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Troponemia Secondary to Air Duster Inhalant Abuse

Jesus Romero[1], Rachel Abboud[2], Sherif Elkattawy[2], Ana Romero[1], Omar Elkattawy[3], Abdel Azez Abu Samak[1], Razan Shamoon[2]

[1]Internal Medicine Department, RWJBarnabas Health/Trinitas Regional Medical Center, Elizabeth, New Jersey, USA

[2]Cardiology Department, St. Joseph's University Medical Center, Paterson, New Jersey, USA

[3]Internal Medicine Department, Rutgers University-New Brunswick, Jersey City, New Jersey, USA

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ABSTRACT

The abuse of inhalants has become a public health concern in the USA over the past decade. Compressed air duster cans currently available in the USA contain highly toxic substances including different hydrofluorocarbons (including tetrafluoroethane and difluoroethane) which exert a psychoactive effect on the central nervous system. Several cases of inhalant-induced lethal arrhythmia such as ventricular fibrillation evolving to torsade de pointes and leading to cardiac arrest, have been reported in the literature. Furthermore, multiorgan failure including liver and kidney injury has been described after inhalant abuse.

We report the case of a 33-year-old man found diaphoretic and with a near syncopal episode after inhalation of several cans of Surf Onn electronic duster gas cleaner, who subsequently developed acute cardiac, liver and kidney injury.

LEARNING POINTS

- Although the pathophysiological mechanisms have not been fully elucidated, the hydrofluorocarbons in air duster cans may exert a psychoactive effect through GABA receptor stimulation and NMDA receptor inhibition.
- Clinicians should maintain a low threshold of suspicion for patients presenting with multiorgan failure, predominantly cardiac arrhythmias after inhalant overdose.
- Prompt recognition and early intervention are imperative for preventing fatal outcomes such as cardiac arrest secondary to life-threatening cardiac arrhythmias.

KEYWORDS

Inhalant, electronic dusters, tetrafluoroethane, cardiac injury

INTRODUCTION

According to the National Institute on Drug Abuse (NIDA), inhalants have been the most popular substance of abuse for the past decade in the USA. Inhalants can be delivered through different pathways such as sniffing, huffing or bagging. Highly toxic substances (like tetrafluoroethane and difluoroethane) are commonly found in inhalants such as computer dusters, and may exert a psychoactive effect through GABA receptor stimulation and NMDA receptor inhibition. However, the extent of the effect has not yet been fully characterized[1]. Surf Onn is an electronic duster gas cleaner that delivers a powerful blast of air to remove dust and debris from hard-to-reach surfaces. Even though this product is free from chlorofluorocarbons, it may contain propellants commonly used in the USA such as tetrafluoroethane and difluoroethane. Tetrafluoroethene has been associated with potentially lethal cardiac arrhythmias and difluoroethane with sudden death [2]. Here we present the case of a man brought into our hospital with a near syncopal episode after inhalation of multiple cans of Surf Onn electronic duster, who was found to have troponemia, along with acute liver and kidney injury.

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CASE DESCRIPTION

A 33-year-old man with a medical history of alcohol abuse disorder was transported to the Trinitas Regional Medical Center emergency department by EMS after he was found diaphoretic and with a near syncopal episode in his car in a Walmart parking lot. He endorsed inhalation of 30–40 cans of Surf Onn electronic duster compressed gas cleaner via a mask after having an altercation with his wife. He reported inhalant abuse in the past. He denied any suicidal ideation or intent. The patient stated his last alcohol intake was 3–4 days prior to presentation, which was consistent with four beers.

On arrival at the emergency department, the patient was afebrile with a temperature of 36.1°C, hypertensive at 147/101 mmHg, tachycardic at 124 per min, non-tachypnoeic with a respiratory rate of 17, and saturating 96% on room air. He was slightly anxious, but denied chest pain, shortness of breath, nausea, emesis or abdominal pain.

The initial blood work was remarkable for troponin of 3.76 (<0.5 ng/ml), CPK of 2693 (38– 174 U/l) and lactic acid of 7.6 (0.5–2.2 mmol/l). Paracetamol, salicylate and blood alcohol levels were negative. Leucocytosis was evidenced with WBC at 20.4 (4.8–10.8 K/µl), AST was elevated at 105 (15–41 U/l), creatinine was 1.82 (0.7–1.2 mg/dl), and calcium was 7.5 (8.9–10.3 mg/dl) with albumin of 4.4 (3.5–4.8 g/dl). The urine drug screen was unremarkable. Initial EKG showed a normal sinus rhythm, with a ventricular rate of 95 per min and QTc of 429 ms (*Fig. 1*). The chest x-ray was unremarkable. Venous ABG showed pH 7.45, pCO2 32 (39–54 mmHg) and HCO3 23.5 mmol/l.

In the emergency department, poison control was contacted who suggested monitoring for developing hypoxia and supportive care treatment. The patient received a 2-litre IV fluid bolus with normal saline, and was admitted to ICU for closer monitoring.

During the hospital course, the patient remained haemodynamically stable and completely asymptomatic. Troponin I peaked at 5.25 (<0.5 ng/ml), and down trended in the following hours with a minimum value of 1.05 (<0.5 ng/ml). HbA1c was 5.3 (4.0–6.2%) and TSH level was 2.64 (0.34–5.6 mIU/l). The CPK level improved with fluid resuscitation, with a minimum value of 2239 (38–174 U/l). The follow-up EKG revealed T-wave inversion in V1–V6 (*Fig. 2*). A transthoracic echocardiogram was performed which revealed a left ventricular ejection fraction of 55–60%, normal global left ventricular systolic function, and trace tricuspid regurgitation. A plan for left heart catheterization was extensively discussed with the patient, but he refused it and decided to leave hospital against medical advice after saying he understood the risks and benefits of his decision.

