




Clinical Case Report of Acute Heart Injury And Acute Rhabdomyolysis Due To Cyanua Poisoning

 Nguyen Dang DUC¹,  Nguyen Phuong SINH²,  Lam Nguyen Hong Anh³

¹Bach Mai Hospital in Viet Nam

²Thai Nguyen University of Medicine and Pharmacy in Viet Nam

³The first year student of Thai Nguyen University of Medicine and Pharmacy in Viet Nam

Abstract

Cyanide poisoning is one of the most dangerous poisonings, it can be absorbed into the body through the mouth, inhalation and through the skin. A 32-year-old female patient was admitted to our poison control center because of high fever, severe vomiting, and seizures. Physical examination found that the patient was drowsy, had a high fever of 40 degrees Celsius, pulse of 140 beats/minute, and increased tendon and bone reflexes. Exploiting the patient's information, it was discovered that the patient bought Cyanide to drink with the intention of committing suicide. The patient was quickly treated with gastric lavage and activated charcoal. Echocardiography recorded EF: 35%, reduced movement of the entire myocardium. CK blood test: 4562 U/L. The patient's condition rapidly deteriorated and the patient was made ECMO, IHD and CVVHDF. After 3 days of treatment, the patient's condition did not improve, so the family asked for the patient to go home. This article aims to describe the rapidly progressing and severe damage to the heart and muscles of patients with cyanide poisoning.

Keywords: Cyanide poisoning, Acute Heart Injury, Acute Rhabdomyolysis

Introduction

Cyanide is one of the fastest-acting and the most toxic poisons. They can be absorbed into the body through the mouth, inhalation and through the skin. Oral poisoning occurs when patients drink cyanide themselves or eat foods containing a lot of cyanide such as cassava, apricot kernels, cherry tree seeds and leaves, apple cores, plums, and peaches. Cyanide can be in the form of compounds such as potassiumcyanide, sodiumcyanide, hydrocyanide, zinc cyanide, silver cyanide... In this report, we having a 32-year-old female patient admitted to the hospital for cyanide poisoning at the 4th hour. She was taken to the hospital with high fever, convulsions, decreased consciousness and rhabdomyolysis. CK: 4562 U/L, reduced movement of entire the myocardium with EF 35%.

Case Report

32-year-old female patient's history: Untreated depression, intended suicide once with sleeping pills. Entered the poison control center with high fever, convulsions, and decreased consciousness. It is known that on the afternoon of the same day, the patient was found by his family in a state of irritability, vomiting a lot, vomiting white fluid, then She reported self- drinking cyanide purchased online. She was

taken to the hospital by her family members with coma Glasgow: 12 points, breathing rate 55l/min, fever 40 degrees Celsius. Then there were many whole body convulsions, each lasting 30 seconds. During the convulsions, the patient could not be awared around, after the convulsions the patient wakes up slowly.

Clinical examination: Patient weighs 54kg, height 152cm, BMI: 23.3. Pulse 140 beats/min, blood pressure: 90/60 mmHg. The patient is agitated and screaming. Called and asked but no response. Pupils on both sides are 3mm, still reflect light, fever is 39 degrees Celsius. Negative stiff neck, negative meningeal stripe. Rapid breathing, respiratory rate: 50-55 L/min. The lungs are ventilated equally on both sides. Spasticity, increased muscle tone in the limbs, increased tendon reflexes. Other agencies have not detected anything unusual.

The patient was tested for gastric fluid and urine with the following results: Cyanide was positive with a concentration of 0.525 mg/L in gastric fluid.

