeniz Technical University Faculty of Medicine, Department of Internal Medicine, Trabzon, Turkey

3-MD, Forensic Medicine Institute Trabzon Group Presidency, Trabzon, Turkey

4 - MD, Emory University School of Medicine, Department of Emergency Medicine, Atlanta, U.S.A.

Introduction:

Ricin is a protein toxin derived from castor beans (*Ricinus communis*) and causes protein synthesis inhibition. It is classified as category-B by the CDC and is considered a potential chemical weapon (1). The symptoms it causes vary according to the type of exposure. which can be fatal. There is no specific antidote for ricin exposure (2). In this case series, two patients who experienced ricin poisoning by accidentally ingesting castor beans without knowing that they are toxic are presented.

Case Series:

Case 1: A 64-year-old female patient presented to the emergency department with abdominal pain, nausea, vomiting and diarrhea 4 hours after the ingestion of 15 unknown seeds . On arrival, her vitals were stable; ECG was normal sinus rhythm and, on her physical examination was normal abdominal tenderness. Lactate level was elevated (28 mg /dl) in the laboratory tests and other biochemical parameters were unremarkable. IV fluids and antiemetics administered as supportive therapy, then her family brought the seeds for identification (Figure 1). The seeds were diagnosed as castor beans. Symptoms resolved at 10 days of observation, she discharged without any complication.

Case 2: 56-year-old female patient was admitted to the emergency department with abdominal pain, severe nausea, recurrent vomiting and diarrhea that started approximately 4 hours after ingestion of 5 unknown beans. On admission her vitals were stable, physical examination was normal except abdominal tenderness. ECG was in normal sinus rhythm. Laboratory tests were unremarkable . After 24 hours of observation and administration of supportive therapy (IV fluids, antiemetics), all symptoms recovered and she discharged.

Both 2 cases' blood and urine samples were analyzed at Forensic Laboratory with LC / MS technique for confirmation. Ricin was detected in all samples. (Figure 2a: Case 1, Figure 2b: Case 2)



Figure 1. Castor beans

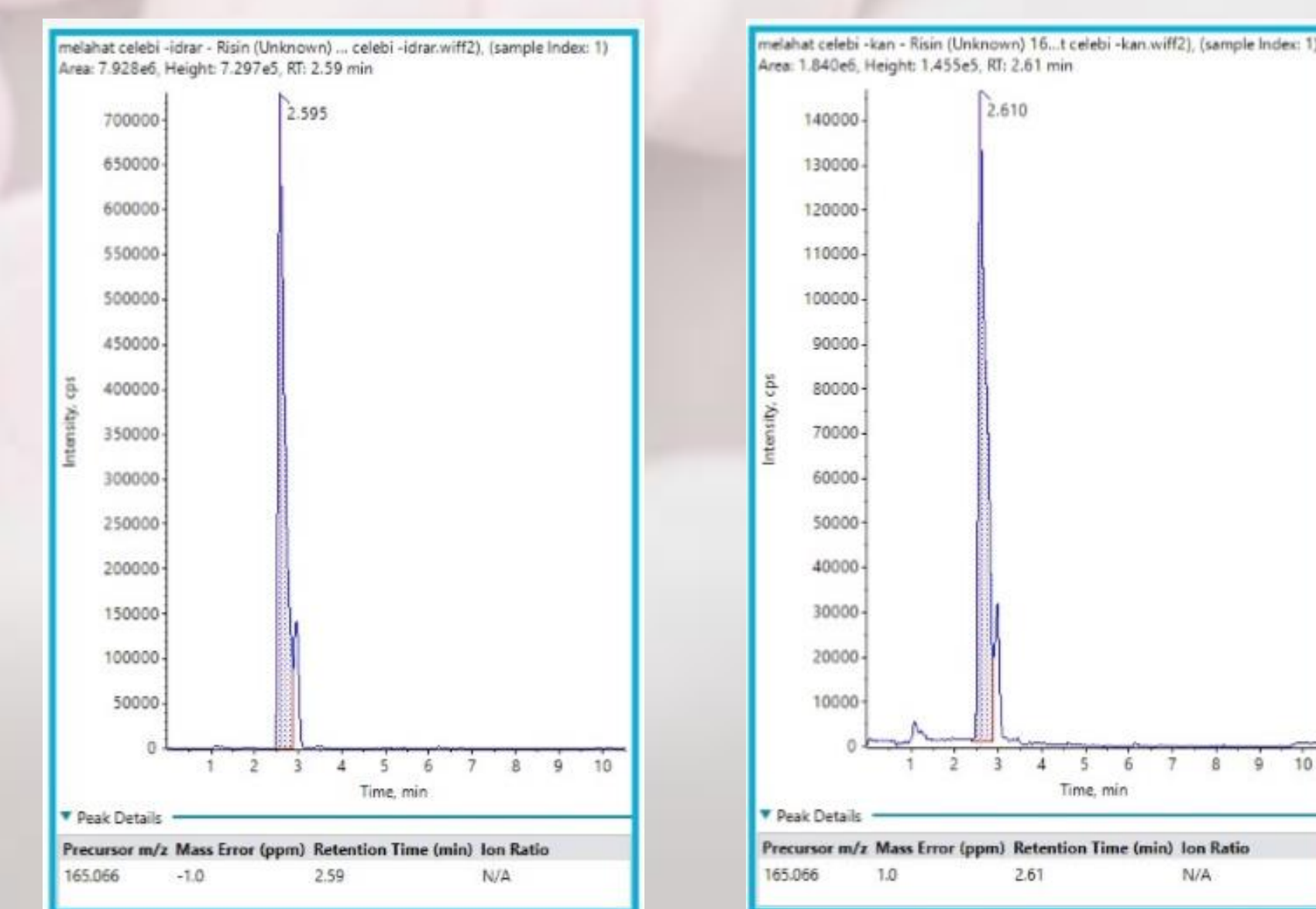


Figure 2a.

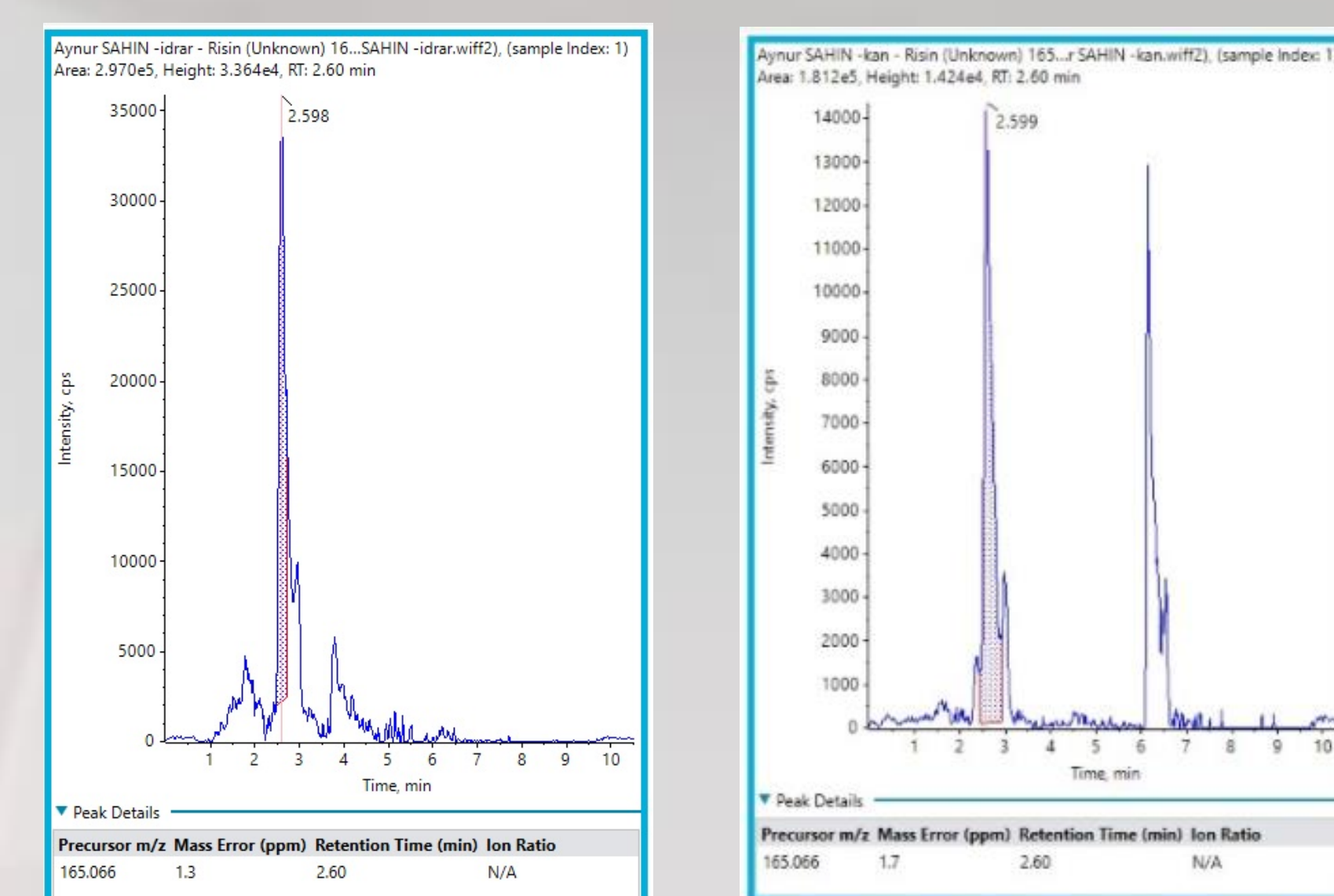


Figure 2b.

Discussion and Conclusion :

The seeds of the castor bean plant (*Ricinus Communis* L) contain ricin toxin. Ricin toxin is one of the most toxic natural substances known. It is a toxalbumin that inhibits the function of ribosomes, the subcellular organelle responsible for protein synthesis (3). It is classified as category-B by the CDC and is considered a potential chemical weapon (1).

Toxicity symptoms vary with exposure pattern. The most toxic form of exposure is the inhalation route. Exposure by inhalation; It is manifested by non-cardiogenic pulmonary edema, diffuse necrotizing pneumonia, interstitial and alveolar inflammation and edema (4). The second most toxic exposure is oral intake. Oral intake; It can cause abdominal pain, diarrhea, vomiting, gastrointestinal bleeding, renal failure due to fluid loss, and hypovolemic shock. If swallowed without chewing, the risk of toxicity is reduced. Local injection of ricin toxin may cause hardening at the injection site, edema in the regional lymph node, hypotension and death (5). In both cases we encountered, there were oral intake and related gastrointestinal symptoms.

Although deaths due to oral ingestion of 2 seeds have been reported in the literature, there are also cases that survived oral ingestion of 200 seeds. Ricin toxin can be detected in blood and other body fluids by ELISA method (6).

Supportive therapy constitutes the basis of the treatment. There is no specific antidote. Correction of fluid and electrolyte imbalances, and monitoring of liver and kidney functions are the first steps of treatment. Coagulopathies should also be corrected (2). Our cases were also discharged with supportive therapy, fluid resuscitation and clinical observation.

As a result, simple case of gastroenteritis can cause fatal outcome depending on the etiology. Physicians should be aware of ricin toxicity in the differential diagnosis after the presentation of unknown seed ingestion.

References:

- 1.CDC (Centers for Disease Control and Prevention). (2016). Ricin: Diagnosis & Laboratory Guidance for Clinicians. Available at: <https://emergency.cdc.gov/agent/ricin/clinicians/diagnosis.asp>. Last accessed: November 30, 2017
- 2.Wedin GP, Neal JS, Everson GW, Krenzelok EP. Castor bean poisoning. Am J Emerg Med. 1986; 4:259-61.
- 3.Musshoff F, Madea B. Ricin poisoning and forensic toxicology. Drug Test Anal. 2009;1;184-91.
- 4.Wilhemsen C, Pitt L. Lesions of acute inhaled lethal ricin intoxication in rhesus monkeys. Vet Pathol.1993; 30:482.
- 5.POISINDEX1 Editorial Staff: Castor beans (Management/Treatment Protocol). In: Klasco RK, ed. POISINDEX1 System. Greenwood Village; Colorado: Thomson MICROMEDEX. (Edition expires [Nov 2003]).
- 6.Poli MA, Rivera VR, Hewetson JF, Merrill GA. "Detection of ricin bycolorimetric and chemiluminescence ELISA" . *Toxicon*. 1994; 32:1371-1377.

COCAINE INDUCED SYNDROME OF INAPPROPRIATE ANTIDIURETIC HORMONE SECRETION AND SEROTONIN SYNDROME

Vildan Ozer ¹, Mehtap Pehlivanlar Kucuk ², Mustafa Uzun ¹ , Aysenur Pir³ , Aynur Şahin ¹

1 –MD, Karadeniz Technical University Faculty of Medicine, Department of Emergency Medicine, Trabzon, Turkey

2– MD, Karadeniz Technical University Faculty of Medicine, Department of Pulmonary Medicine, Trabzon, Turkey

3 – MD, Karadeniz Technical University Faculty of Medicine, Department of Internal Medicine, Trabzon, Turkey

Introduction:

Serotonin syndrome (SS), also called serotonin toxicity, is a potentially life-threatening condition associated with increased serotonergic activity in the central nervous system (1). Syndrome of inappropriate antidiuretic hormone secretion (SIADH) is an impaired water excretion disorder caused by an inability to suppress antidiuretic hormone secretion (2). Cocaine is extracted from the leaves of the coca plant (*Erythroxylum coca*) native to the Andean highlands of South America. The use of cocaine by chewing by the locals in this region by purifying it was first performed in the 1880s as a local anesthetic in surgery. Today, an estimated 18.2 million people around the world use it for various purposes (3). Cocaine can cause various life-threatening clinics, including serotonin syndrome and inappropriate antidiuretic hormone (ADH) syndrome in the case of intoxication. This case report aimed to discuss a rare complication of its use: cocaine induced SIADH and SS.

Case Report:

A 42-year-old female patient was brought to the emergency department at around 3:00 am due to confusion and agitation. Her vitals were stable except that her body temperature was 38.3 ° C. Her consciousness was confused, uncommunicative with her surroundings, and she had agitated behavior. The pupils were isochoric. Meningeal irritation sign and lateralization were not detected. There were hyperreflexia, ocular and inducible clonus. There were no lesions or signs of trauma on the skin. Other system examinations were normal. It was learned that the patient took cocaine at around 11:00 pm, and as the hours passed, her communication with her environment decreased and disappeared. Intravenous hydration was started after monitoring. Central imaging was performed after intravenous 10 mg diazepam was administered. A temporary reduction in agitation was observed after diazepam.

In laboratory tests, sodium 119 mEq / l (136-146), chlorine 88 mEq / l (96-110), creatine kinase(CK) 512 U/l (20-200), myoglobin 379.5 µg / L (25-58), White blood cell count was 14.47 x10³ / µl (4.8-10.8), blood glucose value was 173 mg / dl, and blood urea nitrogen was 5 mg / dl. Urine osmolality was above 100 mosmol / kg. In other results, no obvious pathology was found. SIADH and SS were considered in the patient, secondary to cocaine use. Due to the hyponatremia present in the patient, 150 cc of 3% hypertonic NaCl was added to the treatment and Cyproheptadine was started as an antiserotonergic agent. During the patient's hospitalization, the serum sodium level was 119-128-137-136 mEq / l. The CK level was seen as 512-696-1124-16294 U / L. The patient was conscious at the 26th hour of her admission to the hospital. On the third day of the follow-up, she left the hospital with her own request.

