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Case Report

A Rare Case of Severe Metabolic Acidosis Caused by Toluene Abuse

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ABSTRACT

Toluene is a solvent used in various industries as a raw material to produce organic compounds and in the manufacture of diverse products. Contact with toluene can occur both accidentally and intentionally. Intoxication with toluene and its by-products, whether intentional or accidental, can be toxic to all systems, with the cardio-pulmonary system being primarily affected. The intentional use of toluene leads to psychoactive effects, including euphoria, which makes it the most widely inhaled volatile drug among all age groups in many countries. Renal tubular acidosis is the most common complication arising from toluene intoxication. This case report will discuss a 38-year-old patient who was admitted to the emergency service of our hospital.

INTRODUCTION

Toluene is widely utilized as a raw material in the production of organic compounds, such as benzene, across various industrial sectors. These compounds are extensively applied in different industries and used in products like gasoline, spray paint, cleaning agents, and paint thinners. However, direct inhalation exposure to toluene can result in environmental, accidental, and intentional intoxications. Firstly, its easy accessibility and relatively low cost make it a common choice compared to other inhalants of abuse. Secondly, it can be inhaled either in its pure form or as a component of numerous commercial products. Thirdly, it induces acute psychoactive and neurological effects, such as euphoria, positioning toluene as the most commonly abused inhalant across all age groups in many countries.[1,2] Toluene inhalation is associated with various pathologies, including severe metabolic changes like renal tubular acidosis type 1 (RTA-1). Additionally, toluene can cause severe arrhythmias, including tachyarrhythmia and bradyarrhythmia. ST wave pattern alterations, indicative of hypokalemia and acute myocardial infarction, have been documented in cases of toluene intoxication. Rhabdomyolysis and acute hepatorenal injuries have also been reported. Consequently, toluene exposure may constitute a risk factor for adverse outcomes.[1] Toluene is metabolized into benzoic acid, which the kidneys then eliminate.[3] Distal RTA-1 is traditionally characterized as hyperchloremic with a normal anion gap, accompanied by hypokalemia and muscle paralysis, indicative of toluene intoxication.[4] This case report will discuss a 38-year-old man who was found in an unconscious state due to toluene poisoning.

CASE REPORT

A 38-year-old male patient with a history of toluene abuse

was brought to our emergency room (ER) in an unconscious state. His relatives reported that his symptoms rapidly worsened minutes after they found him sniffing paint thinners in his room, following which he collapsed. They also noted that he had not taken any medications or other substances. Upon arrival at our ER, he had a Glasgow Coma Scale score of 3, with bilateral dilated pupils unresponsive to direct and indirect light. His vital signs were a blood pressure of 70/50 mmHg, a pulse of 52 beats/min, and an oxygen saturation rate of 55%. Nasal oxygen was administered at 2L/min due to the low saturation rate. An ECG showed sinus bradycardia, and vascular access was established in both arms. A complete blood count (CBC), serum biochemistry, and a toxicology panel were conducted. Given the family's statement and the patient's bradycardia and bradypnea, Naloxone at 0.4mg/mL and intravenous fluids were administered. A follow-up showed a pulse of 25 beats/min, prompting the administration of 0.5mg/mL atropine. Initial laboratory results indicated: sodium at 144 mmol/L; potassium at 5.1 mmol/L; chloride at 100 mmol/L; glucose at 309mg/dL; creatinine at 1.38mg/dL. A venous blood gas analysis revealed a pH of 6.400 (7.35-7.45), a pCO2 of 81.8 mmHg, a pO2 of 63.1 mmHg (83-108), bicarbonate at 4.7 mmHg (22.5-26.9), and lactate at 24 mmol/L (0.5-1.6) - indicative of severe metabolic acidosis. Approximately five minutes later, the patient had no palpable carotid or femoral pulse and no spontaneous respiration. Resuscitation efforts were initiated, and rapid sequence intubation was performed. After 10 minutes, a palpable pulse returned along with spontaneous respirations. The patient received 40 mcg/min of inotropic support, 15 mcg/min of dopamine, and 100 mEq of bicarbonate. Post-treatment, the patient's vitals were a blood pressure of 100/50mmHg, oxygen saturation at 99% (while intubated), and a pulse of 90 beats/min. A follow-up venous blood gas showed a slight increase in pH to 6.467 (7.35-7.45). A CT scan under a physician's supervision revealed no pathological findings. The patient was evaluated in the ER by anesthesiologists, and admission to the intensive care unit (ICU) was recommended. The toxicology panel, which was tested on a urine sample, did not detect amphetamines, benzodiazepines, buprenorphine, cannabinoids, cocaine, ecstasy, heroin metabolites, or opioids. The patient was stabilized in the ER and transferred to a hospital with a specialized medical toxicology ICU. The patient remained intubated in the ICU for twenty days; however, he succumbed to his condition twenty days after ICU admission.

