

Case Report

Loss of consciousness in a little traveler

Anahita Alizadeh (MD)¹
Forough Rakhshanizadeh (MD)^{2*}

1. Department of Clinical Toxicology, Faculty of Medicine, Mashhad University of Medical Sciences, Mashhad, Iran

2. Department of Pediatrics, Faculty of Medicine, Mashhad University of Medical Sciences, Mashhad, Iran

* Correspondence:

Forough Rakhshanizadeh,
Department of Pediatrics, Faculty of Medicine, Mashhad University of Medical Sciences, Mashhad, Iran

E-mail:

Rakhshanizadehf@mums.ac.ir

Tel: 0098 5137233014

Fax: 0098 5137233014

Received: 30 Sep 2019

Revised: 1 Feb 2020

Accepted: 15 Feb 2020

Abstract

Background: Loss of consciousness in children can be caused by a wide spectrum of factors, including infection, metabolic disorders, trauma, and poisoning which requires timely and accurate evaluation.

Case presentation: In this paper, we introduce a three-year-old boy who was first referred to the Emergency ward of Mashhad Imam Reza Hospital due to unconsciousness. Having spent a few days in a hotel, this boy, who was a visitor to Mashhad, lost consciousness. During evaluations, hypotension and severe high anion gap metabolic acidosis was observed. Finally, the patient was diagnosed with ethylene glycol poisoning.

Conclusion: Poisoning should be considered as one of the most likely diagnoses in children with loss of consciousness. The identification of the clinical symptoms and the use of appropriate diagnostic algorithms is essential for timely diagnosis and appropriate treatment of specific cases of toxicity.

Keywords: Acidosis, Ethylene glycol, Pediatric, Poisoning, Unconsciousness

Citation:

Alizadeh A, Rakhshanizadeh F. Loss of consciousness in a little traveler. Caspian J Intern Med 2021; 12(1): 103-106.

Loss of consciousness in children may have important and dangerous causes, including infections, metabolic disorders, and different types of toxicity, trauma, and convulsions (1). The obtainment of precise medical history and complete physical examination, especially neurological examination, is required for the accurate and timely diagnosis of the cause of unconsciousness (2). One of the major causes of altered mental status in pediatric patients is toxicity and poisoning. Poisoning in children is a common important complex phenomenon which is sometimes difficult to diagnose. Some poisoning is diagnosed with certain clinical syndromes and identification of these toxidromes is essential to address not only the correct diagnosis but also rules out other differential diagnoses. Physicians' awareness of symptoms of children's common poisoning is of paramount importance and they should consider poisoning in the differential diagnosis of patients with loss of consciousness (3). Alcohols, especially ethylene glycols (EG), are among those materials that cause consciousness status alteration (4). This short communication introduces the case of a three-year-old boy with the initial complaint of abdominal pain and vomiting who was referred to the emergency ward due to loss of consciousness.

Case presentation

This study was conducted on a three-year-old boy from Shahrekord, Iran, as a tourist in Mashhad, Iran, who spent 2 days in a hotel. According to his mother, the boy first got thirsty at night; therefore, they asked the hotel personnel for some water. Abdominal pain began shortly after drinking a little water, followed by one episode of vomiting.

The next morning, the boy was referred to the Pediatric Emergency ward of Mashhad Imam Reza Hospital due to loss of consciousness. His weight was 15kg and his height was 93cm, both in 50 percentiles. He was previously a healthy child with normal development without any history of a specific disease or hospital admission. He was the only child with no history of metabolic disorders in their family and his parents were not consanguine. There was no history of diarrhea, fever, cough, headache, trauma, or any other symptoms.

In the initial examination, the child was lethargic with midsized and reactive pupils. His respiration was deep and rapid. His vital signs demonstrated hypotension (BP: 40/30 mmHg), tachypnea (RR: 42/min), and tachycardia (HR: 156/min). The pulses were weak and rapid, the extremities were cold with capillary refill time more than 3sec. Moreover, heart and lung auscultation were normal and neurological examinations revealed no neurological deficit. Other physical examinations were also normal.

On admission to the hospital, one dose of 20cc/kg normal saline was administered rapidly and the shock was resolved successfully. In the initial tests, the arterial-blood gas test demonstrated severe acidosis (first PH: 7.07, HCO₃:4 meq/L, PCO₂:14 mmHg).

Regarding the severe resistant acidosis, the patient received several doses of sodium bicarbonate; however, the acidosis persisted. The anion gap (AG) was calculated at 26 meq/L, lactate was measured at 58 mg/dl and there was mild hypocalcemia (Ca: 8.8mg/dl) without any other electrolyte disturbance. Blood glucose was normal and no ketone was detected in the urine test. Chest x-ray and ECG (electrocardiogram) findings were normal. Neurologic and cardiologic consultation with pediatric subspecialists recommended correction of hypotension and acidosis. Toxicology tests revealed negative serum concentration of salicylate, acetaminophen, phenobarbital, ethanol, and methanol; however, the serum level of EG was positive (28.8 mg/dl). The results of laboratory examinations are summarized in table 1 and toxicology test result are presented in table 2.