Discussion and Conclusion :

Cocaine is thought to have three different mechanisms; including inhibition of the reuptake of biogenic amines, sodium channel blockage, and stimulating amino acids glutamate and aspartate to increase the concentration in the brain, especially the nucleus accumbens. Cocaine blocks the presynaptic reuptake of biogenic amines at adrenergic receptors (4,5). With this effect, serotonin syndrome, which is also defined as the triad of mental status changes, autonomic hyperactivity and neuromuscular abnormalities in the central nervous system, occurs with increased serotonergic activity. Serotonin syndrome is diagnosed with Hunter criteria according to clinical findings (6). In our patient, in favor of SIADH after cocaine intake; serum Na level was 119 mEq / l, and urine osmolality was above 100 mosmol/kg. Hunter criteria were met by hyperthermia up to 38.5 ° C, high creatine kinase levels, agitation, ocular and inducible clonus.

SIADH is an impaired water excretion disorder caused by the inability to suppress antidiuretic hormone secretion (2). The etiology includes central nervous system disorders, malignancies, drug and substance use, surgery, lung pathologies, hormonal problems, HIV infection, and genetic mutations (7). Supraoptic and paraventricular nuclei in the hypothalamus, which control ADH synthesis and release, receive intense serotonergic and catecholaminergic innervation (8-10). Cocaine increases serotonergic and catecholaminergic effects, affecting these nuclei and causing SIADH. SIADH should be suspected in any patient with hyponatremia, hypoosmolality, and urine osmolality above 100 mosmol / kg (2).

Although there are limited case reports in the literature regarding cocaine induced SS and SIADH, this is the first case report that includes both syndromes. In case of SS due to cocaine, there is usually coingestion of another serotonergic agent in medical history. As a conclusion, physicians should be aware of these syndromes as complication of cocaine use.

References:

- 1)Boyer EW, Shannon M. Serotonin sendromu. N Engl J Med 2005; 352: 1112.
- 2)Rose BD, Post TW. Clinical Physiology of Acid-Base and Electrolyte Disorders, 5th ed, McGraw-Hill, New York 2001. p.703.
- 3)Myers MG, Rohsenow DJ, Monti PM, Dey A. Patterns of cocaine use among individuals in substance abuse treatment. Am J Drug Alcohol Abuse 1995; 21:223.
- 4)Tella SR, Schindler CW, Goldberg SR. Cardiovascular effects of cocaine in conscious rats: relative significance of central sympathetic stimulation and peripheral neuronal monoamine uptake and release mechanisms. J Pharmacol Exp Ther 1992; 262:602.
- 5)Tella SR, Schindler CW, Goldberg SR. Cocaine: cardiovascular effects in relation to inhibition of peripheral neuronal monoamine uptake and central stimulation of the sympathoadrenal system. J Pharmacol Exp Ther 1993; 267:153.
- 6)Dunkley EJ, Isbister GK, Sibbritt D, et al. The Hunter Serotonin Toxicity Criteria: simple and accurate diagnostic decision rules for serotonin toxicity. QJM 2003; 96:635
- 7)Uptodate/Pathophysiology and etiology of the syndrome of inappropriate antidiuretic hormone secretion (SIADH)
- 8)Buijss RM. Ceftat-d M, Pool CW. Hoomeman EM. The dopaminergic innervation of the supraoptic and paraventricular nucleus. A light and electron microscopical study. Brain Res, 1984; 323:65-72.
- 9)Deeavel C. GeffardM, Calas A. Comparative study of dopamine- and noradrenaline-immunoreactive terminals in the paraventricular and supraoptic nuclei of the rat, Neurosci Lett. 1987; 77: 149-154.
- 10)Rossi NF Dopaminergic control of angiotensin II induced vasopressin secretion in vitro. Am J Physiol. 1998; 275.- 687-693,

SCORPION FISH STING: A CASE REPORT

Vildan Ozer¹, Aynur Sahin¹, Engin Ilhan², Sinan Pasli¹, Melih Imamoglu¹, Ziad Kazzi³

1 - Assistant Professor of Emergency Medicine Karadeniz Technical University Faculty of Medicine, Department of Emergency Medicine

2 - Research Assistant of Emergency Medicine Karadeniz Technical University Faculty of Medicine, Department of Emergency Medicine

3 - Associate Professor, Department of Emergency Medicine, Emory University School of Medicine

Introduction:

Marine envenomation is mostly seen in settlements adjacent to the sea due to fishing and marine tourism. These cases can be encountered in a wide range from simple local skin lesions to systemic symptoms that can cause mortality. Delays in diagnosis and treatment can cause mortality, especially in regions where species with venoms that may cause systemic effects are not endemic. In this case, a patient admitted to the emergency service with pain and redness in the extremity after contact with scorpionfish is presented.

Case Report:

A 52-year-old female patient who had a medical history of hypertension and hypothyroidism was admitted to the emergency room with severe pain, burning, redness on her right hand after scorpion fish sting 40 minutes ago (Figure 1). On her admission, she was oriented, cooperated, GCS 15 points, her vital signs were stable except the blood pressure was 190/100 mmHg. On her physical examination, there were 6-7 sting marks on the right hand thenar region, but no foreign body was detected. The right hand was slightly edematous, and there was a redness that started in the thenar region of the right hand and extended to 2-3 cm proximal to the wrist (Figure 2). the laboratory tests were normal. Steroid (dexamethasone 8 mg) and antihistaminic (pheniramine 50 mg) therapies were administered as support therapies. The patient's hand was kept in hot water (42-45 ° C) immersion as much as patient can. The temperature of the water was controlled at regular intervals. At 6th hour of observation, the pain, redness recovered. (Figure 3). the patient was discharged with antibiotic and antihistaminic prescription at the end of 6-hour observation.



Fig 1: Black scorpion fish



Fig 2: Redness on the patient's hand at the time of admission



Fig 3: Redness at the 5th hour of the follow-up

Discussion and Conclusion :

Scorpaena porcus, locally known as the black scorpion fish, is common in the Mediterranean and the Black Sea (1). It is a fish species frequently encountered by coastal fishermen in the Eastern Black Sea region (3). In particular, cases of poisoning are seen with the stings during the cleaning of the fish. The contact of swimmers with these fish species in the seas is another important cause of poisoning. The most common complaint of patients is severe pain after contact. Pain peaks in a few minutes and usually resolves in 24 hours (1,4). Symptoms are usually localized in the affected limb. Edema, ecchymosis, warmth and erythema are common local symptoms. Systemic symptoms are extremely rare. Systemic symptoms may include hypotension, sweating, respiratory distress and abdominal tenderness (2,4,6). In this case, it was observed that the patient was severe hypertensive on admission and normotensive in the follow-up. The main purpose of treatment is to provide analgesia. The agents to be used in patients range from simple analgesics to opioid analgesics. Paracetamol was sufficient in this case. Hot water application to the affected extremity is important for patient comfort. It is recommended that the affected limb be immersed in water at a temperature sufficient for the patient to tolerate (usually 40-45 ° C) (2,4,7,8). In the treatment of hot water immersion, the temperature of the water should be checked regularly and changed when the water starts to cool. Local anesthetic and nerve blockage can be tried in pain management (4,7,8). Prophylactic antibiotics are generally not recommended for small lesions that are not necrotic, foreign bodies are not detected (8,9). Steroid and antihistamine application are among alternative treatment options (7,8). Tetanus prophylaxis is recommended for these patients as they have both bite wounds and traumatic injuries (1,4,7,8). It has been reported that the wounds healed in the late period. Therefore, patients should be informed about complications (infection, anaphylaxis, etc.) in such injuries.

In conclusion, even if there is no fatal case reported in the area of residence in patients admitted to the emergency room with marine envenomation, it should be considered that local symptoms may progress, follow-up periods should be prolonged and systemic symptoms should be prevented by initiating early treatment.

References:

- 1)Goldfrank, Lewis (2007), Goldfrank'ın Toksikolojik Aciller El Kitabı, (Çev. Salim Satar), 1. Baskı içinde (920-922), Nobel Kitabevi, Adana
- 2)Hifumi, T., Fukuchi, Y., & Otani, N. (2020). Marine Envenomation. SN Comprehensive Clinical Medicine, 1-5.
- 3)Samsun, S., & Sağlam, N. E. (2018). Karadeniz'deki (Samsun, Ordu, Giresun) İskorpit (Scorpaena porcus Linnaeus, 1758) Balığının Biyolojisi. Süleyman Demirel Üniversitesi Eğirdir Su Ürünleri Fakültesi Dergisi, 14(4), 291-302.
- 4)Olson, Kent (Ed) (2008), Poisoning & Drug Overdose, 7th Edition içinde (292-293), The McGraw-Hill Education, United States of America
- 5)Kizer, K. W., McKinney, H. E., & Auerbach, P. S. (1985). Scorpaenidae envenomation: a five-year poison center experience. Jama, 253(6), 807-810.
- 6)Rensch, G., & Murphy-Lavoie, H. M. (2019). Lionfish, Scorpionfish, And Stonefish Toxicity. StatPearls [Internet].
- 7)Hornbeak, K. B., & Auerbach, P. S. (2017). Marine envenomation. Emergency Medicine Clinics, 35(2), 321-337.
- 8)Balhara, K. S., & Stolbach, A. (2014). Marine envenomations. Emergency medicine clinics of North America, 32(1), 223-243.
- 9)Diaz, J. H. (2015). Marine scorpaenidae envenomation in travelers: epidemiology, management, and prevention. Journal of travel medicine, 22(4), 251-258

Successful Management of Severe Verapamil Overdose with VA-ECMO

Vildan Ozer¹, Oguzhan Ahmet Kucuk², Kibar Yasar Guven³, Huseyin Cetin Ketenci⁴, Aynur Sahin¹, Ziad Kazzi⁵

1 - Karadeniz Technical University Faculty of Medicine, Department of Emergency Medicine

2 - Karadeniz Technical University Faculty of Medicine, Department of Anesthesiology & Reanimation

3 - Karadeniz Technical University Faculty of Medicine, Department of Cardiac Surgery

4-Forensic Medicine Institute Trabzon Group Presidency,

5-Emory University School of Medicine Department of Emergency Medicine,

Introduction:

Calcium channel blocker (CCB) poisonings are the most common agents causing mortality in worldwide and new approaches and updates on antidotal and supportive treatment are still under the investigation. Verapamil is a Class-IV antiarrhythmic agent used most commonly for the treatment of supraventricular tachy-arrhythmia and hypertension, and the nondihydropyridine group is a Calcium Channel Blocker (CCB) (1, 2) The purpose of this case report was to discuss the clinical management of a patient who was stabilized with VA ECMO in the early period of cardiovascular failure, recovering without sequelae in late pharmacotherapy in clinical follow-up after high dose verapamil intake.

Case Report:

A 36-year-old male with a history of hypertension and multiple suicide attempts presented to our emergency department (ED) approximately 45 minutes after ingesting in a suicide attempt, his own 42 tablets of Tarka forte® (Verapamil Hydrochloride/Trandolapril-Immediate Release) 240/4 mg per tablet, 9 tablets of Zestat® (Mirtazapine) 15 mg per tablet, and 14 tablets of Isordil® (Isosorbide Dinitrate) 5 mg per tablet. He was alert, oriented and cooperative. His initial vital signs were stable (heart rate: 90 beats per minute, blood pressure: 110/80 mmHg) and the electrocardiogram (ECG) showed normal sinus rhythm with no other abnormalities(Figure-1). In the ED, gastric decontamination was performed with orogastric lavage tube and activated charcoal and a large number of tablets were aspirated via irrigation. Two hours after arrival, the blood pressure decreased to 80/50 mm Hg and the heart rate increased to 120 beats per minute. The patient's ejection fraction (EF) and wall motion was deemed normal upon bedside echocardiography performed by the emergency physician (ECHO). The patient's blood pressure improved after intravenous fluid bolus and low-dose norepinephrine infusion (0.05 mcg/kg/minute) . Fifty-five hours after the ingestion, the blood pressure decreased to 70/50 mmHg and the heart rate was 62 beats per minute. He was given 3 g of calcium gluconate IV, 1 U/kg bolus of intravenous regular insulin followed by a 1 U/kg/hour insulin infusion. The ejection fraction remained normal by bedside echocardiogram despite the low systolic blood pressure that remained low after increasing the insulin infusion rate to 10 U/kg/h and administering 4 g IV of glucagon.

The patient was intubated and mechanically ventilated followed by placement on the ECMO Device with epinephrine infusion 60 hours post-ingestion. After ECMO, the patient's need for epinephrine decreased, high dose insulin (HDI) therapy was weaned, urine output increased, and serum lactate decreased. In the bedside ultrasound imaging, the thickness of the intestinal wall was measured as 5 millimeters in this region, differential diagnosis were ischemic intestine, Ileus, or pharmacobezoars however, no further imaging was obtained because the patient was attached to ECMO Device. Fifteen minutes cardiac arrest period developed at the 72 hours post-ingestion and return of spontaneous circulation occurred after 15 minutes of cardiopulmonary resuscitation (CPR). Diuretic treatment was initiated due to the development of non-cardiogenic pulmonary edema. Vasopressor support was discontinued on the 7th day of post-ingestion. The ECMO catheter was discontinued on the 8th day of hospital admission and the patient was discharged on the 11th day of hospital admission. The blood drug level of the patient was analyzed with LC/MS/MS Device in the Forensic Medicine Laboratory. Mirtazapine, Trandolapril, and Isosorbide dinitrate levels were negative, Serial serum verapamil and lactate levels are shown in Figure-2