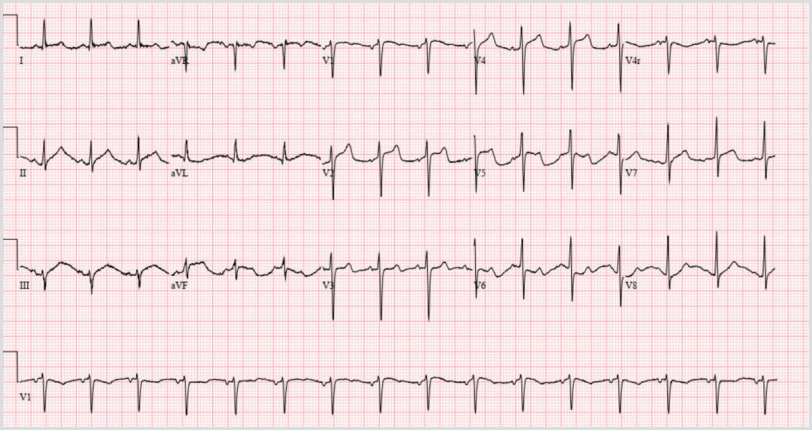

Figure 1. Initial EKG showing normal sinus rhythm

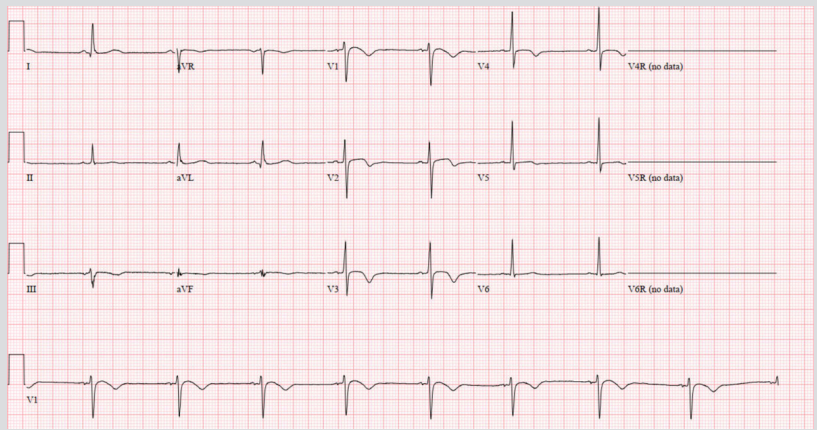

Figure 2. Follow-up EKG showing T-wave inversion in V1–V6

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DISCUSSION

An estimated 22 million Americans aged 12 and older (at least 9% of the US population) have reported the use of inhalants [3]. The use of chlorofluorocarbons in compressed air duster cans has been banned for over a decade in the USA. However, most of the cans currently available use hydrofluorocarbons such as tetrafluoroethane and difluoroethane as the propellant.

Several cases of huffing associated with cardiac, kidney and liver failure have been reported in the literature. One report described a 38-year-old woman who developed torsade de pointes which degenerated into ventricular fibrillation leading to cardiac arrest after tetrafluoroethane inhalation. Initial laboratory tests revealed a high sensitivity troponin I level of 2406 ng/l and elevated serum lactate of 6.3 mmol/l. An EKG revealed a rate-corrected QT interval (QTc) of 517 ms with multifocal ventricular ectopy [4]. This patient had also demonstrated troponemia secondary to inhalant abuse, but it was more significant than in our patient in the setting of post-cardiac arrest myocardial injury.

Cates and Cook describe the case of a 34-year-old man with a history of recurrent and heavy hydrocarbon huffing who developed a generalized seizure and torsade de pointes leading to cardiac arrest. The Advanced Cardiac Life Support protocol was initiated and the patient was successfully resuscitated. In the following days, the laboratory tests were remarkable for lactic acidosis (4.7 mmol/l), rhabdomyolysis (CK 5058 U/l), shock liver (AST 17,432 U/l), acute kidney injury (creatinine 1.86 mg/dl) and troponemia (20.2 ng/ml) [5]. This case demonstrated the impact of acute inhalant use leading to multiorgan involvement including cardiac, renal and liver injury, findings similar to our patient's presentation.

Furthermore, Cao et al. described a 35-year-old man who presented with chest pain after inhaling multiple duster cans every day. The EKG showed sinus tachycardia and prominent T-wave inversions in the inferior lead consistent with ischaemia. Laboratory tests were remarkable for acute kidney injury, transaminitis, and troponemia with a troponin I level of 1.85 ng/ml. The patient was started on the acute coronary syndrome protocol, and subsequently underwent cardiac catheterization which showed clear coronary arteries consistent with acute cardiac injury secondary to duster inhalation [6]. Similarly to our case, this patient developed troponemia and EKG changes consistent with ischaemia. Unfortunately, our patient decided to leave hospital against medical advice before further cardiac work-up could be performed.

We describe the case of a 33-year-old man who presented with a near syncopal episode after inhalation of multiple duster cans. Initial laboratory tests were consistent with acute cardiac, kidney and liver injury. These findings were similar to numerous cases previously reported in the literature which demonstrated a correlation between duster can inhalation and cardiac injury ranging from troponemia likely secondary to demand ischaemia, to torsade de pointes leading to ventricular fibrillation and cardiac arrest. A limitation of our case report is the lack of a complete cardiac work-up since our patient decided to leave hospital against medical advice even after extensive information had been given to him regarding the importance, risks and benefits of left heart catheterization.

Clinicians should maintain a low threshold of suspicion for patients presenting with acute coronary syndrome and/or multiorgan failure after inhalant overdose. Further studies are needed to fully elucidate the pathophysiological changes triggered by inhalant abuse.

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