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CLINICAL VIGNETTE

Skeletal Fluorosis and “Sniffer’s Dermatitis” After Inhalant Abuse with 1,1-Difluoroethane

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A 37-year-old male with history of post-traumatic stress disorder, depression, and polysubstance abuse presented to the Emergency Department (ED) for burns and swelling of the face after inhalant abuse. Ten days prior the patient had begun sniffing a computer duster as an intoxicant, which contained the propellant 1,1-difluoroethane (DFE). He had been using up to 10 canisters daily.

On presentation to the ED, the patient’s initial vital signs were: temperature 97.5°F, pulse 106 beats/min, blood pressure 148/98, respiratory rate 18 breaths/min, and oxygen saturation 100% on room air. Physical examination was significant for perioral swelling/blistering and a first degree burn to the right hand.

Laboratory values were notable for an elevated alkaline phosphatase (ALP) of 192 U/L. Chemistry and liver panels were otherwise unremarkable. Right hand X-rays were only notable for soft tissue swelling.

After psychiatric evaluation and poison control consultation, the patient was deemed to have no acute indication for admission. The patient was provided with substance abuse, social work, and primary care follow up.

Over the following three months the patient continued inhalant abuse with up to 10 cans of computer duster daily. He developed bilateral hand, foot, and knee pain with persistent swelling and tenderness to bilateral hands. ALP continued to rise as high as 644 U/L, which was confirmed to be bone specific ALP. Repeat hand x-rays at that time were significant for new fluffy periosteal bone formation with mild diffuse soft tissue swelling.

Rheumatology confirmed elevated serum fluoride to 1.6 mg/L (normal <0.13 mg/L). The patient’s joint pains, swelling, laboratory tests, and radiographic findings were attributed to skeletal fluorosis due to inhalant abuse of computer duster containing 1,1-difluoroethane (DFE). It was again recommended the patient abstain from further inhalant abuse.

Over the following several months the patient had intermittent medical and psychiatric follow up. His symptoms continued as did his pattern of inhalant abuse until nine months after his initial presentation when he agreed to inpatient substance abuse treatment.

Discussion

1,1-difluoroethane (DFE) is a volatile organofluorine compound widely used as a refrigerant and propellant in products such as spray cleaners for electronic devices.¹ When “huffed” or inhaled, it causes euphoria and intoxication.¹ Because of these effects, DFE has become a popular substance of abuse.² This case highlights the multiple organ systems that can be affected in unique ways by inhalant abuse in general, and DFE in particular.

In addition to the acute central nervous system effects, our patient also experienced the classic dermatologic perioral eczematoid “sniffer’s rash”. He subsequently experienced skeletal fluorosis—a painful osteomalacia caused by excessive fluoride deposition in bone.³

In addition to the findings in our patient, DFE toxicity has also caused a number of other complications, including cardiomyopathy,⁴ pneumopericardium,⁵ acute kidney injury,⁶ fulminant hepatitis,⁴ airway compromise,⁷ and angioedema.⁸ However, inhalant abuse of volatile hydrocarbons, such as DFE, can pose an even greater threat.

In the 1960s, reports surfaced of teenagers seemingly spontaneously dying after sniffing volatile hydrocarbons.⁹ This phenomenon was termed Sudden Sniffing Death Syndrome. In this syndrome, a patient who sniffs volatile hydrocarbons experiences sudden cardiovascular collapse after a surge of catecholamines, often triggered by physical exertion or surprise.⁹ It is thought that volatile hydrocarbons sensitize the myocardium to catecholamines which can lead to ventricular tachydysrhythmias which are refractory to standard therapy.¹⁰ This syndrome can occur even in first time users after a single session of inhalant use.²

During a resuscitation, it is important to be able to recognize a patient presenting with ventricular fibrillation in the setting of Sudden Sniffing Death Syndrome because the management differs from current American Heart Association (AHA) guidelines.⁹ Current AHA Guidelines recommend administration of epinephrine for ventricular fibrillation that persists after at least one attempt at defibrillation and 2 minutes of CPR.¹¹ However, in patients presenting with ventricular fibrillation secondary to Sudden Sniffing Death Syndrome, administration of epinephrine (and norepinephrine) is likely to exacerbate their condition.

These patients should be treated with standard cardiopulmonary resuscitation and early defibrillation. Since these tachyarrhythmias are usually refractory to defibrillation, early use of amiodarone or lidocaine should be considered.¹² Case reports have also suggested that use of propranolol or esmolol may counter the myocardial sensitization and help terminate the dysrhythmia.¹³

This case highlights the multiple organ systems that can be affected by inhalant abuse including intoxication, “sniffers dermatitis”, and skeletal fluorosis. Many of these sequelae can be treated with abstinence and supportive care alone. Sudden Sniffing Death Syndrome, however, requires special mention since standard treatments may actually worsen the condition and alternative agents may prove life-saving.

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