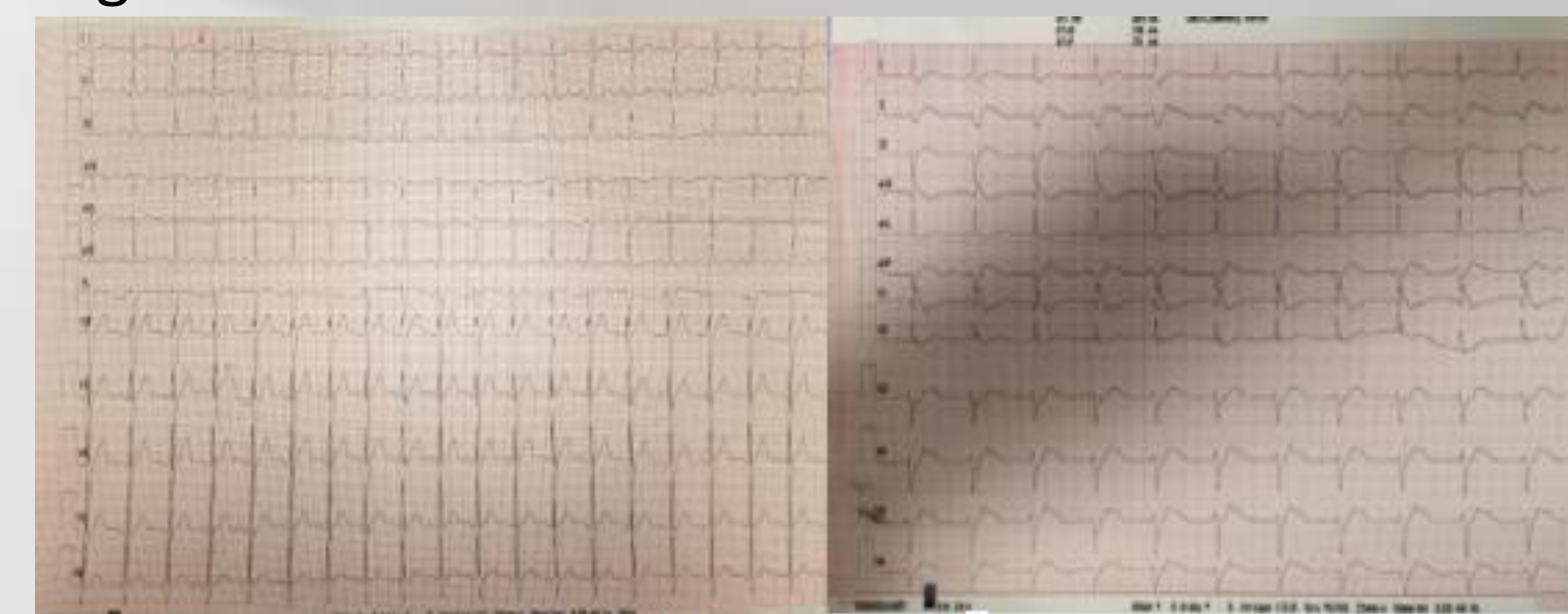


Figure-1: A) ECG during the patient's admission, B) The patient's ECG just before being placed on ECMO after 55 hours

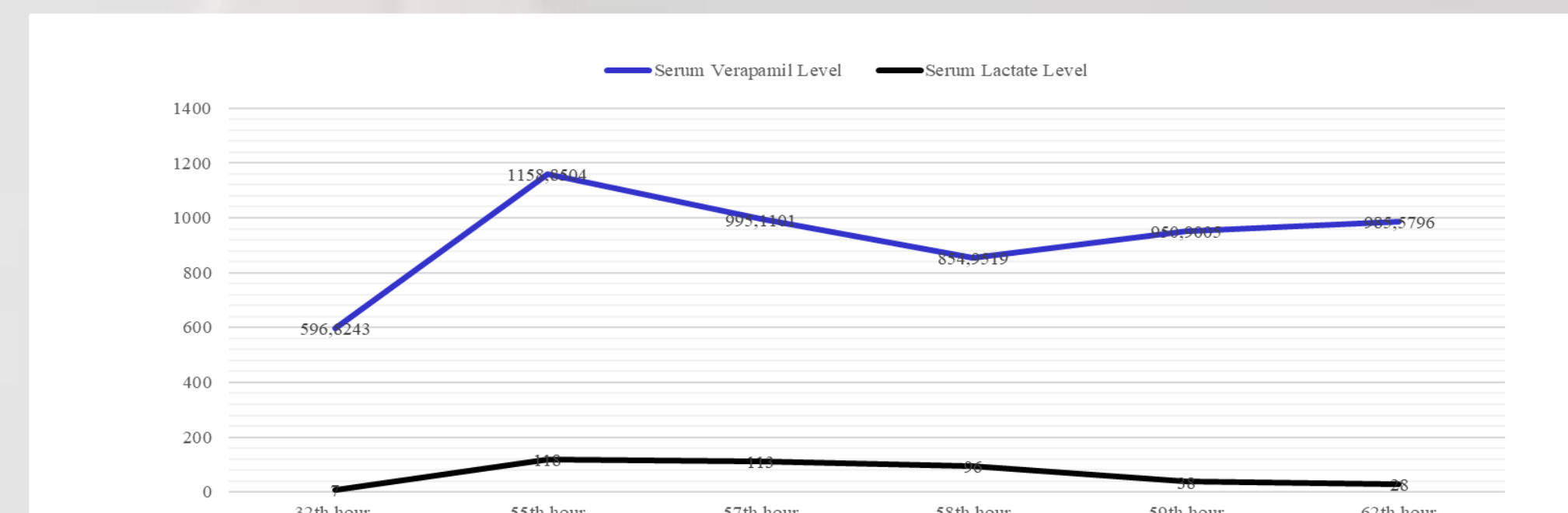


Figure-2: Serum Verapamil (ng/mL) and Lactate (mg/dL) levels

Discussion and Conclusion :

Previous reports of verapamil poisoning where serial serum verapamil levels were measured, have shown that the peak serum level was achieved and then gradually dropped at earlier hours of presentation (3-6). In this case report, serum verapamil levels peaked at 55 hours after the ingestion despite being an immediate-release formulation. This can be due to hypotension-induced hypoperfusion of the intestinal tract and secondary slowing of peristalsis. Again, the decrease in peristalsis due to the antimuscarinic effect of mirtazapine, which was another medication ingested by the patient, may have contributed to slowing peristalsis although the ingestion of this drug was not confirmed by LCMS. Also, it was reported in the literature that pharmaco bezoars occur in intestinal structures in multidrug overdoses, and that causes delayed serum peaks and deterioration in the patient's clinical manifestation with the dissolution of these bezoars in decreased intestinal motility(1) . It is recommended to start HDI early in cases of CCB poisoning in cases of hemodynamic insufficiency with impaired cardiac contractility (2). Although cardiac EF was checked twice, EF did not decrease, and HDI treatment was given due to the patient's non-responsive status to IV fluid, IV vasopressor and IV calcium, but no clinical response was obtained. ECMO should be considered early in cases where the history and initial clinical findings point towards a critical overdose with high risk of death. VA-ECMO in high dose metoprolol and amlodipine intoxication, and VV-ECMO in high dose verapamil intoxication were successful when applied before cardiac arrest (1, 10). In this case, the early recommendation of VA-ECMO in addition to the advance therapies administered, had a likely role in the favorable outcome of this critical poisoning.

Considering the potential risk of cardiac collapse in severe calcium-channel and beta-blocker poisonings, it is important to plan for potential need of advanced therapies like ECMO.

References:

- 1) Nordmark Grass, Johanna, et al. "A case of massive metoprolol and amlodipine overdose with blood concentrations and survival following extracorporeal corporeal membrane oxygenation (ECMO)." *Clinical Toxicology* 57.1 (2019): 66-68.
- 2) Nelson LS, Howland MA, Lewin NA, Smith SW, Goldfrank LR, Hoffman RS. *High Dose Insulin*. Goldfrank's Toxicologic Emergencies, Eleventh Edition New York. McGraw-Hill Education 2019, A21: 953-958.
- 3) Ramoska EA, Spiller HA, Myers A. Calcium channel blocker toxicity. *Ann Emerg Med*. 1990; 19:649-53. [CrossRef]
- 4) Kolcz, Jacek, et al. "Extracorporeal life support in severe propranolol and verapamil intoxication." *Journal of intensive care medicine* 22.6 (2007): 381-385.
- 5) Holzer, Michael, et al. "Successful resuscitation of a verapamil-intoxicated patient with percutaneous cardiopulmonary bypass." *Critical care medicine* 27.12 (1999): 2818-2823.
- 6) Kuhlmann, U., et al. "Plasmapheresis in life-threatening verapamil intoxication." *Artificial Cells, Blood Substitutes, and Biotechnology* 28.5 (2000): 429-440.
- 7) Durward, Andrew, et al. "Massive diltiazem overdose treated with extracorporeal membrane oxygenation." *Pediatric Critical Care Medicine* 4.3 (2003): 372-376.
- 8) Engbrechtsen, Kristin M., et al. "High-dose insulin therapy in beta-blocker and calcium channel-blocker poisoning." *Clinical toxicology* 49.4 (2011): 277.
- 9) Masson, Romain, et al. "A comparison of survival with and without extracorporeal life support treatment for severe poisoning due to drug intoxication." *Resuscitation* 83.11 (2012): 1413-1417.
- 10) Ho, A. T. N., T. N. Pham, and C. R. Barrios. "Catastrophic Non-Cardiogenic Pulmonary Edema Secondary to Massive Verapamil Overdose and Treatment with Extracorporeal Membrane Oxygenation." *A43. CRITICAL CARE CASE REPORTS: TOXICOLOGY AND POISONINGS*. American Thoracic Society, 2020. A1665-A1665.

Acute yellow oleander (Thevetia peruviana) poisoning: a case report of asymptomatic ingestion in Saudi Arabia, is it luck?

Noof Alabdulwahab¹, Halah Almuheidib¹, Shaikhah Al-Otaibi¹⁻², Dunya Alfaraj¹⁻².

1- Imam Abdulrahman Bin Faisal University, Dammam, Saudi Arabia

2- Department of Emergency medicine, King Fahad hospital of the university, Alkhobar, Saudi Arabia.



جامعة الإمام عبد الرحمن بن فيصل
IMAM ABDULRAHMAN BIN FAISAL UNIVERSITY



Introduction

- Thevetia peruviana or yellow oleander (Y.O.) is a plant that belongs to the Dogbane family.
- Used widely as a home plant.
- It is a tree with a green leaves with yellow flowers and triangular fruit containing seeds
- Yellow oleander is considered a fetal plant, as it contains cardiac glycosides in all its parts with different concentrations, which is more in the seeds. ^(1,2)
- Ingestion of any part of the plant can result in poisoning with variable pharmacokinetics.
- Following the yellow oleander's ingestion, clinical features are gastrointestinal symptoms, neurological features and cardiovascular symptoms.^(1,4)
- Asymptomatic cases following the yellow oleander ingestion are rare, and case reports of such cases are limited.
- We report a case of a 55 years old Saudi lady that presented asymptotomatically to our emergency department after the ingestion of yellow oleander fruit and finally discharged healthy.

Case Report

- A 57-year-old Saudi lady known case of type two diabetes mellitus and dyslipidemia.
- Presented to the emergency department five hours after ingestion of the home-grown yellow oleander fruit without its' seeds (figure 2,3) as she thought it is an edible fruit.
- The fruit was 3.4 grams without the seeds.
- The patient was asymptomatic.
- Vital signs and physical examination was normal.
- ECG showed normal sinus rhythm, with normal intervals.
- Labs showed normal electrolyte, renal & liver function. Digoxin level was normal.
- The patient was admitted for close observation.
- Serial ECG, VBG, electrolyte panel, and digoxin level done and was all normal.
- After 24 hours, the patient remained asymptomatic.
- All serial Digoxin level was normal <0.19n/ml (normal range 0.8-2.0 ng/ml).
- After discharge, the patient was asked to monitor her heart rate at home 48 hours, 72 hours, and four days after ingestion and was in normal range from 90 - 99 beats/minute.
- Figure (1) shows the home-grown yellow oleander tree with the yellow flower. Figures were taken from the patient by her consent.



