A Case of Multisystem Organ Failure in a Patient With Inhalant Use Disorder

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Inhalant use, or "huffing" of halogenated hydrocarbons, has an associated euphoria, making it an attractive form of recreational drug use. Halogenated hydrocarbons are compounds that can be found in household products such as cleaning supplies, including canisters of compressed air used to air dust keyboards ("dusters"). We present a case of a previously physically healthy man who developed severe multisystem organ failure due to hydrocarbon toxicity from recreational use of compressed air.

Case Report

The patient was a 32-year-old man with a history of alcohol, tobacco, and inhalant use disorders, who developed shortness of breath and chest pain after inhalation of six cans of compressed air. He presented to an outside hospital emergency department (ED) where he experienced rapid clinical deterioration. Initial electrocardiogram showed sinus tachycardia with a heart rate of 170 beats/minute. Shortly after, he experienced 4 episodes of alternating pulseless electrical activity and supraventricular tachycardia with torsades de pointes, requiring repeated defibrillation, amiodarone, and lidocaine infusions. He was placed on multiple vasopressors for management of hypotension. An echocardiogram performed after the episodes of cardiac arrest revealed an ejection fraction of 5%. He was taken to the cardiac catheterization lab for bilateral heart catheterization and had a catheter base ventricular assist device placed for cardiac support. He then began having worsening respiratory distress, which ultimately led to intubation and mechanical ventilation. The specific pulmonary diagnosis was acute respiratory failure with hypoxia and hypercapnia secondary to inhalation of halogenated hydrocarbons. The patient was transferred to our facility for extracorporeal membrane oxygenation (ECMO). On admission, the patient was found to have a creatinine level of 3.35 mg/dL, blood urea nitrogen of 28 mg/dL, and an estimated glomerular filtration rate (eGFR) of 21 mL/minute. He was diagnosed with acute renal failure and acute tubular necrosis secondary to cardiogenic shock and was placed on continuous veno-venous dialysis. The patient was also diagnosed with shock liver with an admission aspartate transferase of 16,380 U/L and alanine transaminase of 6,615 U/L.

After successful extubation and transitioning to room air, the addiction psychiatry team was consulted to discuss substance use with the patient. This patient began using compressed air canisters when he was 16 years old and reported normally inhaling 3 to 4 cans at a time up to 3 to 4 times per week. He reported using this amount at the same frequency with consistent effects, indicating that he did not develop tolerance to inhalants. He was able to recall the night he went to the ED for shortness of breath and chest pain and remembered having inhaled 5 to 6 cans of compressed air shortly prior to developing symptoms. He reported using more than his usual amount that day but did not identify any particular reason or trigger for increased use. The patient also shared his history of alcohol use disorder and reported drinking a fifth (750 mL) of vodka daily, often in the setting of inhalant use. He had previously been in residential treatment for alcohol use.

The patient showed clinical improvement during his hospital stay. On hospital day 2, a repeat echocardiogram revealed an improvement of ejection fraction to 60%–65%. He was transitioned to intermittent hemodialysis on hospital day 8. During the hospital stay, his creatinine level improved to 1.66 mg/dL and eGFR peaked at 48 mL/minute. The patient's liver function tests improved to within normal range by the time of discharge. He experienced delirium earlier in his hospitalization but appeared to approach his cognitive baseline over the course of his hospital stay. He was recommended to obtain neuropsychological testing in the outpatient setting to further characterize any specific neurocognitive deficits that may have persisted. He worked with physical and occupational therapy while hospitalized to regain strength and was subsequently discharged home with outpatient hemodialysis on hospital day 16. The patient's follow through on neuropsychological testing and substance use treatment is unknown. However, at an outpatient nephrology appointment 1 month after discharge, his creatinine level had improved to normal range (1.2 mg/dL) with an eGFR of 71 mL/minute, and intermittent hemodialysis had been stopped altogether.

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Prim Care Companion CNS Disord 2023;25(3):22cr03349

To cite: Hoody L, Cronin M, Balasanova AA. A case of multisystem organ failure in a patient with inhalant use disorder. *Prim Care Companion CNS Disord*. 2023;25(3):22cr03349.

To share: https://doi.org/10.4088/PCC.22cr03349

Discussion

Halogenated hydrocarbons are lipid soluble and are systemically absorbed via the lungs through inhalation, after which they can rapidly diffuse throughout the body and into the central nervous system (CNS). Based on animal studies, they appear to cause CNS depression by affecting several neurotransmitter receptors in a dose-dependent fashion. Both antagonist activity on excitatory *N*-methyl-D-aspartate receptors and potentiation of inhibitory γ-aminobutyric acid receptors have been implicated.[1,2] The specific clinical effects that have been observed include somnolence, headache, ataxia, dizziness, blurred vision, weakness, fatigue, lethargy, stupor, seizures, and coma.[1,3] In addition, hypoxia can result from displacement of oxygen in alveoli or from aspiration pneumonitis, which can then cause secondary CNS toxicity.[4] Potential long-term CNS sequelae include neurocognitive impairment, cerebellar dysfunction, and peripheral neuropathy.

Major cardiovascular problems that can occur after hydrocarbon inhalation include cardiac arrhythmias and myocardial dysfunction. Halogenated hydrocarbons are particularly known for causing fatal ventricular arrhythmias. A phenomenon where a patient has cardiovascular collapse associated with inhalation called "sudden sniffing death" has been reported with all classes of hydrocarbons and although rare is unpredictable and can even occur in people who are using inhalants for the first time.[1,5] This condition is thought to be due to catecholamine sensitization of the myocardium, which can also be accentuated by hypoxia experienced during inhalant use.[6] This results in increased susceptibility to delayed after-depolarizations, leading to ventricular dysrhythmias induced by catecholamines.[1,7,8] Our patient was found to have severe biventricular failure, shock liver, acute renal failure, and necrotic foot ulcer that were thought to be secondary to hypoxia experienced during repeated episodes of cardiac arrest.

Several previous reports[9–11] have described the potential cardiotoxic effects of halogenated hydrocarbons such as cardiac arrhythmias, cardiomyopathy, and cardiac arrest. To our knowledge, this is the first report of a patient with multisystem organ failure due to inhalant use. Our patient experienced repeated episodes of cardiac arrest and decompensated heart failure to the point of requiring ECMO, in addition to severe acute renal failure requiring dialysis, respiratory failure prompting intubation, and CNS depression. This case shows that the hydrocarbon toxicity from inhalant use can result in multisystem organ function in an otherwise healthy adult.

Conclusion

There is limited literature on the effects of inhalants and their toxic hydrocarbons on multiple organ system functions. Our patient experienced multisystem organ failure after inhaling 5 to 6 cans of compressed air, requiring 2 weeks in the hospital and intensive care. This case aims to shed light on the possible presentations and complications of inhalant use, as well as to show the potential for rapid clinical improvement with prompt recognition and management.

Published online: May 2, 2023.
Relevant financial relationships: None.
Funding/support: None.
Patient consent: Consent was received from the patient to publish the case report, and information has been de-identified to protect anonymity.

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