Finally, based on the clinical evidence, physical examinations, and paraclinical test results, the diagnosis was confirmed as EG poisoning. Treatment response was favorable using appropriate fluid therapy, administration of sodium bicarbonate (1meq/kg stat, then 5meq/kg continues infusion in 24h), ethanol (1gr/kg of 20% solution stat, then 0.1

gr/kg/hr to 24h), thiamin and pyridoxine (1mg/kg/day) (5). Acidosis recovered after about 12h, the EG level reached zero after 24h and the child was discharged from the hospital in good general condition after 2 days.

Table 1: Laboratory evaluations of the patient on arrival to the emergency department

Laboratory test	Patient levels	Normal range
WBC (cell/ μ L)	15,300	4000-11,000
Neutrophil (%)	85.9	33-42
Lymphocyte (%)	10.3	50-59
RBC (cell/ μ L)	4,100,000	3,800,000-5,500,000
Hb (g/dL)	11.4	11.5-13.5
HCT (%)	35	34-40
PLT (cell/ μ L)	230,000	150,000-450,000
PT (sec)	12	10-11.30
PTT (sec)	20	24-36
BS (mg/dL)	120	60-100
Na (mEq/L)	134	135-145
K (mEq/L)	3.2	3.4-4.7
Cl (mEq/L)	109	98-107
Mg (mg/dL)	1.9	1.6-2.4
Ca (mg/dL)	8.8	9-11
Urea (mg/dL)	20	25-40
Cr (mg/dL)	0.5	0.3-0.7
Albumin (g/dL)	3.9	3.6-5.2
AST (U/L)	31	20-60
ALT (U/L)	15	25-40
PH	7.07	7.35-7.45
Pco ₂ (mmHg)	14	35-45
Hco ₃ (mEq/L)	4	22-26
ESR (mm/hr)	15	4-20
CRP (mg/dL)	Negative	0-0.5
Ammonia (micromol/L)	35	<50
Lactate (mg/dl)	58	<18

WBC, White Blood Cell; RBC, Red Blood Cell; Hb, Hemoglobin; HCT, Hematocrit; MCV, Mean Corpuscular Volume; PLT, Platelet; PT, Prothrombin Time; PTT, Partial Thromboplastin Time; BS, Blood Sugar; Na, Sodium; K, Potassium; Cl, Chloride; Mg, Magnesium; Ca, Calcium; Cr, Creatinine; AST, Aspartate aminotransferase; ALT, Alanine aminotransferase; PH, Plasma Hydrogen; Pco₂, Plasma Carbon dioxide; Hco₃, bicarbonate; ESR, Erythrocyte Sedimentation Rate; CRP, C-reactive Protein

Table 2: Toxicology evaluations of the patient

Urine screen	
Benzodiazepines	negative
Phenothiazine	negative
Cannabis	negative
Tramadol	negative
Morphine	negative
Barbiturate	negative
Amphetamines	negative
TCA	negative
Salicylate	negative
Serum concentration range	
Acetaminophen	Not detect
Ethanol	10 mg/dl (negative)
Methanol	0.7 mg/dl (negative)
Ethylene Glycol	28.8 mg/dl (positive)

Discussion

This case presentation is unique for the following reasons: first, it is unlikely that an ethylene glycol solution wrongly offers to a guest when he requested for drinking water in a hotel, the problem that this patient unfortunately experienced. Second, when a pre-healthy child suddenly develops unconsciousness, the diagnosis of a toxicant such as ethylene glycol is typically not made for a pediatrician in the first differential diagnosis, and it may be considered too late due to lack of adequate awareness. Third, the standard approach for the treatment of ethylene glycol poisoning recommends the use of fomepizole. Even if fomepizole was not available in our center, the patient was successfully treated with other medications.

As mentioned in the references, the anion gap should be first calculated in metabolic acidosis. Significant causes of metabolic acidosis with high anion gap ($AG > 13$ meq/L) include diabetic ketoacidosis, kidney failure, tissue hypo perfusion, an inborn error of metabolism, as well as poisoning with aluminum phosphide, alcohol and salicylates (6). In our case, diabetic ketoacidosis was ruled out due to the normality of blood glucose and the absence of any ketone in the urine test. Glomerular filtration rate (GFR) was normal and the findings of renal failure were not observed. Hypotension caused by aluminum phosphide tablet poisoning is highly resistant to regular treatments and the probability of this poisoning was reduced since the patient's blood pressure reached normal range upon receiving 20 cc/kg of normal saline. (7). Our patient was previously healthy with normal

growth and development without any history of metabolic disorders in the family and also his parents were not consanguineous. Therefore, the probability of inborn error of metabolism was less suggested. Poisoning with alcohol was strongly suggested due to the negative result of the serum salicylate level test. In addition, a suspicion existed concerning the history of consuming a fluid like water. It was reported that the child was completely healthy before drinking that fluid and the symptoms appeared after its consumption. Therefore, complementary tests were requested to check the serum level of alcohols, including ethanol, methanol and EG. The results of the tests confirmed EG poisoning.