Figure 1

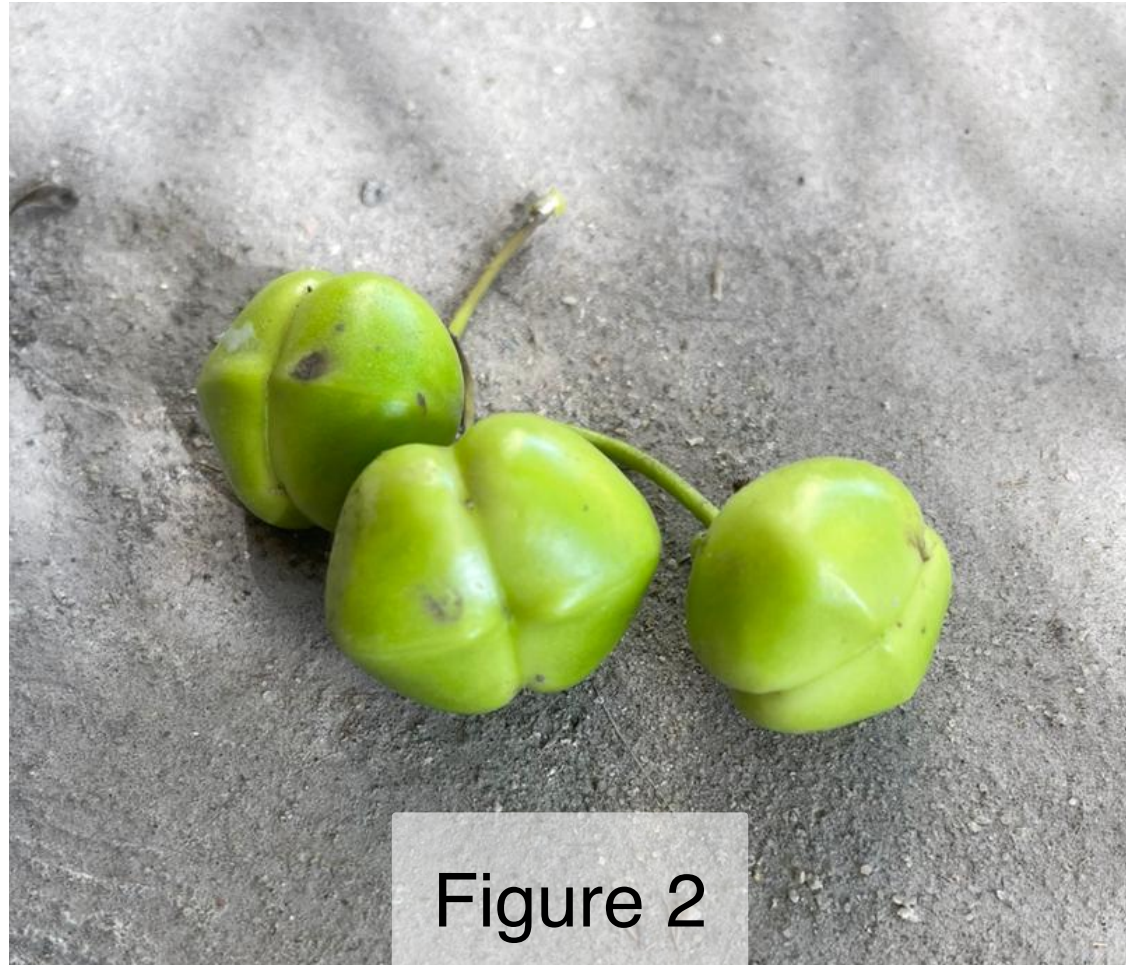


Figure 2

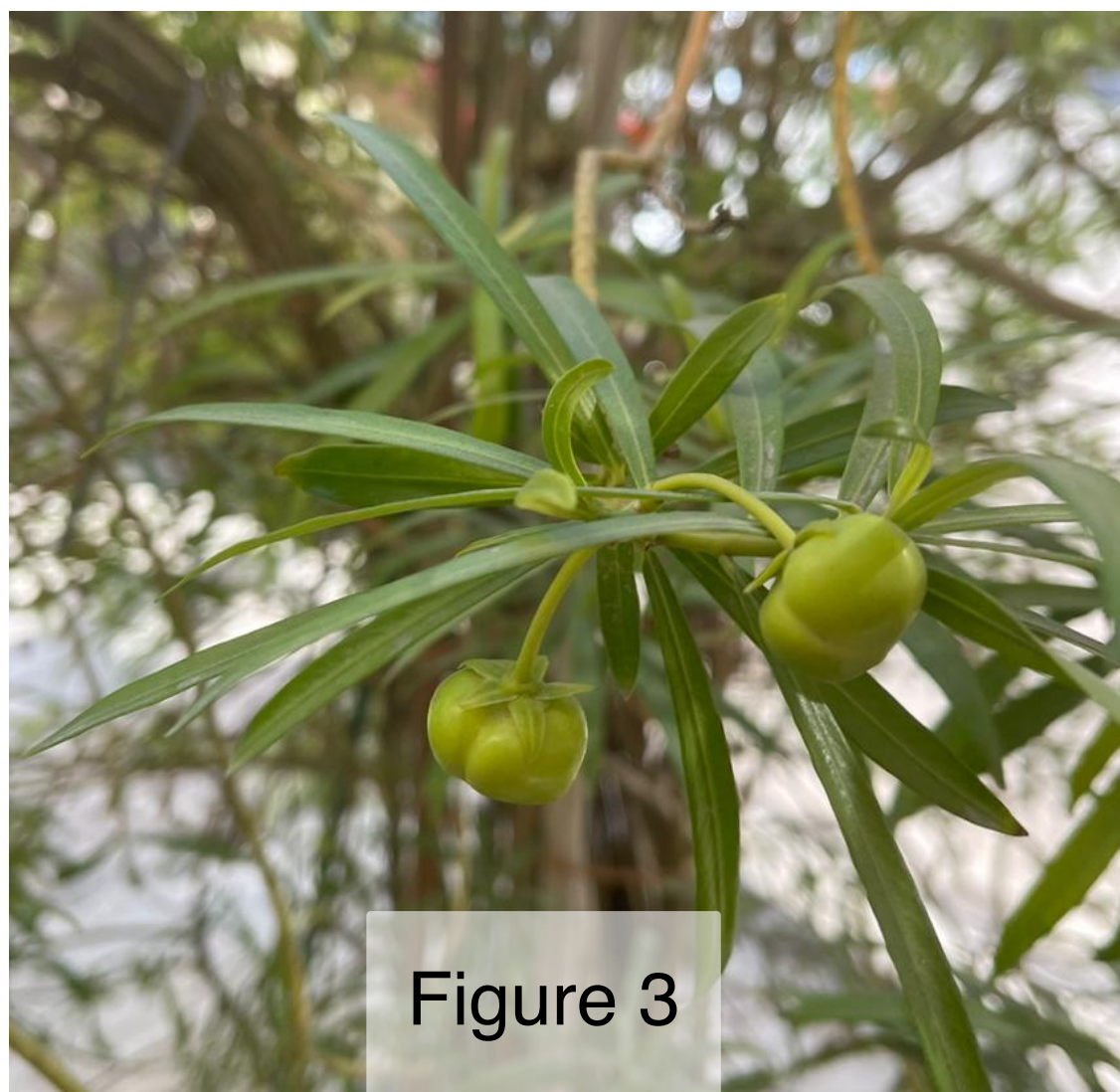


Figure 3

- Figure 1: Tree of yellow oleander, showing the yellow flower.
- Figure 2: Close view of the yellow oleander's fruit.
- Figure 3: Showing the fruit of yellow oleander.

Discussion

- Thevetia peruviana contains the cardiac glycoside, which causes digoxin-like toxicity.
- All the tree parts like flowers, leaves, fruits are poisonous, but seeds and roots are the most toxic parts⁽⁵⁾.
- Ingestion of these parts is potentially fatal and should be monitored closely for 24hrs even if no symptoms of toxicity, as the absorption kinetics is varied.
- Continuous ECG monitoring is necessary to detect arrhythmias ⁽⁶⁾
- The literature stated that ingestion of 2-3 fruits caused nausea, vomiting, diarrhea, and lethargy. ⁽⁷⁾

Mechanism of toxicity:

- Cardiac glycoside works by binding to sodium/potassium ATPase pump on the cardiac cells, which leads to increase calcium concentration.
- Resulting in increased contractility and myocardial automaticity^(1,2).
- The above explain the echocardiogram (ECG) changes following the intoxication. ^(1,11,12)
- Our case did not show any ECG changes upon admission nor upon discharge.

Management:

- In asymptomatic patients, supportive measures are the mainstay of management.
- Single-dose activated charcoal (SDAC) is frequently used if the patient presented within 1-2 hrs⁽⁶⁾.
- Multidose activated charcoal (MDAC) inhibits cardiac glycosides' absorption.
- In our patient, we did not give SDAC as she presented 5 hr. after the ingestion, neither MDAC as she remained asymptomatic throughout her hospital course.
- Digoxin-specific fragments could be given with a recommended initial dose of at least 800 mg ⁽¹⁴⁾.
- Atropine could be given in cases presenting with bradycardia. ^(1, 11, 13)
- if the patient has been asymptomatic and hemodynamically stable, looks well, and has a normal ECG 24 h after ingestion, it is likely safe to discharge him ⁽⁶⁾.
- In our case, the patient underwent only close observation and monitoring and had serial ECG, digoxin level testing, and electrolyte levels, which all were within normal limits.

Conclusion

- Although its highly toxic, Yellow oleander is commonly used as a home plant, and peoples might eat it as they thought its edible plants.
- Public health awareness about identifications of the plants and its toxicity should be raised.
- Our patient presented asymptotomatically after ingestion of 1 yellow oleander's fruit which is equal to 3.4 grams without the seeds and was discharged healthy with no complaints.
- We need more studies regarding the Yellow oleander's fruit toxic dose as we can't rely on the numbers of fruits where the sizes are varied.

References

1. Bandara V, Weinstein SA, White J, Eddleston M. A review of the natural history, toxinology, diagnosis and clinical management of Nerium oleander (common oleander) and Thevetia peruviana (yellow oleander) poisoning. Toxicon. 2010;56(3):273-81.
2. Dart RC. Medical toxicology / [edited by] Richard C. Dart ; authors E. Martin Caravati ... [et al.]. 3rd ed. ed. United States: Philadelphia : Lippincott, Williams & Wilkins, c2004.; 2004.
3. Fentanes E. Eating seeds from the 'be still' tree, yet having lucky nut poisoning: a case of acute yellow oleander poisoning. BMJ Case Rep. 2014;2014.
4. Eddleston M, Ariaratnam CA, Sjöström L, Jayalath S, Rajakanthan K, Rajapakse S, et al. Acute yellow oleander (Thevetia peruviana) poisoning: cardiac arrhythmias, electrolyte disturbances, and serum cardiac glycoside concentrations on presentation to hospital. Heart. 2000;83(3):301-6.
5. Anand S, Appaji S, R S. Suicide tree poisoning. International Journal of Contemporary Pediatrics. 2018;5:1690.
6. Rajapakse S. Management of yellow oleander poisoning. Clin Toxicol (Phila). 2009;47(3):206-12.
7. Samal K.K., Sahu HK, Gopalakrishnakone P. Clinico-pathological study of Thevetia peruviana (yellow oleander) poisoning. Journal of Wilderness Medicine. 1992;3(4):382-6.
8. González-Stuart A, Rivera JO. Yellow Oleander Seed, or "Codo de Fraile" (Thevetia spp.): A Review of Its Potential Toxicity as a Purported Weight-Loss Supplement. J Diet Suppl. 2018;15(3):352-64.
9. Osterloh J, Herold S, Pond S. Oleander interference in the digoxin radioimmunoassay in a fatal ingestion. Jama. 1982;247(11):1596-7.
10. D A, Pandit VR, Kadhiravan T, R S, Prakash Raju KNJ. Cardiac arrhythmias, electrolyte abnormalities and serum cardiac glycoside concentrations in yellow oleander (Cascabela thevetia) poisoning - a prospective study. Clin Toxicol (Phila). 2019;57(2):104-11.
11. Karthik G, Iyadurai R, Ralph R, Prakash V, Abhilash K, Sathyendra S, et al. Acute oleander poisoning: A study of clinical profile from a tertiary care center in South India. Journal of Family Medicine and Primary Care. 2020;9:136.
12. G B, A M, V.P. K. A STUDY OF CARDIAC ARRHYTHMIAS IN YELLOW OLEANDER SEED POISONING- AN OBSERVATIONAL STUDY IN A TERTIARY CARE CENTRE. Journal of Evidence Based Medicine and Healthcare. 2018;5:2100-4.
13. Abid Hossain M, Ahmed S, Shamsuzzaman NH, Islam AKMN, Rashid A, Rahim M, et al. Yellow oleander poisoning : a case report. Journal of Nepal Medical Association. 2003;41(142).
14. Eddleston M, Rajapakse S, Rajakanthan, Jayalath S, Sjöström L, Santharaj W, et al. Anti-digoxin Fab fragments in cardiotoxicity induced by ingestion of yellow oleander: a randomised controlled trial. Lancet. 2000;355(9208):967-72.

TYPE 2 BRUGADA PHENOCOPY DUE TO SUPRA-THERAPEUTIC PHENYTOIN LEVEL

Introduction

Brugada syndrome (BS) is a hereditary cardiac disease leading to sudden cardiac death. Brugada phenocopy (BrP) is an evolving term for Brugada like ECG patterns due to reversible causes. Type 2 Brugada pattern secondary to supratherapeutic phenytoin level is not previously described including its significance in terms of evaluation, disposition, and follow- up.

Case Presentation

A 29- year old male was brought after episodes of witnessed seizure with loss of consciousness and postictal confusion. He denied trauma, tongue bite, urinary, bowel incontinence, aura, chest pain, palpitations, or shortness of breath. He denied family history of cardiovascular diseases including sudden death. His physical exam, blood investigations, and CT head were unremarkable. The diagnosis of epilepsy was made. He was loaded with slow IV phenytoin 1500 mg (18.75mg/kg) infusion. After the infusion, patient developed severe dizziness, nystagmus, and limb ataxia. His blood sugar was 5.6 mmol/l. His ECG showed type 2 BrP (Figure 1,2). His phenytoin level was 127.6 umol/l (therapeutic range: 40-79 umol/l). A toxicology consult was made and he was admitted. His phenytoin level was 65.2 umol/l at 46 hours of observation. His symptoms were completely resolved with normal ECG (Figure 3).

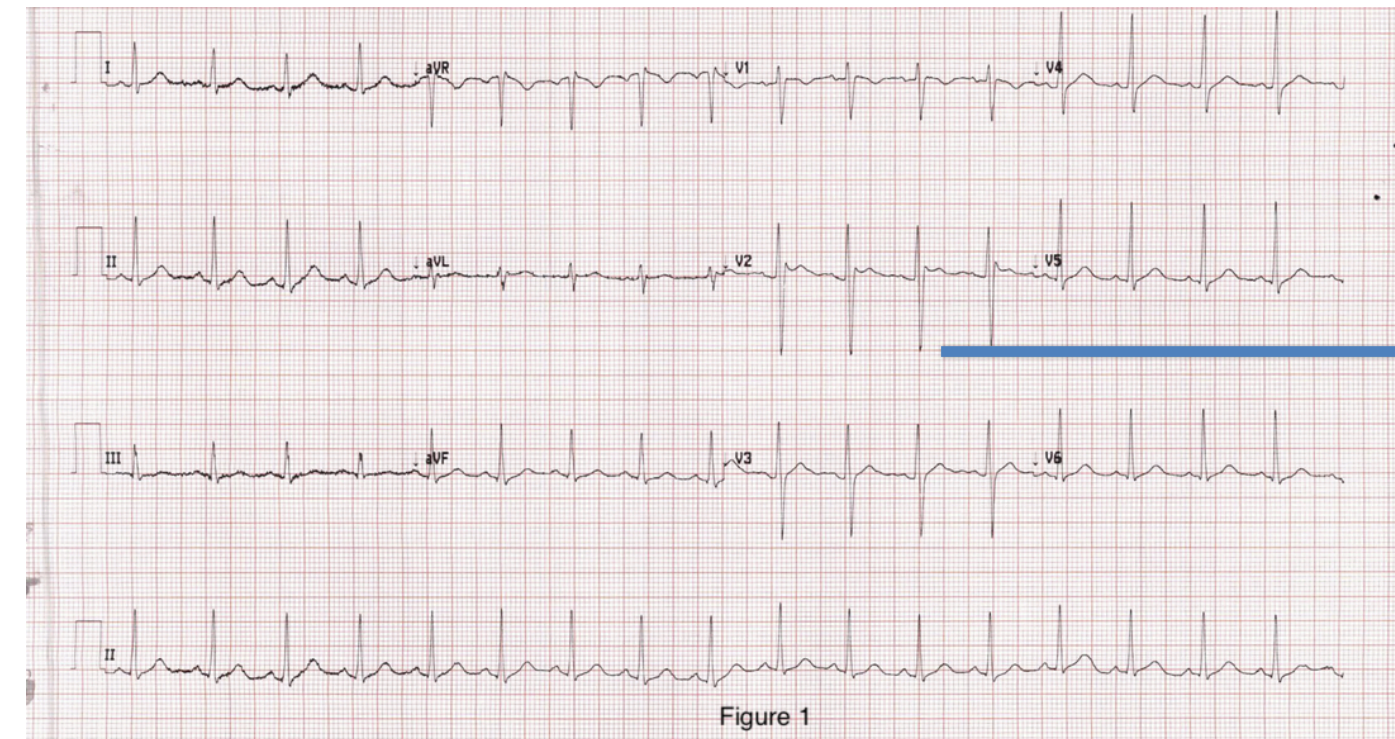
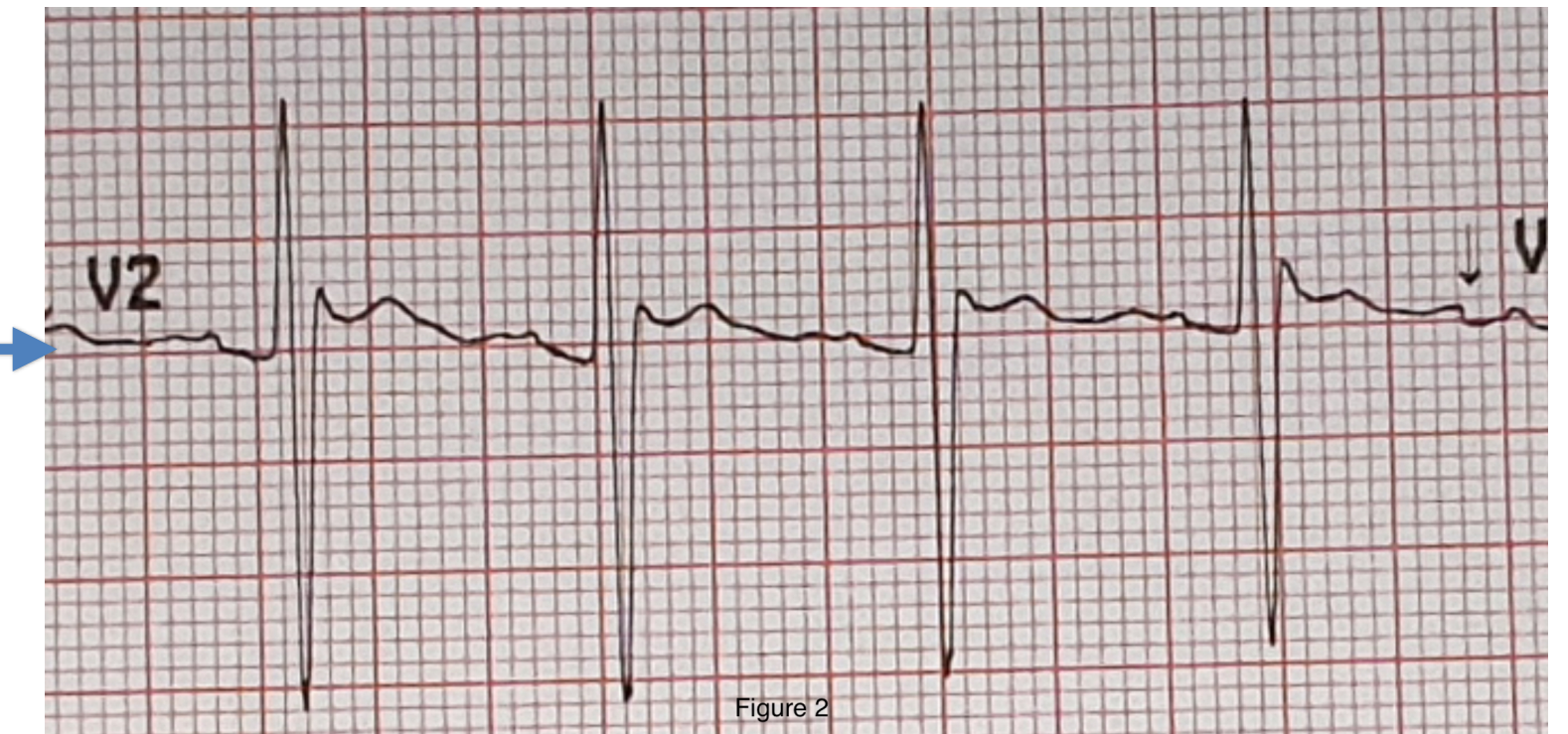