DISCUSSION

Death related to aliphatic and aromatic hydrocarbon intoxication most commonly results from asphyxia. Guo described a phenomenon known as "sudden sniffing death," which refers to cardiovascular collapse following the deliberate inhalation of aliphatic and aromatic hydrocarbons within a few hours of exposure. While the abuse of toluene is globally widespread, instances of sudden sniffing death are rare.[5] Toluene rapidly diffuses into the bloodstream and penetrates lipid-rich tissues. It is oxidized in tissues, leading to the formation of lipid peroxidation products and free radicals.[3] The metabolism of toluene by cytochrome P-450 results in benzoic and hippuric acids, which are excreted by the kidneys and detectable in urine.[1,3] Exposure to toluene can also lead to electrolyte imbalances and acid-base disturbances. It is reported that metabolic acidosis occurs in 87% of toluene intoxication cases, indicative of distal tubular acidosis.[6] In the present case, the initial venous blood gas showed a pH of 6.400 (7.35-7.45), signifying severe metabolic acidosis. Toluene sniffing can also induce normal anion gap acidosis by hindering renal elimination of ammonium ions, the primary carrier for excess hydrogen ions.[7] Hokenek et al.[8] analyzed 274 patients in intensive care and found that lactate levels of 4.64±4.696 mmol/L are associated with early mortality. In this case, the lactate level was alarmingly high at 24.0 mmol/L (0.5-1.6). Moreover, toluene intoxication can impact the lungs, heart, liver, kidneys, and central nervous system. Cardiac involvement is one of the primary consequences of toluene intoxication, with tachyarrhythmia being the generally expected clinical presentation. However, there have been reports that toluene can also induce bradyarrhythmia, heart block, and AV dissociation.[9] In this case, sinus bradycardia was observed.

Conclusion

In managing patients exposed to toxic levels of toluene, it is critical to recognize that they may present with severe metabolic acidosis and elevated lactate levels, which are associated with an increased risk of mortality. Moreover, cardiac complications can manifest not only as severe tachyarrhythmia but also as bradyarrhythmia, heart block, and AV dissociation. It is essential to acknowledge that intentional toluene inhalation poses a significant public health issue and presents a considerable challenge in emergency medicine due to its potential to cause sudden death through severe metabolic and cardiopulmonary outcomes.

Peer-review

Externally peer-reviewed.

Authorship Contributions

Concept: C.A.T.; Design: A.U.S.; Supervision: O.G.; Fundings: D.Ö.; Materials: C.A.T.; Data: A.U.S.; Analysis: O.G.; Literature search: O.G.; Writing: O.G.; Critical revision: C.A.T.

Conflict of Interest

None declared.

REFERENCES

1. Camara-Lemarroy CR, Rodríguez-Gutiérrez R, Monreal-Robles R, González-González JG. Acute toluene intoxication: Clinical presentation, management and prognosis: A prospective observational study. BMC Emerg Med 2015;15:19. [CrossRef]
2. Agency for Toxic Substances and Disease Registry. Toxicological pro-

file for toluene. Available at: http://www.atsdr.cdc.gov/toxprofiles/. Accessed Dec 20, 2023.
3. White RE, Sligar SG, Coon MJ. Evidence for a homolytic mechanism of peroxide oxygen-oxygen bond cleavage during substrate hydroxylation by cytochrome P-450. J Biol Chem 1980;255:11108–11. [CrossRef]
4. Carlisle EJ, Donnelly SM, Vasuvattakul S, Kamel KS, Tobe S, Halperin ML. Glue-sniffing and distal renal tubular acidosis: Sticking to the facts. J Am Soc Nephrol 1991;1:1019–27. [CrossRef]
5. Guo TJ. A Rare but Serious Case of Toluene-Induced Sudden Sniffing Death. J Acute Med 2015;5:109–11. [CrossRef]
6. Tsao JH, Hu YH, How CK, Chern CH, Hung-Tsang Yen D, Huang CI. Atrioventricular conduction abnormality and hyperchloremic metabolic acidosis in toluene sniffing. J Formos Med Assoc 2011;110:652–4. [CrossRef]
7. Dickson RP, Luks AM. Toluene toxicity as a cause of elevated anion gap metabolic acidosis. Respir Care 2009;54:1115–7.
8. Hökenek NM, Seyhan AU, Erdoğan MÖ, Tekyol D, Yılmaz E, Korkmaz S. Evaluation of blood gas analysis as a mortality predictor. South Clin İst Euras 2019;30:228–31. [CrossRef]
9. Yasar S, Yildirim E, Koklu M, Gursoy E, Celik M, Yuksel UC. A case of reversible cardiomyopathy associated with acute toluene exposure. Turk J Emerg Med 2016;16:123–5. [CrossRef]

Toluen Kullanımının Neden Olduğu Derin Metabolik Asidoz Vakası

Amaç: Toluen, birçok endüstride organik bileşikler üretmek için hammadde olarak kullanılan ve çeşitli ürünlerin yapımında kullanılan bir çözücüdür. Toluen ile temasa geçmek hem kazara hem de kasıtlı olabilir. Toluen ve yan ürünleri ile hem kasıtlı hem de kazara zehirlenme, başta kardiyopulmoner sistem olmak üzere tüm sistemlerde toksik olabilir. Toluenin kasıtlı kullanımı, öfori de dahil olmak üzere psikoaktif sonuçlara neden olur ve bu da onu birçok ülkede her yaştan insan arasında en yaygın olarak solunan uçucu ilaç haline getirir. Toluen zehirlenmesinin en yaygın komplikasyonu renal tübüler asidozdur. Bu durumda hastanemiz acil servisine başvuran 38 yaşındaki hastanın raporu tartışılacaktır.

Anahtar Sözcükler: Acil departmanı; metabolik asidoz; toluen; zehirlenme.