A rare but serious case of toluene-induced sudden sniffing death

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Abstract

Sudden sniffing death is rare but serious during volatile substance sniffing or in the subsequent hours, especially in patients with toluene (super-glues) sniffing. We present the case of a 38-year-old female who sustained out-of-hospital cardiac arrest temporally related to toluene sniffing. Resuscitation of sudden sniffing death is seldom successful in the previous published reports. Our patient initially survived after aggressive resuscitation. Toluene abuse is associated with major toxicities including severe metabolic acidosis with lactate accumulation, CNS depression, ventricular arrhythmias, rhabdomyolysis, and liver toxicity. A possible mechanism of sudden sniffing death related to toluene abuse is ventricular arrhythmias, yet the true mechanism is still unknown. Severe metabolic acidosis may be another possible and important cause of sudden death in our patient. Sudden sniffing death should be considered in evaluating out-of-hospital cardiac arrest related to toluene sniffing, especially in the severe metabolic acidosis condition.

Keywords: sudden sniffing death; toluene; metabolic acidosis; hyperlactemia; rhabdomyolysis; liver toxicity

1. Introduction

Toluene, the major component of glues, is commonly used as an organic solvent in many industrial processes and household products. Toluene abuse is prevalent around the world because toluene is inexpensive and legal to buy. We report an unusual case of sudden sniffing death with severe mixed acidosis, hyperlactemia, rhabdomyolysis, and liver toxicity resulted from toluene (super-glue) abuse.

2. Case report

A 38-year-old female patient presented to our emergency department for out-of-hospital cardiac arrest on 7th April in 2015. She received cardiopulmonary resuscitation (CPR) for 10 minutes at the emergency department and then return of spontaneous circulation (ROSC) was noted. According to her family’s statements, she was a heavy toluene (super-glues) sniffer and she had frequent history of admission due to toluene intoxication. In the evening of April 7th, she sniffed 20 tubes of super-glue and then her family witnessed her sudden death. Her family called the ambulance immediately and performed CPR for 5 minutes on the scene. She was sent to our emergency department 30 minutes later.

On physical examination post ROSC, her Glasgow coma scale was E1VeM1. Breathing sound of coarse crepitation was noted on right side chest auscultation. The auscultation of the sound on her left side chest was unremarkable. There was no trauma mark on the physical examination. We noted that her oral cavity was clear during intubation. The “super-glue” odor was also noted during resuscitation. She was hemodynamically unstable post ROSC (pulse rate: around 130 beats per minute) and profound shock was noted (blood pressure post ROSC: 49/27 mmHg).

Arterial blood gas interpretations showed severe mixed acidosis with pH 6.74 (7.35-7.45). White blood cell count was 44.4 x1000/μL, hemoglobin was 11.9 g/dL and platelet count was 395 x1000/μL. Cardiac enzymes were normal. Other laboratory studies showed the following: serum creatinine, 1.66 mg/dL; blood urea nitrogen, 3.3 mg/dL; sodium,
141 mmol/L; potassium, 4.4 mmol/L; blood glucose, 152 mg/dL; phosphate, 4.5 mg/dL; ionized calcium, 4.6 mg/dL; uric acid, 9.5 mg/dL; C-reactive protein, 0.87 mg/L; lactate, 3.6 mmol/L; and ammonia, 549 ug/dL. She had elevated liver enzymes, with aspartate transaminase (AST) 1158 unit/L, alanine transaminase (ALT) 1328 (unit/L). Toxicology screens were negative for alcohol, PCP, BZD, amphetamine, cocaine, or cannabis.

Serial chest radiographs were obtained which showed increased infiltrates on the right side. Computed tomography (CT) of the brain showed brain edema. ECG showed sinus tachycardia, without prolonged QTc, heart rate: 130 beats per minute.

Patient received vasopressors, mechanical ventilation, empiric antibiotics, and cooling therapy based on status post successful cardiopulmonary resuscitation. Cooling therapy was started immediately after admission in the intensive care unit (ICU). We used empiric antibiotics for her aspirated pneumonia. On the 2nd day, her acidosis was improved with arterial blood gas pH 7.20, but she started to develop rhabdomyolysis. Her creatine kinase (CK) increased from 300 to 25011 unit/L. On the 3rd day, her acidosis was further improved with arterial blood gas pH 7.25. Her ammonia level dropped to 90 ug/dL and CK decreased to 13927 unit/L. Her WBC decreased to 29.2 x1000/uL. However, she was hemodynamically unstable with shock status and still received vasopressors and mechanical ventilation. On the 4th day her family refused further resuscitation because her consciousness did not recover (Glasgow coma scale: E1VeM1). Her family signed “Do Not Resuscitation” consent and finally she was discharged from the ICU on the 4th day in a critical condition.