Figure 1 & 2 ECG showing type 2 Brugada pattern



Discussion and Learning Points

- ✓ This case illustrates the need for thorough history taking to exclude arrhythmia, syncope, or family history of sudden cardiac death in such cases.
- ✓ If history is concerning, patients need a cardiology follow-up to test for true BS including unmasking with sodium channel blocking agents.
- ✓ Repeat ECG on discharge is helpful to confirm the reversibility.
- ✓ To summarize, Brugada phenocopy can be observed in patients taking antiepileptic drugs, particularly with supratherapeutic levels.

References

1. Baranchuk A et al.: [Brugada phenocopy: new terminology and proposed classification](#). Ann Noninvasive Electrocardiol. 2012, 17:299-314.
2. Aloul B Al et al.: [Brugada pattern electrocardiogram associated with supratherapeutic phenytoin levels and the risk of sudden death.](#) . Pacing Clin Electrophysiol. 2007, 21:713-5.

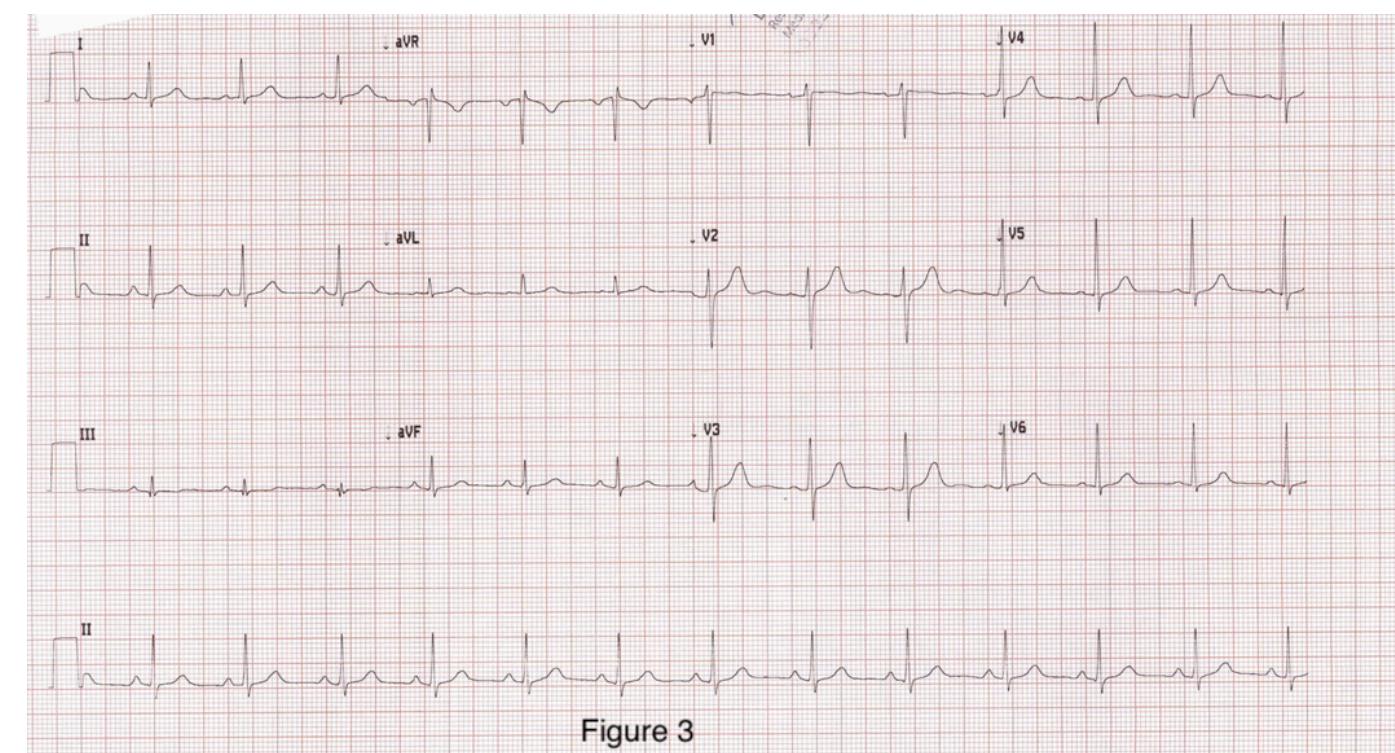


Figure 3 showing normal ECG with normal phenytoin level

SULTAN QABOOS UNIVERSITY
Sultan Qaboos University Hospital



MENATOX 2021

Alcohol Related Emergency Department Visits in Tertiary Hospital








Dr. Suad Al-Abri
Emergency Medicine Consultant &
Medical Toxicologist
Sultan Qaboos University Hospital
Muscat, Oman




1

Outline




- Introduction
- Methods
- Results
- Discussion
- Limitation
- Summary

2

Introduction

- WHO stated that alcohol lead to a 3 million deaths in 2016,
- Alcohol ranked as the fifth risk factor causing high rates of deaths and injuries in 2010 around the world

3

Some Statistics

- ▶ 3.3 million people world wide die from alcohol related illness p.a. (5.9%)
- ▶ Over 5% of annual burden of disease as DALYs is due to alcohol worldwide
- ▶ 25% of deaths in 20-39 yr olds world wide are attributable to ETOH
- ▶ ETOH is implicated in over 200 different health conditions
- ▶ 1 in 3 presentations to the ED in Australia are related to alcohol
- ▶ In 2010 estimates 3.5% Australian pop are dependent or use harmfully
- ▶ Australian men drink 20L per yr, women drink 9L per yr of pure alcohol




4

Ontario women and young adults experiencing greatest rise in ED visits for alcohol use

IC/ES

Researchers say interventions may be necessary to reduce harmful alcohol use, particularly in women and young adults.

Researchers looked at data for more than 765,000 emergency department (ED) visits in Ontario due to alcohol use between 2003 and 2016.

Overall, the increase in ED visits due to alcohol was **4.4x greater** than the increase in ED visits due to all other causes combined.



AMONG WOMEN
ED visits due to alcohol increased by 86%, compared to 53% in men.

AMONG YOUNG ADULTS
ED visits due to alcohol increased by 175%, the largest change observed.

Myran DT et al. CMAJ 2019.

ICES Data. Discovery. Better Health.
ices.on.ca









5

Alcohol-Related Emergency Department (ED) Encounters in California, 2008 – 2017

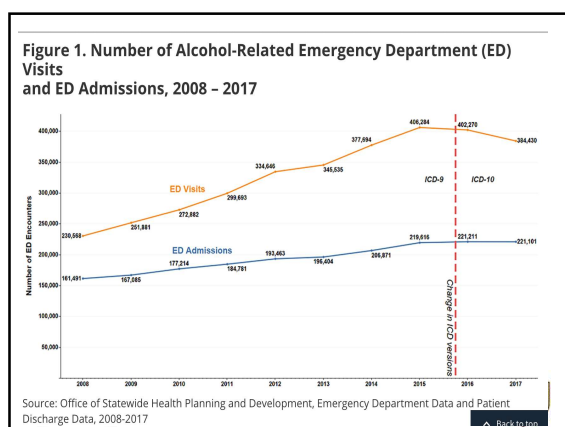
Data Pulse

There was a 66.7 percent increase in alcohol-related ED visits from 2008 to 2017, and a 36.9 percent increase for alcohol-related ED admissions.

A recent national study on alcohol-related visits to emergency departments (ED) found a nearly 50 percent increase in these visits between 2006 and 2014 (White et al., 2018.) In response to this report, the Office of Statewide Health Planning and Development (OSHPD) explored whether a similar trend was occurring in California and found a 66.7 percent increase in alcohol-related ED visits from 2008 to 2017.

6



7

Rationale

-This study is the first comprehensive study about alcohol related Emergency department visits in Oman.

- Serve as a good source for future prevention and health care and health education in the community.

8

Specific Objectives

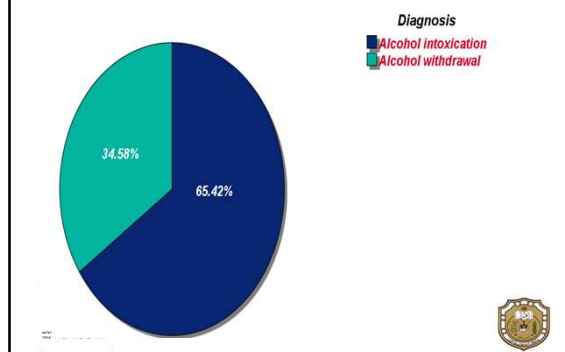
1. To describe the prevalence of alcohol intoxication and alcohol withdrawal related ED visits in patients seen at SQUH from 2016 to 2020.
2. To describe demographic, medical, psychiatric background, clinical, laboratory and outcome variables

9

Study design	A retrospective chart review study
Period	2016-2020
Ethical approval	No. MERC#2246 Obtained on 15 of September 2020
Setting	SQUH emergency department
Data source	- TrakCare system of SQUH - Hospital information system
Sample size	- 107 patients
Collected data	Demographic variables, medical background, alcohol intake history, clinical manifestation, diagnosis and outcome
Data analysis	SPSS program Statistical analysis: mean, percentages, range, chi square and pie chart

10

Results



11

Baseline characteristics

Demographics		
Age	Mean	42.53
	Range	21-75
Sex	Male	105 (98.1)
	Female	2 (1.9)
Nationality	Omani	101 (93.5)
	Non Omani	6 (5.6)

12

Baseline Characteristics

Social history	
Smoking	40 (37.4)
Drug abuse	31 (29.0)
Past medical history	
Diabetes	28 (26.2)
Hypertension	29 (27.1)
Psychiatric disease	37 (34.6)
Cardiac disease	24 (22.4)
Liver disease	44 (41.1)

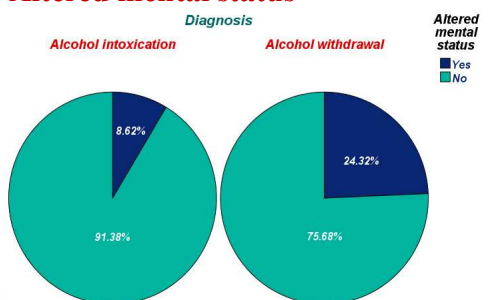
13

Clinical Presentations

Seizure	31 (29.0)
Shortness of breath	17 (15.9)
GI related symptoms	42 (39.3)
Clinical investigation (Mean)	
PH	7.29451
PCO2	42.10667
Lactate	6.57955
Potassium	4.4
HCO3	19.73864

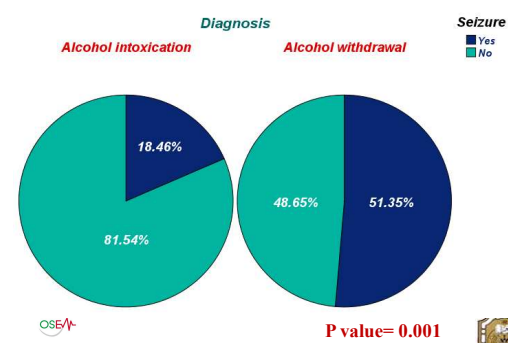
14

Altered mental status



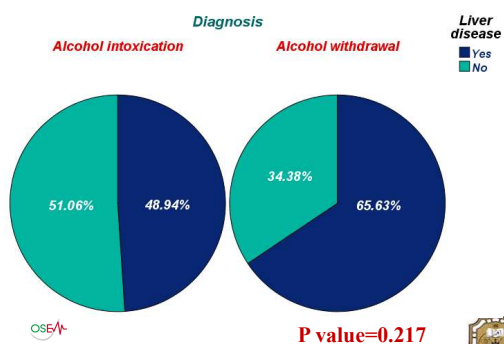
15

Seizure



16

Liver Disease

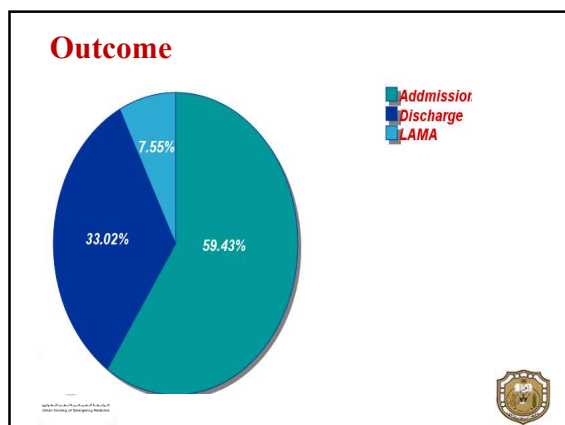


17

Management

Benzodiazepines	31 (29.0)
IV fluids	83 (77.6)
Antibiotics	15 (14.0)
Anticonvulsant	13 (12.1)
Naloxone	11 (10.3)
Intubation	2 (1.9)
Dialysis	3 (2.8)

18



19

Original Article

Trends in Alcohol-Related Emergency Department Visits in the United States: Results from the Nationwide Emergency Department Sample, 2006 to 2014

Aaron M. White, Megan E. Slater, Grace Ng, Ralph Hingson, Rosalind Breslow

First published: 02 January 2018
<https://doi.org/10.1111/acer.13559>
 Citations: 50

The annual percentage change in rates of all alcohol-related ED visits was larger for females than for males (5.3% vs. 4.0%).
Other drug involvement increased the likelihood of admission for inpatient treatment.