EG is an alcohol mainly used as an antifreeze for machine engine oil; however, it is sometimes used as a solvent, cleanser, or formifier. This alcohol is sweet, with no color and smell. Although EG toxic dose is 0.5-2CC / Kg, very low amount can be very toxic (8). Signs and symptoms of EG poisoning include vomiting, drunkenness, high anion gap metabolic acidosis, hypotension, respiratory distress, dysrhythmia, and renal failure. During metabolism, EG is converted into oxalate which causes hypocalcemia with binding to calcium (9). Our patient demonstrated mild hypocalcemia; however, no kidney damage occurred. The lactate level in our case was measured at 58 mg/dl. Although this elevation could be real, it could also result from a laboratory error since some laboratory instruments cannot differentiate between lactate and glycolate (10).

The proposed treatments for EG poisoning included the use of ethanol, vitamin B1 and B6, as well as fomepizole as an antidote (5). Hann G et al. reported a 2-year-old boy presented with vomiting, lethargy, tachycardia, and deep rapid respiration with high anion gap metabolic acidosis. Comparable to our approach, they ruled out causes of high anion gap metabolic acidosis and the EG level was estimated at 84.624 mg/dL. The patient received sodium bicarbonate, intravenous fluids, and fomepizole. Although EG poisoning management in children usually needs admission to a pediatric intensive care unit (PICU), he was managed in a general hospital with multi-disciplinary team management (11). We also treated our patient successfully in the emergency department since no PICU bed was available.

Hemodialysis is recently recommended for severe metabolic acidosis, very high EG concentrations, renal failure, deteriorating condition despite supportive measures, and severe electrolyte imbalance (11). Caravati et al. reported six pediatric patients who were diagnosed with EG poisoning.

Their serum EG level and serum bicarbonates were within the range of 62-304 mg/dL and 4-17 mEq/L, respectively. They were successfully treated with intravenous fluid, bicarbonate, fomepizole, and ethanol without hemodialysis. They concluded that hemodialysis might not be indicated in some children with EG level \leq 50 mg/dL and normal renal function (12). Although no fomepizole was available in our center, the child was successfully treated with ethanol, vitamin B1 and B6 fluid therapy and bicarbonate consumption, devoid of any need for hemodialysis.

It is recommended that poisoning, especially alcohol poisoning, be considered in the differential diagnosis of children with unconsciousness and/or high anion gap metabolic acidosis. In addition, to prevent children's poisoning, it is necessary to increase public awareness and adopt effective legislation on the access to hazardous and toxic substances, especially in public places.

Acknowledgments

Thanks to Pediatric Emergency staffs of Emam Reza hospital of Mashhad for helping us during this study and in data gathering process.

Financial Disclosure: There is no financial disclosure.

Conflicts of interest: The authors declare that they have no conflict of interest. Moreover, they take full responsibility for the content and writing of this article.

References

1. Fouad H, Haron M, Halawa EF, Nada M. Nontraumatic coma in a tertiary pediatric emergency department in Egypt: etiology and outcome. *J Child Neurol* 2011; 26: 136-41.
2. Bates D. The management of medical coma. *J Neurol Neurosurg Psychiatry* 1993; 56: 589-98.
3. Abbruzzi G, Stork CM. Pediatric toxicologic concerns. *Emerg Med Clin* 2002; 20: 223-47.
4. Caballería J. Current concepts in alcohol metabolism. *Ann Hepatol* 2003; 2: 60-8.
5. Howland MA. Pyridoxin. In: Nelson LS, Howland MA, Lewin NA, et al. *Goldfrank's toxicologic emergencies*. 11th ed. New York, MC Graw Hill 2019; pp: 865-7
6. Kraut JA, Madias NE. Metabolic acidosis: pathophysiology, diagnosis and management. *Nat Rev Nephrol* 2010; 6: 274-85.
7. Mehrpour O, Asadi S, Yaghoubi MA, et al. Cardiogenic shock due to aluminum phosphide poisoning treated with intra-aortic balloon pump: a report of two cases. *Cardiovasc Toxicol* 2019; 19: 474-81.
8. Bruno M, Borron SW, Baud FJ. Toxic alcohols. Shanon M, Borron SW, Burns M. In: Haddad and Winchester's clinical management of poisoning and drug overdose. 4th ed. Philadelphia: Saunders Elsevier 2007; pp: 611-21.
9. Wiener SW. Toxic alcohols. In: Nelson LS, Howland MA, Lewin NA, et al. *Goldfrank's toxicologic emergencies*, 11th ed. New York: MC Graw Hill 2019; pp: 1421-35.
10. Brindley PG, Butler MS, Cembrowski G, Brindley DN. Falsely elevated point-of-care lactate measurement after ingestion of ethylene glycol. *CMAJ* 2007; 176: 1097-9.
11. Hann G, Duncan D, Sudhir G, West P, Sohi D. Antifreeze on a freezing morning: ethylene glycol poisoning in a 2-year-old. *BMJ Case Rep* 2012; 2012:bcr0720114509.
12. Caravati EM, Heileson HL, Jones M. Treatment of severe pediatric ethylene glycol intoxication without hemodialysis. *J Toxicol Clin Toxicol* 2004; 42: 255-9.