3. Discussion

Death related to volatile substance abuse mostly results from asphyxia, suffocation, choking, and trauma due to dangerous behavior during exposure to volatile substance or in the subsequent hours after sniffing volatile substance. “Sudden sniffing death” refers to a patient with cardiovascular collapse during exposure to volatile substance or in the subsequent hours after sniffing volatile substance. “Sniffing” refers to the direct nasal inhalation from containers. Reported cases of sudden sniffing death related to volatile substance abuse is few, and most of these patients are associated with halogenated hydrocarbons abuse. Halogenated hydrocarbon abuse can cause a fatal arrhythmia. Reported cases of sudden sniffing death with aromatic hydrocarbon abuse have been even fewer, especially with toluene abuse which, at the same time, is extremely fatal. Toluene, the major component of super-glue, is a kind of aromatic hydrocarbons (C₇H₈). Cases of toluene abuse therefore have been reported extremely rarely; however, toluene abuse is prevalent around the world. Unfortunately, resuscitation of sudden sniffing death is seldom successful in the previous published reports. Our patient got ROSC after the initial aggressive resuscitation. Favorable prognosis could be expected with early diagnosis and proper interventions.

Toluene is toxic to many body systems. The most important toxicities are central nervous system (CNS) depression, ventricular arrhythmia, and metabolic acidosis. A well-described complication of toluene abuse is renal tubular acidosis and liver toxicity may also occur. Acute respiratory distress syndrome has been reported in the previous published reports. CNS depression rarely causes respiratory arrest and death. Toluene intoxication can result in cerebellar dysfunction and cranial neuropathies. Brain CT may demonstrate loss of brain mass. Fatal arrhythmias or myocardial infarction may rarely occur. Ventricular dysrhythmias may result from an increase in QT duration and QT dispersion. Yet there was no evidence of prolonged QT interval noted in our patient's ECG. Besides, our patient's ECG and cardiac enzymes were not consistent with an acute coronary syndrome event. Heavy toluene abuse can cause severe metabolic acidosis with lactate accumulation and few patients with a pH less than 6.8 survived. Toluene is metabolized to hippuric acid. Because of an excess of hippuric acid, toluene abuse can result in metabolic acidosis. Potassium is combined with hippurate anions, so the resulting is hypokalemia and weakness, especially in patients with chronic toluene abuse. Severe metabolic acidosis may be one of the possible causes of her sudden death. Our patient initially survived after aggressive resuscitation and perhaps favorable outcome could be achieved. Her CK was significantly elevated on the 2nd day and this consequence was probably owing to rhabdomyolysis. It was regrettable that her family refused further resuscitation.

Although exposure to toluene may be confirmed by detection of hippuric acid in urine or direct measurement in the blood, these laboratory data are not quickly available and don't change management priorities. Thus, these laboratory studies were not performed in our patient and exposure to toluene was confirmed by detection of the “super-glue” odor and her medical history. Management of toluene intoxication is supportive treatment. There is no specific antidote to treat toluene intoxication. Patients who have ventricular arrhythmias after toluene abuse should receive amiodarone rather than epinephrine because epinephrine can theoretically worsen arrhythmias in the irritable myocardium.

Sudden sniffing death has been documented since the 1970s. The probable mechanism of sudden sniffing death after toluene abuse may be cardiac arrhythmias, but the true mechanism is still unknown. The previous experimental evidence suggested cardiac arrhythmias be resulted from toluene abuse. Human researches are limited due to ethical concerns. Specific post-mortem features of toluene abuse have not yet been identified. Consequently, in all unnatural deaths of toluene abuse, toxicological examination is strongly recommended. Chronic heavy toluene abuse was reported to result in increases in QT interval, QTc duration, and QT dispersion, good predictors for sudden cardiac death. However, QT interval, QTc duration, and QT dispersion did not increase in our patient's ECG. Unfortunately, death from cardiac arrhythmia after toluene abuse has been unpredictable so far and thus resuscitation is rarely successful.
In conclusion, toluene abuse is associated with major toxicities including metabolic acidosis with lactate accumulation, CNS depression, ventricular arrhythmias, rhabdomyolysis, and liver toxicity. Sudden sniffing death should be considered in evaluating out-of-hospital cardiac arrest related to toluene sniffing, especially in the severe metabolic acidosis condition. Aggressive resuscitation should be carried out because favorable outcome is expected with early diagnosis and proper interventions.

References