20

Alcohol related deaths

Prevalence=4.70%

	Patient#1	Patient #2	Patient#3	Patient #4	Patient#5
Age	38	58	56	46	75
Gender	Male	Male	Male	Female	Male
Diagnosis	Alcohol intoxication	Alcohol intoxication	Alcohol withdrawal	Alcohol withdrawal	Alcohol intoxication
Presentation	Cardiac disease GI disease Liver disease	Hypertension Cardiac disease Diabetes Renal disease	Diabetes Hypertension GI disease	GI disease	Liver disease Hypertension Cardiac disease GI disease
Cause of death	GI bleeding	Sepsis Multi organ failure	Sepsis Multi organ failure	Sepsis Multi organ failure	Multi organ failure

21

One in 20 Ontarians with 2 or more alcohol-related ED visits within 12 months is dead a year later

IC/ES

These visits present an opportunity to intervene with a high-risk population to reduce a substantial mortality burden.

Researchers identified 25,813 Ontarians aged 16 or older with at least 2 emergency department visits for mental and behavioural disorders related to alcohol in a 12-month period from 2010 to 2016 and determined mortality at 1 year.

- Among individuals, **66%** had 2 ED visits, **22%** had 3-4 visits and **12%** had 5 or more visits.
- Their overall death rate at 1 year was about **5%**, but for people with **5 or more ED visits**, it was nearly **10%**.
- At greatest risk were men aged 45-64 and those living in urban centres and in poorer neighbourhoods.

ICES Data. Discovery. Better Health.
[ices.on.ca](https://www.ices.on.ca)

camh

22

Limitations

- Small sample size
- Missing data
- Retrospective study

23

Summary

- Most of alcohol related ED visits in our study are alcohol intoxication
- Seizure is more significantly associated with alcohol withdrawal than alcohol intoxication
- High health care cost as Most of alcohol related ED visits end up with admission

24

Summary

- Most of alcohol related ED visits in our study are alcohol intoxication
- Seizure is more significantly associated with alcohol withdrawal than alcohol intoxication
- High health care cost as Most of alcohol related ED visits end up with admission



24

References

- 1. World Health Organization (2018) Global status report on alcohol and health 2018. Available at: <https://apps.who.int/iris/bitstream/handle/10665/274603/9789241565639-eng.pdf?ua=1>.
- 2. National Institute on Alcohol Abuse and Alcoholism (no date) NIH. ... Turning Discovery Into Health @ National Institute on Alcohol Abuse and Alcoholism Alcohol Facts and Statistics. Available at: <https://www.niaaa.nih.gov>.
- 3. Morgan, M. Y. (2015) Acute alcohol toxicity and withdrawal in the emergency room and medical admissions unit. Available at: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4953238/pdf/486.pdf>.
- 4. Sachdeva, A., Choudhary, M. and Chandra, M. (2015) 'Alcohol withdrawal syndrome: Benzodiazepines and beyond', *Journal of Clinical and Diagnostic Research, Journal of Clinical and Diagnostic Research*, pp. VE01–VE07. doi: 10.7860/JCDR/2015/13407.6538.



25

References

- 5. Becker, H. C. (1998) 'Kindling in alcohol withdrawal', *Alcohol health and research world*, pp. 25–33.
- 6. Rehm, J. (2011) The Risks Associated With Alcohol Use and Alcoholism. Available at: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3307043/pdf/arrh-34-2-135.pdf>.
- 7. Sachdeva, A., Choudhary, M. and Chandra, M. (2015) 'Alcohol withdrawal syndrome: Benzodiazepines and beyond', *Journal of Clinical and Diagnostic Research, Journal of Clinical and Diagnostic Research*, pp. VE01–VE07. doi: 10.7860/JCDR/2015/13407.6538.
- 8. Shield, K. D., Pamy, C. and Rehm, J. (2013) Chronic Diseases and Conditions Related to Alcohol Use 155 Chronic Diseases and Conditions Related to Alcohol Use. Available at: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3908707/pdf/arcr-35-2-155.pdf>.
- 9. Thavorncharoensap, M. et al. (2009) 'The economic impact of alcohol consumption: A systematic review', *Substance Abuse: Treatment, Prevention, and Policy*, p. 11. doi: 10.1186/1747-597X-4-20.
- 10. Uko Jaius, E. et al. (2017) 'Effect of Alcohol Consumption on Students' Academic Performance in Tertiary Institution', 6(2), pp. 66–78. Available at: www.ijsm.humanjournals.com www.ijsm.humanjournals.com.
- 11. Willmore, J. et al. (2017) 'The burden of alcohol-related morbidity and mortality in Ottawa, Canada', *PloS ONE: Public Library of Science*, 12(9), p. 19. doi: 10.1371/journal.pone.0185457.



26



27



28

Winning A Battle Against A Death Stalker:

Apistobuthus pterygocercus envenomation induced cardiomyopathy, Case report



وزارة الصحة
Ministry of Health

Adawiya Al Jamei^{1,2}, Hamood Al Shueili^{1,2}, Badria Al Hatali³

¹Nizwa Hospital, Nizwa, Oman. ²Ministry of Health, Muscat, Oman. ³Poison Control Section, Ministry of Health, Muscat, Oman



INTRODUCTION

Scorpion sting is a common presentation in Oman. *Apistobuthus* is a genus of scorpions in the family Buthidae, which can be fatal in children. It causes excessive catecholamine release, leading to myocardial sensitivity and cardiomyopathy. We report a confirmed case of *Apistobuthus pterygocercus* (Fig 1) envenomation induced cardiomyopathy



CASE REPORT

A 6 -year-old child presented at primary hospital 10 minutes after the sting at his right foot dorsum. He developed vomiting, sweating, confusion and irritability. His heart rate was 180 beats/min, and blood pressure 150/85mmHg with saturation of 99% in room air. Child was treated as anaphylaxis and transferred to a secondary care hospital. Twelve hours later, he was noted to be agitated, confused, hallucinating, tachypneic with saturation <80% in room air. His heart rate was 160 beat/min and blood pressure was 97/50mmhg. He required 4 doses of Saudi scorpion antivenom, 5 mL each and repeated doses of benzodiazepines to control his agitation. Subsequently, he developed evidence of cardiomyopathy with troponin of 2208 pg/mL, BNP of >35000 pg/ml, creatinine kinase of 3334 u/l. His echocardiography showed severe dilated cardiomyopathy with EF <30%. Child was intubated 22 hours after the sting and kept on adrenaline, dobutamine and frusemide infusion. His condition improved over time and extubated 3 days later. He was discharge at day 6 of admission with normal echocardiography



DISCUSSION

Scorpions envenomation can cause serious complications like cardiomyopathy. Availability of antivenom is crucial in the management



Figure 1, *Apistobuthus pterygocercus*

Introduction

- ❖ **Castor beans** comes from castor oil plant (*Ricinus communis*)
- ❖ The seed is only toxic if the outer shell is broken or chewed
- ❖ **Ricin** is contained in the bean pulp following the separation of the oil from the beans
- ❖ No ricin remains in the oil as it is inactivated during extraction if done under heated conditions

Cases presentation

We report **two patients**, A 27 years old man and a 26 years old with no previous medical illness, presented to two different emergency departments, with **severe abdominal pain and diarrhea** which was initially watery very frequent, then became bloody. The first patient got it from local herbal store for constipation, while the second patient got it as a prank to induce diarrhea from his friend. The first patient developed the symptoms after 1 hour while the second patient had it within 30 minutes of ingestion. On clinical examination, both were tired and dehydrated, otherwise vitals and systematic examination unremarkable. Initial investigations were normal including ECG. Both patients were admitted for 48 hours for supportive care and were discharged on good conditions



Figure 1: Castor beans, retrieved from the first patient

Traditional herbal shops visits

- ❖ We did random visits for **five different herbal shops in Ibri** asking for laxatives beans.
- ❖ Three of the shops **recommended** using it as laxatives with dose ranging from 1/3 to one bean.
- ❖ No special precautions or warning were given

Discussion

- ❖ Castor beans toxicity had been known since long, but the practices of using these beans among the traditional and herbal stores as means of laxatives should be investigated
- ❖ The deadly nature of pure ricin has led to intensive awareness campaigns and research to even develop an antiricin vaccine, while ingestion of castor beans harness lesser toxicity and mortality rates of 8.1 % if untreated and 0.4% if treated

- ❖ We discovered that most of the products in herbal shops are not regulated and based on the individual practices and knowledge

Conclusion

- ❖ **Castor beans** are used commonly in traditional herbal shops as laxatives with no special precaution. Health awareness is needed about the beans toxicity.

references

- ❖ Al-Tamimi FA, Hegazi AE. A case of castor bean poisoning. Sultan Qaboos University Medical Journal. 2008 Mar;8(1):83.
- ❖ de Haan P, Reidinga-Saenen LM, Korporaal-Heijman JA. Intoxication by ingestion of castor beans. NETHERLANDS JOURNAL OF CRITICAL CARE. 2016 Mar 1;24(2):20-2.
- ❖ Despott E, Cachia MJ. A case of accidental ricin poisoning.
- ❖ Doan LG. Ricin: mechanism of toxicity, clinical manifestations, and vaccine development. A review. Journal of Toxicology: Clinical Toxicology. 2004 Jan 1;42(2):201-8.
- ❖ Assiri AS. Ricin poisoning causing death after ingestion of herbal medicine. Annals of Saudi medicine. 2012 May;32(3):315-7.
- ❖ Figure 1, courtesy to Dr Waleed Alsukaiti MD

Waleed.S.Alsukaiti MD (1), Ayman. J. Mahmoud MMBS (1), Salman. A MBBS (1)

1. Department of Emergency Medicine, Ibri Hospital, Sultanate of Oman

Introduction

- ❖ **Buthidae** family of scorpions are abundant in the Middle East and Oman, especially the genus *Androctonus* and *Leiurus* *Quinquestriatus*, one of the most dangerous scorpions worldwide.
- ❖ ***Leiurus quinquestriatus***, also known as the **deathstalker**, is a large venomous species of scorpions measuring up to 110 mm in length and weigh around 1-2.5 g. They are yellowish with brown spots on the metasomal segment V and sometimes on the carapace and termites.

Case presentation

- ❖ We are reporting, a twenty-one-year-old male presented with pain in the right foot after a scorpion sting; he developed a **tonic-clonic movement** associated with facial muscle twitching, **clonus** of the lower limbs and **inability to speak** during the whole stay in the emergency department. His brother described the scorpion, which was matching the **deathstalker scorpion**. The jerky movements stopped after administering 5 mg diazepam, but the clonus and inability to speak continued. He had facial and tongue fasciculations with remarkable clonus on examination. A **polyvalent scorpion antivenom** with a dose of 2 vials diluted in 50 ml 0.45 NS administered. The patient had a full recovery during admission.



Figures 1, *Leiurus quinquestriatus*, *The deathstalker scorpion*

Discussion

- ❖ The **lethal dose (LD50)** of the *leiurus* *quinquestriatus* is **0.25**, which is the lowest LD50 among other scorpion species and reflects the highest lethality
- ❖ The massive surge of neurotransmitters and vasodilators after the sting affects multiple organs and manifest as autonomic, sympathetic and neuromuscular symptoms. Our patient's presentation is considered serious with systemic manifestations.
- ❖ **Motor aphasia** is the inability to produce speech with intact comprehension

Conclusion

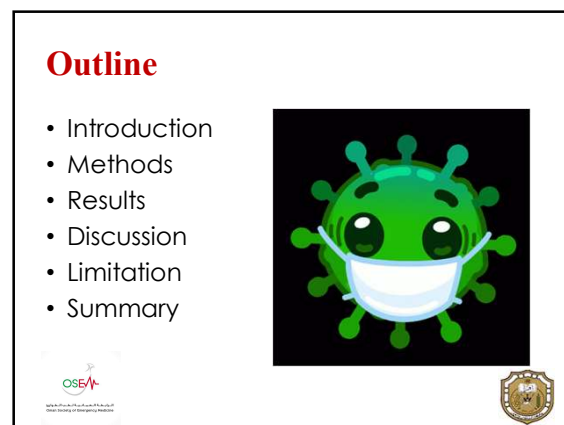
- ❖ This case highlights the severe clinical manifestations due to a sting from the ***Leiurus quinquestriatus*** scorpion. The early recognition and treatment with benzodiazepines and antivenom may have lead to complete recovery.

References

1. Isbister, G. K., & Bawaskar, H. S. (2014). Scorpion envenomation. *New England Journal of Medicine*, 371(5), 457-463.
2. Katsas, D., Stathi, I., & Fet, V. (2008). Scorpions of the Eastern Mediterranean. *Advances in Arachnology and Developmental Biology. Papers dedicated to Professor Božidar PM Čurčić*. Belgrade-Vienna-Sofia, 209-246.
3. Lall SB, Al-Wahaibi SS, Al-Riyami MM, Al-Kharusi K. Profile of acute poisoning cases presenting to health centres and hospitals in Oman. *East Mediterr Health J* 2003 Sep; 9(5-6):944-954.
4. Ceceli, A. and J. Horsfield 2012. "*Leiurus quinquestriatus*" (On-line). *Animal Diversity Web*. Accessed June 05, 2020 at https://animaldiversity.org/accounts/Leiurus_quinquestriatus/
5. Quintero-Hernández, V., Jiménez-Vargas, J. M., Gurrola, G. B., Valdivia, H. H., & Possani, L. D. (2013). Scorpion venom components that affect ion-channels function. *Toxicon*, 76, 328-342.
6. Mullen, G. R., & Sissom, W. D. (2019). Scorpions (Scorpiones). *Medical and Veterinary Entomology*, 489-504. doi:10.1016/b978-0-12-814043-7.00023-6
7. Kshirsagar, V. Y., Ahmed, M., & Colaco, S. M. (2012). Motor aphasia: a rare complication of scorpion sting. *Journal of pediatric neurosciences*, 7(3), 231.
8. Al Abri, S., Al Rumhi, M., Al Mahruqi, G., & Shakir, A. S. (2019). Scorpion Sting Management at Tertiary and Secondary Care Emergency Departments. *Oman medical journal*, 34(1), 9.
9. Bonilha, L., Cendes, F., Ghizoni, E., Vieira, R. J., & Li, L. M. (2004). Epilepsy due to a destructive brain lesion caused by a scorpion sting. *Archives of neurology*, 61(8), 1294-1296.
10. Jarrar, B. M., & Al-Rowaily, M. A. (2008). Epidemiological aspects of scorpion stings in Al-Jouf province, Saudi Arabia. *Annals of Saudi medicine*, 28(3), 183-187.
11. Cavari, Y., Lazar, I., Shelef, I., & Sofer, S. (2013). Lethal brain edema, shock, and coagulopathy after scorpion envenomation. *Wilderness & environmental medicine*, 24(1), 23-27.
12. National Management Guidelines of Poisoning, Third Edition- 2018, Sultanate of Oman.
13. Deathstalker scorpion, *Leiurus quinquestriatus*, figures 1, courtesy to Mr. Ahmed Albusaidi



