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A Rare Case of Metabolic Acidosis Associated with Paint Thinner Abuse

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ABSTRACT

Introduction: A paint thinner is a solvent used to dilute oil-based paints in the industry and at home. Misuse among adolescents is common worldwide.

Case Report: We report a case of metabolic acidosis in a 30-year-old male patient who inhaled a paint thinner and who presented to our emergency department with nausea, vomiting, and clouding of consciousness.

Conclusion: Metabolic acidosis due to thinner inhalation is a rare clinical condition. This report aimed to describe metabolic acidosis associated with thinner abuse and its treatment.

Keywords: Thinner, abuse, metabolic acidosis

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Introduction

A paint thinner is a volatile solvent containing various aromatic hydrocarbons such as toluene, benzene, acetone and hexane. Its misuse has recently considerably increased in Turkey. Its abuse is particularly increasing among young people in regions with low socioeconomic levels, particularly as other narcotic substances are expensive and difficult to obtain. (1) It has been reported that the world has reached up to 20% even once in abusive life in various regions of adolescents (2, 3). Paint thinner abuse can have neurotoxic, hepatotoxic, myotoxic, nephrotoxic, and cardiotoxic effects and can even be fatal. The most common presentation symptoms in the emergency department are vomiting and respiratory distress. This report describes a case of metabolic acidosis in a patient presenting to our emergency department due to vomiting and consciousness disturbance after paint thinner abuse.

Case Report

A 30-year-old male patient presented to our emergency department due to nausea–vomiting, clouded consciousness, and agitation. We learned that he had a inhaled paint thinner from a plastic bag for purposes of abuse approximately 3 h previously. His blood pressure was 100/70 mmHg, respiration rate was 32/min, heart rate was 136/min, and body temperature from the tympanic membrane was 36.3°C. On performing a physical examination, the patient was found to be confused and agitated and his pupils were isocoric and mydriatic. Examinations of other systems were normal. On performing biochemical tests, the lactate dehydrogenase (LDH) level was 306 U/L (normal level: <247 U/L), CK-MB (kreatin kinaz-MB) level was 11.1 ng/L (normal: 0.6–6.3 ng/L) and CK level was 314 U/L (normal: <171 U/L). Blood urea nitrogen, blood sugar, creatine, sodium potassium, and other electrolyte levels were within normal limits. The saturation level in arterial blood gasses was 97.9%, pH was 7.189, pCO_2

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was 6 mmHg (normal: 32–48 mmHg), pO_2 was 135 % (normal 83-108), methemoglobin level was 1.1% (normal: 0–1.5%), HCO_3 level was 7.3 mmol/L, (22-26 mEq/L) and base deficit was −26 mmol/L. Decompensated metabolic deficit was suspected due to the low base deficit and bicarbonate levels and as the anticipated pCO_2 was not observed. HCO3 loading at 140 meq/h was performed to raise the patient's bicarbonate levels to above 12 meq/L. Arterial blood gasses were monitored hourly, and the pH rose to 7.301 and HCO_3 level rose to 10 mmol/L. Rapid sequence intubation was performed, and the patient was admitted for receiving intensive care. The patient was extubated 6 h after receiving intensive care. Bicarbonate was administered in line with the blood gas status, and the patient was discharged in a healthy condition on the second day after intravenous fluid therapy.

Discussion

A paint thinner is a compound containing the organic compounds toluene and xylene. It may be described as either toluene- or xylene-based, depending on the proportions involved. Thinners misused by patients generally contain a high proportion of toluene. The standard proportions of thinners in Turkey are 90% toluene, 9% ethyl acetate, <1% benzol. Toluene-related effects therefore generally predominate in paint thinner intoxications (4). Patients may present to the emergency department with various symptoms, depending on the length of use, the quantity involved, and the frequency of abuse. Symptoms and findings during acute intoxication include fever, agitation, nausea, vomiting, central nervous system depression, hepatic and renal toxicity findings, methemoglobinemia, dysrhythmia, and sudden death (5, 6). Our patient was confused and agitated, but severe vomiting was present. High ion gap metabolic acidosis was present in arterial blood gasses, while methemoglobin levels were normal. The metabolic effects of paint thinners include renal tubular acidosis, hypokalemia, hypophosphatemia, hyperchloremia, and azotemia. One of the principal reasons for this is that it triggers anaerobic respiration causing hypoxia in cells (7). We principally attributed high ion gap metabolic acidosis in our patient to cellular hypoxia. In addition, it must not be forgotten that methanol, hexane and xylene contained in paint thinners, albeit in small quantities, can lead to anion gap. Hypercapnia and hypoxia may normally be expected in abuse involving paint thinners being inhaled from a plastic bag, and we ascribe the metabolic acidosis in our patient to cellular hypoxia (8). Additionally, high LDH, CK, and CK-MB levels were corroborative of hypoxia at the cellular level. Although cases of renal tubular acidosis, methemoglobinemia, and sudden death associated with thinner inhalation have been reported in the literature, we believe that this case is particularly significant as no previous cases of isolated metabolic acidosis have been reported thus far. Symptomatic treatment is generally sufficient during thinner intoxication. However, full monitoring and observation during intensive care are useful in the follow-up of complications that may develop secondary to metabolic acidosis.

Conclusion

Emergency physicians should be aware that paint thinner abuse by inhalation may give rise to isolated metabolic acidosis. Blood gasses should be analyzed in detail in these patients, even if presentation symptoms in the emergency department are mild.

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