Blood count and coagulation tests were within normal limits. Cre test: 84 $\mu\text{mol/l}$, CK increased from 4562->54532 U/L within 1 day. Troponin Ths increased 497->772 in 1 day. Lactac: 7.0. Echocardiography EF: 35%, uniformly reduced movement in many areas of the myocardium. The patient received gastric lavage and a dose of activated charcoal (1g/kg). The patient's condition then worsened very quickly with pulse rapidly increasing to 200 beats/

Corresponding Author: Duc Nguyen DANG

e-mail: dangducnguyen79@gmail.com

Received: 27.02.2024 • **Revision:** 04.04.2024 • **Accepted:** 29.04.2024

DOI: 10.55994/ejcc.1443800

©Copyright by Emergency Physicians Association of Turkey -

Available online at <https://dergipark.org.tr/tr/pub/ejcc>

Cite this article as: Duc ND, Sinh NP, Anh LN. Clinical Case Report of Acute Heart Injury And Acute Rhabdomyolysis Due To Cyanua Poisoning. Eurasian Journal of Critical Care. 2024;6(1): 46-47

min, blood pressure dropping very quickly to 50/30 mmHg, consciousness rapidly decreasing from 12 down 8 points. Patients were given vasopressors including: Dobutamine: 20mcg/kg/minute. Noadrenalin: 1.5mcg/kg/min, Adrenalin: 1.5mcg/kg/min, endotracheal tube, mechanical ventilation. Then ECMO was performed to support the heart. 2 days after ECMO, the patient's condition did not improve, the patient's heart appeared in continuous ventricular fibrillation and was electroshocked. IHD and CVVHDF dialysis. However, the condition was not improved, the family asked for the patient to go home.

Discussion

Cyanide is one of the most deadly poisons known. Cyanide is known as sodium cyanide and potassium cyanide. In its gaseous form it is usually hydrocyanide. The mechanism of toxicity is Cyanide binds tightly to the iron ion (Fe^{3+}) of cytochrome oxidase a3, inhibiting this final enzyme in the cytochrome complex of mitochondria. When the activity of this enzyme is blocked, oxidative phosphorylation ceases. Therefore, the cell must switch to anaerobic metabolism with glucose to produce ATP. This leads to increased blood lactase and increased oxygen toxicity in venous blood because cells cannot use oxygen. The heart, muscles and central nervous system are the organs most affected by cyanide poisoning. Cardiovascular disorders occurring after cyanide ingestion may include rapid, shallow pulse, tachycardia, congestive heart failure, increased Troponin Ths, and hypotension. Rhabdomyolysis and increased serum creatine kinase levels are also signs of cyanide poisoning. Metabolic acidosis is seen in 67% of patients with acute oral cyanide poisoning. Headache, loss of consciousness, convulsions, positive Babinsky sign, hemiplegia, difficulty speaking, Parkinson's syndrome, coma, and death may occur due to the effects of nervous system damage after cyanide ingestion. . Additionally, a strong almond odor to the breath and multiple clear pink patches on the skin may be seen on physical examination. Cardiovascular complications in our report included Tachycardia, uniform regional hypokinesia of myocardial regions with reduced ejection fraction EF of 35%. Our patient was intubated and mechanically ventilated using vasopressors, IHD dialysis and CVVHDF and also received ECMO.



Figure 1: Image of a patient's cyanide poisoning test

Conclusion

In our patient, the patient's signs and symptoms manifested in cyanide-sensitive organs including the brain, heart, and muscles. Identifying patients with cyanide poisoning is through medical inquiries and toxicology tests. Once again we want to emphasize that cyanide poisoning is one of the severe poisonings with a high mortality rate. Early identification of toxins and close monitoring of the patient's clinical and paraclinical condition can help the patient have a higher chance of being saved.

References

1. Hall AH, Rumack BH. Clinical toxicology of cyanide. *Ann Emerg Med* 1986; 15:1067.
2. Mizock BA. Lactic acidosis. *Dis Mon* 1989; 35:233.
3. Bryson, PD. *Comprehensive Review in Toxicology for Emergency Clinicians*, 3rd, Taylor and Francis, Denver 1996. p.352.
4. Agency for Toxic Substances and Disease Registry. *Toxicological profile for cyanide (update)*. Atlanta: US Department of Health and Human Services, Public Health Service, 2006 June, 1-341. ATSDR/TP-92/09