1



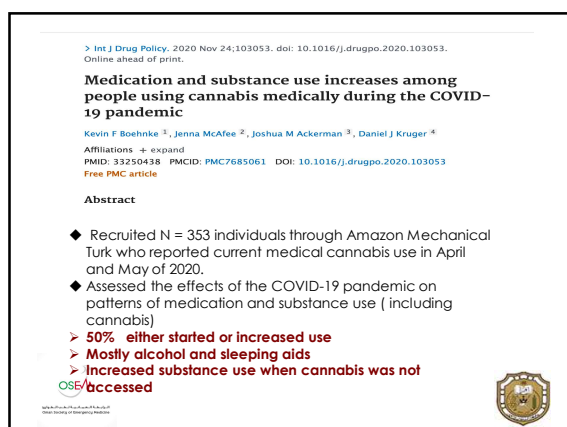
2



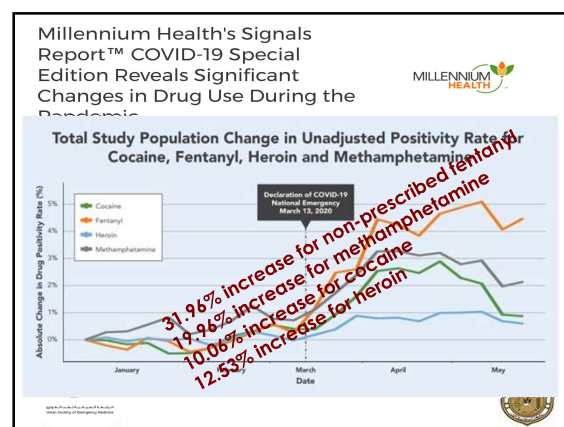
3



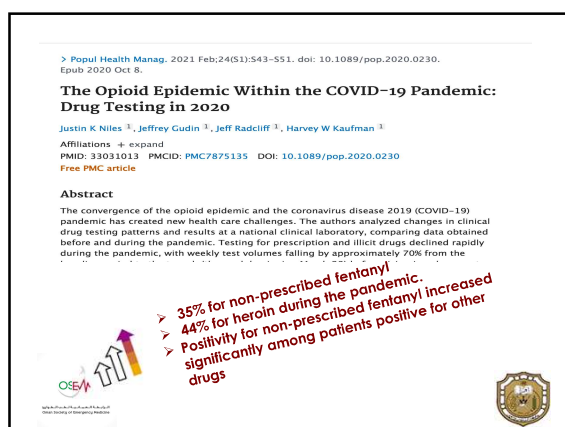
4



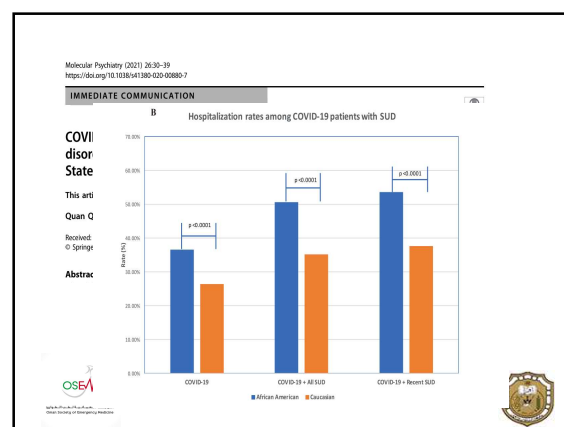
5



6



7



8

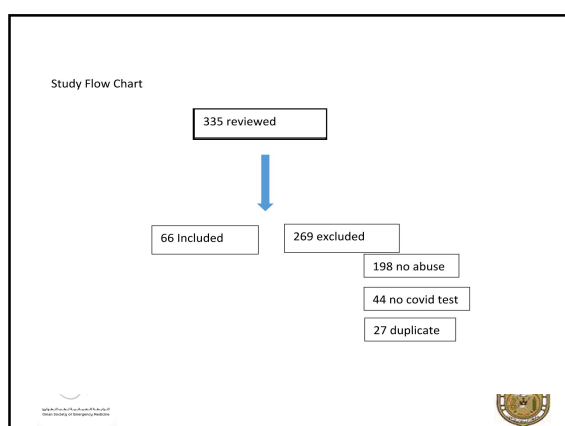
Specific Objectives

1. To describe the prevalence of Covid-19 in drug abusers and outcome in patients presented to tertiary care hospital emergency department

9

Study design	A retrospective chart review study
Period	March 2012 to January 2021
Ethical approval	No. MERC#2384
Setting	SQUH emergency department patients who had drug abuse screening
Data source	- Hospital information system
Sample size	- 335 patients (only 66 included) - Patients were included if they are drug abuser and covid-19 swab was taken for them.
Data analysis	SPSS program Statistical analysis: mean, percentages, range, chi square and pie chart

10



11

Baseline characteristics

Demographics		
Age	Mean	41.5
	Range	21–62
Sex	Male	65 (98.5 %)
	Female	1 (1.5 %)
Nationality	Omani	65 (98.5%)
	Non Omani	1 (1.5%)

12

Baseline Characteristics	
Social history	
Smoking	37 (56%)
Alcohol	40 (60.6 %)
Past medical history	
Diabetes	3 (4.5 %)
Hypertension	2 (3%)
Psychiatric disease	24(36.4%)
Infective Endocarditis	10 (15%)


13

Clinical Presentations	
Respiratory Symptoms	66 (100 %)
Urine drug abuse Results	
Morphine(+ heroin)	55(83.3%)
Benzodiazepines	15 (22.7%)
THC	11 (16.6%)
Methamphetamine(+amph)	17 (25.7%)

14

Covid Status


- Covid was positive in 7.6% (5/66)
- Covid negative 92.4% (61/66)



15

Disposition


- 22.7 % (15/66) patients discharged from ED
- 77.2 % (51/66) admitted to hospital



16

Mortality

- Mortality was 10.6 % (7/66) in general
- 40% mortality (2/5) in Covid positive drug abuser.
- Smoking as risk factor in 57% (4/7)



17

Mortality

Chinese Center for Disease Control and Prevention

- COVID-19 has a case fatality rate of 6.3% for individuals with chronic respiratory disease,
- Compared with 2.3% overall
- Chronic smokers and SUDs worsen prognosis with other coronaviruses

18

Limitations

- Small sample size
- Retrospective study
- No control group



19

Summary

- Prevalence of covid-19 in our drug abusers population was slightly high (10%);
- The mortality rate of Covid positive drug abuser was high
- Further studies are required to determine factors that would predict a worse prognosis in Covid positive drug abuser



20

References

1. Al Wahaibi, N., Al Lawati, A., Al Ruqeshy, F., Al Khatri, A., Al-Farsi, Y., Juma, T. M. A., Al Hinafi, F., Al-Sibani, N., Mahadevan, S. and Al-Adawi, S. (2019) The characteristics and patterns of utilization of healthcare services among Omanis with substance use disorders attending therapy for cessation, PLoS ONE, 14(1) <https://doi.org/10.1371/journal.pone.0210532>.
2. Al-Hinafi, H., Al-Busaidi, I., Al-Farsi, B. and Al-Saidi, Y. (2021) The Prevalence of Substance Misuse and its Effects among Omani College Students: A Cross-sectional Study, Oman Medical Journal, 36(1)
3. Czeisler, M. É. (2020) Mental Health, Substance Use, and Suicidal Ideation During the COVID-19 Pandemic — United States, June 24–30, 2020, MMWR. Morbidity and Mortality Weekly Report, 69. <https://doi.org/10.15585/mmwr.mm6932a1>.
4. LC, M. H. (no date) Millennium Health's Signals Report™ COVID-19 Special Edition Reveals Significant Changes in Drug Use During the Pandemic. Available at: <https://www.prnewswire.com/news-releases/millennium-healths-signals-report-covid-19-special-edition-reveals-significant-changes-in-drug-use-during-the-pandemic-301089684.html> (Accessed: 18 March 2021).



21

References

5. The Opioid Epidemic Within the COVID-19 Pandemic: Drug Testing in 2020 | Population Health Management (no date). Available at: <https://www.liebertpub.com/doi/10.1089/POP.2020.0230> (Accessed: 18 March 2021).
6. Volkow, D. N. (2020) New Evidence on Substance Use Disorders and COVID-19 Susceptibility, National Institutes of Health (NIH), October. Available at: <https://www.drugabuse.gov/about-nida/noras-blog/2020/10/new-evidence-substance-use-disorders-covid-19-susceptibility>.
7. Volkow, N. D. (2020) Collision of the COVID-19 and Addiction Epidemics, Annals of internal medicine, 173(1): 61–62. <https://doi.org/10.7326/M20-1212>.
8. Wang, Q. Q., Kaelber, D. C., Xu, R. and Volkow, N. D. (2021) COVID-19 risk and outcomes in patients with substance use disorders: analyses from electronic health records in the United States, Molecular Psychiatry, Springer US, 26(1): 30



22



23

Massive Pulmonary Hemorrhage & Cardiac Arrest with Good Survival Outcome After Chlorpyrifos Poisoning Treated with Standard Treatment & Fresh Frozen Plasma and High-Frequency Oscillatory Ventilation



Prem Kumar¹, Zahraa Al Lawati¹, Raghad Abdwani¹, Suad Al Abri², Nagaram Dhande¹, Ahmed Idress³, Hiam³
(1) Pediatric Intensive care (PICU) & (2) Emergency Department (ED) at Sultan Qaboos University Hospital, Muscat, Oman

Background

Significant organophosphorus poisoning is uncommon in children in our region. Chlorpyrifos is a chlorinated organophosphate with significant toxicity and is commonly used as an anti-termite treatment. It comes in highly concentrated liquids making even a small amount of accidental ingestion significant in children.

Case Presentation

7 years old girl with Trisomy 21, presented to the ED after 30 minutes from chlorpyrifos liquid ingestion, stored in a juice bottle. Initial assessment revealed nausea on history and pinpoint pupils with normal vitals and systemic examination. Within 30 minutes of ingesting activated charcoal, she started vomiting and rapidly became unresponsive.

Management

The patient was immediately intubated and ventilated, followed by Atropine and Pralidoxime bolus and infusion. She was then admitted to PICU and developed severe bronchorrhea and pulmonary hemorrhage within 1 hour from admission to PICU; this led to cardiac arrest secondary to hypoxia, requiring CPR for 5 minutes. She also developed hypotension, requiring high doses of inotropes. Due to pulmonary hemorrhage, increase Oxygen requirement, and severe bronchorrhea, she was put on high-frequency oscillatory ventilation (HFOV) for 4 days, in addition to IV factor VIIa and fresh frozen plasma (FFP) during the active process of pulmonary hemorrhage. Atropine infusion lasted for 3 days and Pralidoxime was continued for 5 days.

Complications

Patient developed Atropine toxicity within 18 hours from the infusion, manifested as tachycardia, dry mucosa, and hyperthermia managed by tapering the infusion rate and active cooling, symptoms resolved within 24 hours. As a known complication of Chlorpyrifos, she also developed acute kidney injury secondary to rhabdomyolysis treated supportively.

Outcome

After 5 days of PICU admission, she was extubated successfully and, within 10 days, was transferred out of PICU then was discharged home without any neurological deficit.

Key Point

- Activated charcoal can be considered within 1 hour of toxin ingestion if the patient is conscious, cooperative, and has intact airway reflexes
- The key factor in determining outcomes is the timing of Pralidoxime administration.
- A high-dose regimen of Pralidoxime reduces the need for intubation, duration of mechanical ventilation, and doses of atropine.
- Atropine toxicity should be considered if the patient developed dilated pupils, hyperthermia, and tachycardia.
- Pulmonary hemorrhage should be managed with blood products including FFP, factor VIIa, and high-frequency oscillatory ventilation.

Disclosure

All authors have nothing to disclose

Reference

- (1) Worek F; Horst Thiermann H; &Wille T. (2020,June). Organophosphorus compounds and oximes: a critical review. Archives of Toxicology (VOL.94, pp.2275–2292).
- (2) Bajracharya SR; Prasad PN;& Ghimire R. (2016,Sept). Management of Organophosphorus Poisoning. J Nepal Health Res Counc (Vol.14,pp.131-8)
- (3) Eddleston M; Buckley NA; Eyer P; &Dawson AH. (2008).Management of acute organophosphorus pesticide poisoning. Lancet (vol.371.pp597–607)

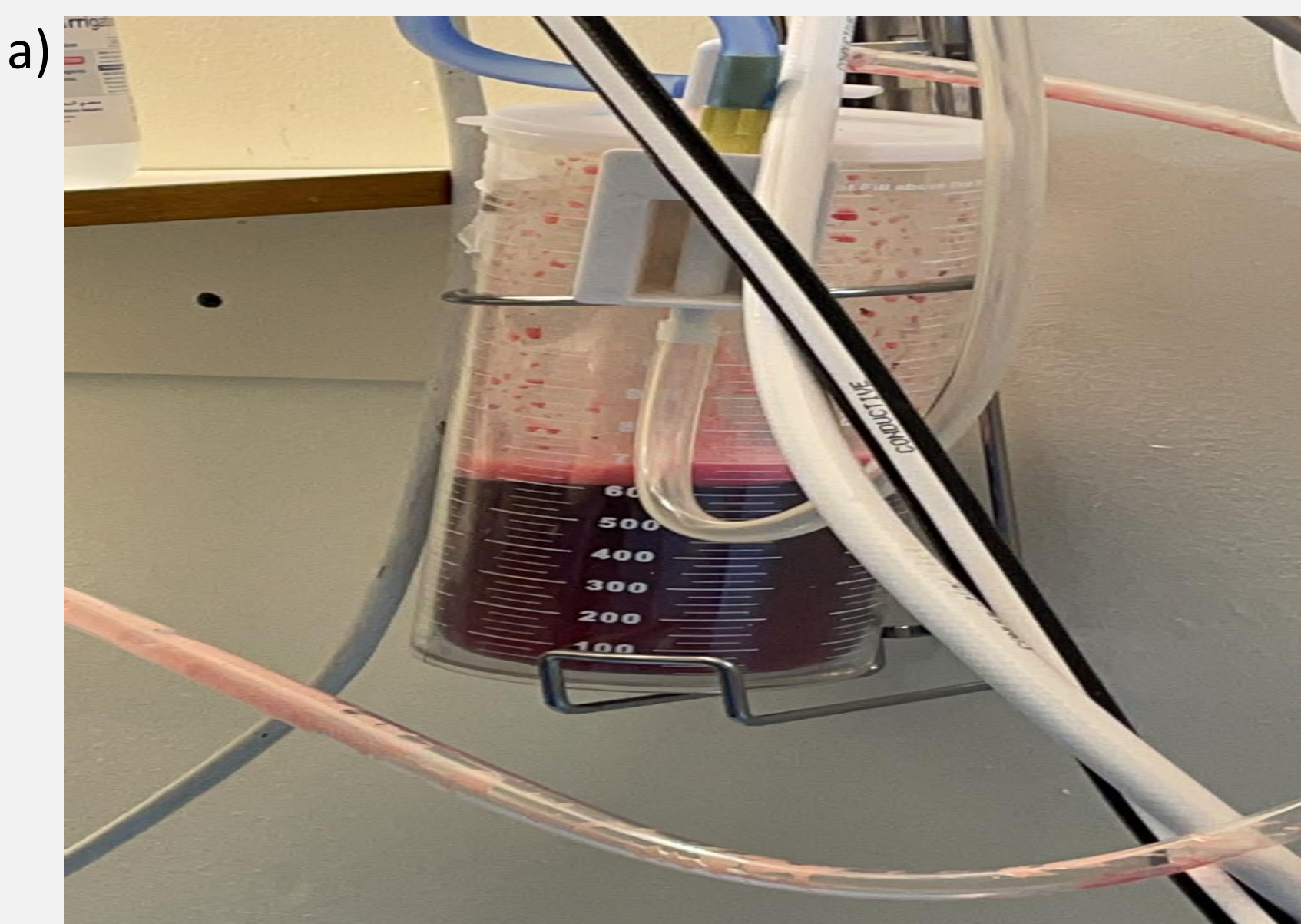


Figure a. Pulmonary Hemorrhage



Figure b. CXR after pulmonary hemorrhage

BITTER CUCUMBER TOXICITY

DR RANA J H HUSSEIN (MBBS, MRCEM, CABEM); MANAA A VINCENT(PharmD)

INTRODUCTION

There is a growing trend for traditional medicine across the globe. Many people prefer to take herbal medicines and remedies instead of pharmaceutical products. *Citrullus colocynthis* (bitter cucumber), is used by diabetic patients as a hypoglycaemic agent, but it has been reported to cause gastrointestinal disorders in some patients. In this case report we highlight the toxicity caused by *Citrullus colocynthis* (bitter cucumber).

DISCUSSION

Citrullus colocynthis, from the family Cucurbitaceae, is popularly named bitter apple or bitter cucumber in English and called Kadu Hanzal (bitter ground) in Persian. It is believed to have many therapeutic effects as an antidiabetic, antihyperlipidemic, laxative, anti-inflammatory, analgesic, antibacterial, and antifungal. But it can also cause disturbing side effects as diarrhoea, haematochezia, nephrosis, vomiting, and liver impairment. *C. colocynthis* is one of the toxic plants which may induce liver necrosis and impairment. Its medicinal benefit as a laxative was reported back in the 19th Century. Because of its toxicity it is been replaced by less toxic laxatives, although its use in homeopathic medicine still persists. The active components of the plant are colocynthin, colocynthidin, elaterin, pectin and albuminoid. The glucoside, colocynthin possess the laxative properties and may result in toxic bloody colitis. There is not much literature available on bitter cucumber toxicity. The few case studies that had been published stated the presence of bloody or non-bloody diarrhoea as the hallmark of the toxicity, whereas the colonoscopy studies showed congested mucosa and exudates but no ulcers. All those abnormal findings resolve in 14 days.

CASE HISTORY

A 47 year old Egyptian male presented to the Emergency Department with abdominal pain and bloody diarrhoea after consuming bitter cucumber juice for weight loss. He had 30 episodes of acute diarrhoea associated with abdominal pain. The diarrhoea was watery at first but became bloody in the course of his illness. He has a past history of peptic ulcer disease. His vitals were BP: 150/84 mm of Hg, PR: 88 bpm, Temperature: 36.9 C, RR: 18/min. Examination showed mild tenderness in the epigastrium, no guarding. He had no jaundice or pallor. Proctoscopy was performed and showed no anal fissures, no external or internal haemorrhoids, no blood in the rectum and the distal rectal mucosa was found to be normal. He was started on conservative management (antibiotic, proton pump inhibitors and hydration). Blood tests for CBC, kidney and liver functions, lipase, venous blood gas, coagulation profile were all within normal limits. Stool occult blood test was negative. In view of the profuse haemorrhagic diarrhoea, gastroenterologist was consulted and he advised to admit the patient overnight for observation and possible colonoscopy/endoscopy in the morning. Patient was put on symptomatic management and close monitoring for hemodynamic instability. The next day, the frequency of diarrhoea had decreased and the bleeding had stopped. The patient was feeling better. No colonoscopy was performed and the patient was discharged on PPI and antibiotics. He was given an urgent appointment for follow up in the Gastroenterology clinic.

CONCLUSION

C. colocynthis may have multiple therapeutic benefits, but it does cause significant adverse effects too. It can lead to hepatocyte necrosis and fibrosis. Further studies are necessary to understand the nature of its toxicity.

REFERENCES

- 1- The Clinical Investigation of *Citrullus colocynthis* (L.) Schrad Fruit in Treatment of Type II Diabetic Patients: A Randomized, Double Blind, Placebo-controlled Clinical Trial.
H. Fallah Huseini^{1*}, F. Darvishzadeh², R. Heshmat², Z. Jafariazar³, Mohsin Raza⁴ and B. Larijani²
- 2- The Toxic Effect of Alcoholic Extract of *Citrullus colocynthis* on Rat Liver
Farzaneh Dehghani and Mohammad Reza Panjehshahin
- 3- Case Report: *Citrullus colocynthis* as the Cause of Acute Rectorrhagia
Hamid Reza Javadzadeh,¹ Amir Davoudi,¹ Farnoush Davoudi,² Ghasem Valizadegan,³ Hasan Goodarzi,¹ Sadrollah Mahmoodi,¹ Mohammad Reza Ghane,¹ and Mehrdad Faraji¹
- 4- <http://www.floraofqatar.com> (Fig 1)



Fig 1

NONE OF THE AUTHORS HAVE
ANY DISCLOSURE TO DECLARE

Contact :
ranajaffer1979@yahoo.com

PEGANUM HARMALA INTOXICATION: THE TRADITIONAL HERBAL ANXIOLYTIC

DR RANA J H HUSSEIN (MBBS, MRCEM, CABEM); DR HARIS IFTIKHAR (MBBS, MRCEM)

BACKGROUND

Peganum harmala is a perennial plant that grows in the Mediterranean region, Middle East and North Africa. Popularly known as Harmal or Syrian Rue. It is used as an anxiolytic, mood enhancer, aphrodisiac, sedative, antibacterial and abortifacient. It causes toxic effects affecting the gastrointestinal, nervous and cardiovascular systems. There are limited studies delineating the toxicity caused by P. harmala. We are reporting an interesting case of toxicity induced by ingestion of powdered P. harmala. Our aim from this case report is to highlight the potential adverse effects caused by this herbal traditional remedy.

DISCUSSION

P harmala is popularly used in Turkey and Iran for treating depression and anxiety. Nowadays it is consumed as a psychoactive substance in Northern America and Europe, in the form of tea. All parts of the plant are toxic especially the roots and seeds. It contains active compounds as b-Carboline (Harmine, Harmaline & Tetrahydroharmine) and Quinazoline alkaloids (Vasicine and Vasicinone). Harmine and Harmaline are reversible monoamine oxidase inhibitors (MAO-A). Whereas Tetrahydroharmine inhibits serotonin uptake. Hence it has the potential to cause hypertensive crises and serotonin syndrome, respectively. Gastrointestinal symptoms and hallucinations are common symptoms experienced by majority of patients. The diagnosis of the intoxication is based on the recognition of the plant and the identification of alkaloids by either liquid/gas chromatography or mass spectrometry. Rapid and adequate therapeutic support is important, there is no antidote available. Gastrointestinal decontamination by gastric lavage and activated charcoal may be considered in severe toxicity with early presentation. Close observation for cardiac arrhythmias, hemodynamic instability and seizures is essential.

CASE HISTORY

32-year-old female patient with no significant comorbidities presented to the ED with complaints of vomiting, epigastric pain and loose stools since midnight after taking Peganum harmala to relieve anxiety. She had taken a quarter of a small coffee cup of the medication half an hour before commencement of her symptoms. This herbal medication was given to her by a traditional Iranian healer. She had taken the same medication about 2 months ago without complications. But this time she had taken twice the dose and developed visual hallucinations with gastrointestinal symptoms. The patient has history of mild gastritis. She has been intermittently having anxiety and panic attacks but was not willing to see a psychiatrist due to social stigma. She denied fever, vomiting, dizziness, weakness, tingling, euphoria, agitation, unsteady gait or convulsions. Physical exam showed temperature of 37.2 °C (Oral) Respiratory rate of 18/min, blood pressure of 122/78 mm of Hg and saturations of 99%. She appeared well with no signs of dehydration or jaundice; while abdominal and nervous system examination was normal. She was managed conservatively with fluids, anti-emetics and proton pump inhibitors. Investigations were done including renal & hepatic functions, metabolic panel, coagulation profile, acetaminophen level, lactic acid, creatine kinase, lipase, venous blood gas, electrocardiogram, random blood sugar and serum pregnancy test. All reports and ECG were normal. Patient was admitted for observation as per toxicologist's consultation. She was observed for hemodynamic instability, altered mental status and seizures. The diarrhoea and vomiting improved and the visual hallucination resolved on the 2nd day of admission. She was discharged and advised not to use the herbal medication again and consult a psychiatrist.

CONCLUSION

P. harmala may have therapeutic effects against several ailments but it can cause serious toxicity too. Management is supportive. Prognosis is generally favourable, but can be fatal at very high doses.

REFERENCES

1. Frison, G., Favretto, D., Zancanaro, F., Fazzin, G., & Ferrara, S. D. (2008). A case of β -carboline alkaloid intoxication following ingestion of Peganum harmala seed extract. *Forensic Science International*, 179(2–3), 37–43. <https://doi.org/10.1016/j.forsciint.2008.05.003>
2. Mahmoudian, M., Jalilipour, H., & Salehian, P. (2002). Toxicity of Peganum harmala: Review and a Case Report. *Iran. J. Pharmacol. Ther.*, 1(1), 1–4. <https://doi.org/10.1016/j.fct.2009.12.019>
3. Herraiz, T., González, D., Ancín-Azpilicueta, C., Arán, V. J., & Guillén, H. (2010). β -Carboline alkaloids in Peganum harmala and inhibition of human monoamine oxidase (MAO). *Food and Chemical Toxicology*, 48(3), 839–845. <https://doi.org/10.1016/j.fct.2009.12.019>
4. Yuruktumen, A., Karaduman, S., Bengi, F., & Fowler, J. (2008). Syrian rue tea: A recipe for disaster. *Clinical Toxicology*, 46(8), 749–752. <https://doi.org/10.1080/15563650701323205>
5. Bahadir Bakim, Sencan Sertcelik & Onur Tankaya. A Case of Serotonin Syndrome with Antidepressant Treatment and Concomitant use of The Herbal Remedy (Peganum Harmala)
6. www.plantsoftheworldonline.org (fig 1)



Fig 1

NONE OF THE AUTHORS HAVE ANY DISCLOSURE TO DECLARE

Contact :
ranajaffer1979@yahoo.com

THIAMETHOXAM TOXICITY: ATROPINE IS NOT ALWAYS THE RESCUE

DR RANA J H HUSSEIN (MBBS,MRCM,CABEM); DYOMGY DEVI (PharmD)

BACKGROUND

Intentional consumption of pesticides and insecticides has often been a means of committing suicide in the Asia - Pacific region. Neonicotinoids are a new generation of insecticides which have a better safety profile than organophosphates. Most of the literature found on neonicotinoids is based on animal studies. While human studies and case reports are scarce. In this case report we would like to highlight the toxicity caused by Thiamethoxam, a third generation neonicotinoid.

DISCUSSION

Neonicotinoids act selectively as agonists on nicotinic acetylcholine receptors of insects, with mild effects on humans because they can not cross the blood brain barrier; although their end products can. Humans can be intoxicated through ingestion, dermal contact or inhalation. Clinical presentation may include vomiting, oral burns, odynophagia, abdominal pain, shortness of breath, palpitations, hypertension/hypotension seizures, respiratory centre depression and coma. The solvents in the insecticides are irritant and corrosive so can cause aspiration pneumonia and gastrointestinal tract burns. There are no diagnostic tests for detecting the insecticide. Management is by supportive care, no antidotes required. Atropine and oximes should only be given if patient has muscarinic symptoms, as in organophosphorus poisoning. Early intubation is preferred in obtunded patients, while decontamination with lavage and charcoal should be avoided due to the high risk of aspiration

CASE HISTORY

A young Pakistani male presented to the emergency department with seizures after intentionally consuming a bottle of Thiamethoxam one hour before arriving to the hospital, attempting suicide. He had diabetes mellitus and was on oral medications but had no history of depression. Vital signs were BP140/90, PR 125 bpm, RR 30/min, SpO2: 98%, T: 36.4 C, blood glucose 18mmol. ECG showed sinus tachycardia. Examination did not show any miosis, no moist skin, no bradycardia, no wheezes/crackles. He was started on Lorazepam 4 mg IV and 1L of 0.9% normal saline. ABG showed severe metabolic acidosis, pH 6.9, lactic acid 7 mmol/l and he was given sodium bicarbonate IV 50ml, 8.4% . Blood tests were obtained for complete blood count, renal & liver functions, electrolytes, coagulation, ketones, acetaminophen, salicylate, tricyclic antidepressant and ethanol levels. Despite being drowsy, the patient had a nasogastric tube inserted and 300 ml of milky fluid was aspirated followed by administration of 50mg of activated charcoal. Toxicologist advised to consider intubation, conservative treatment and admission for close monitoring. Chest x-ray showed a right lower lobe infiltrate. Rest of the blood reports were normal. He was admitted to the intensive care unit. He continued to have tachypnea and acidosis, then the intensivist intubated him. Patient was started on fentanyl, midazolam and propofol infusion. Also insulin infusion and IV fluids were commenced. Overnight, the tachypnea and acidosis improved. But the next day he developed fever and the blood culture showed growth for Klebsiella pneumoniae. He was started on amoxicillin/clavulanic acid. The patient was extubated on 3rd day of admission and continued on the antibiotics orally. He was discharged after 10 days of hospital admission with oral benzodiazepines and follow up in the psychiatric clinic.

CONCLUSION

In this case report we wanted to highlight that although neonicotinoids act selectively on insects, they are not void of human toxicity. Further research needs to be conducted to investigate their short term and long term effects.

REFERENCES

- 1.Acute Poisoning with Neonicotinoid Insecticide. Nicolai Nistor, Otilia Elena Frăsinariu and Violeta Ștreangă
- 2.Acute Poisoning with Neonicotinoid Insecticides: A Case Report And Literature Review. Pei-Chen Lin1, Hung-Jung Lin1, 2, Yu-Ying Liao1, How-Ran Guo3 and Kuo-Tai Chen1, 4
3. Thiamethoxam, a Neonicotinoid Poisoning Causing Acute Kidney Injury via a Novel Mechanism. Sakthirajan Ramanathan1, Senthil Kumar M2, Gopal Sanjeevi2, Babu Narayanan3 and Anila Abraham Kurien4 (FIG 1)
4. Effects of Neonicotinoid Pesticide Exposure on Human Health: A Systematic Review. Andria M. Cimino, 1 Abbe L. Boyles, 2 Kristina A. Thayer, 2 and Melissa J. Perry1
5. Acute Human Self-Poisoning with Imidacloprid Compound: A Neonicotinoid Insecticide. Fahim Mohamed1*, Indika Gawarammana1, Thomas A. Robertson2, Michael S. Roberts2, Chathura Palangasinghe1, Shukry Zawahir1, Shaluka Jayamanne3, Jaganathan Kandasamy4, Michael Eddleston1,5, Nick A. Buckley1,6, Andrew H. Dawson1,7, Darren M. Roberts1,8
6. A critical review of neonicotinoid insecticides for developmental neurotoxicity. Larry P. Sheetsa, Abby A. Lib, Daniel J. Minnema, Richard H. Collierd, Moire R. Creeke and Richard C. Pefferc



FIG 1: Thiamethoxam Granules

NONE OF THE AUTHORS HAVE ANY DISCLOSURE TO DECLARE

Contact :
ranajaffer1979@yahoo.com