Inhalation of Aerosol Sprays can be Dangerous: Case Report

Rouhollah Qurishi1*, Anouk Bergmans1, Anton JM Loonen2, HPCM Heijmen3 and Cornelis AJ De Jong4

1Novadic-Kentron Addiction Care Network, Sint-Oedenrode, The Netherlands
2University of Groningen, Groningen Research Institute of Pharmacy, Unit of Pharmacotherapy, Epidemiology and Economics, Antonius Deusinglaan, Groningen, The Netherlands
3Cardioloog, Elkerliek Ziekenhuis Helmond, Afdeling Cardiologie, Helmond, The Netherlands
4Nijmegen Institute for Scientist-Practitioners in Addiction (NISPA and Behavioral Science Institute, Radboud University, The Netherlands

*Corresponding Author: Rouhollah Qurishi, Novadic-Kentron Addiction Care Network, Sint-Oedenrode, The Netherlands.

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Abstract

Inhalation of aerosol sprays (IAS) is widely practiced because these products are legal, cheap, and relatively easy to obtain. The heart and brain are particularly susceptible to the toxic effects of aerosols. Ventricular dysrhythmias are the leading cause of death from IAS.

In this paper, we present two cases of life threatening misuse of aerosol deodorant sprays. The first patient was admitted to an intensive care unit because of a previous cardiac arrest due to extensive deodorant inhalation. The second patient developed seizures and cardiac irregularities after inhaling deodorant.

Keywords: Volatile Organic Substance; Deodorant; Abuse; Complication; Psychoactive Substances; Substance Related Disorders

Introduction

It is well known that young people start using psychoactive substances in early adolescence [1]. Some will continue to use such substances and become addicted. In the experimental phase, not only the well-known substances such as alcohol, nicotine or cannabis will be used but also volatile substances like butane, propane or nitrous oxide. Cigarette lighters and gas cartridges from whipped cream canisters provide a pure and readily-available source of these volatile substances, but many other products, such as aerosol deodorant sprays use gases as propellants, but these are not pure sources. It is these impure sources that are resorted to when the common psychoactive substances and pure sources cannot be found. In the late 1990s, the prevalence of the inhalation of aerosol sprays (IAS) was very high in the United States (US) but has since been in decline [2]. During this period of time, anti-drug campaigns started educating young people about the hazards of IAS. The United Kingdom (UK) started the same educational campaigns in 1992, which resulted in a substantial reduction in aerosol spray misuse among school children aged 11 - 15 years: from 21% in 1995 to 9% in 2007 [3,4]. However, despite the reducing misuse rates in the US and UK, IAS seems to be increasing in other countries.

The most commonly misused substances in recent studies are fuels such as butane or petrol and compressed gas dusters and deodorants that may contain fluorocarbons and/or butane, isobutane, propane alpha-isomethyl ionone, benzyl alcohol, benzyl benzoate, benzyl salicylate, butylphenyl methylpropional, citral, coumarin, eugenol, geraniol, and hydroxyisohexyl-3-cyclohexene. Many deodorants and antiperspirants also contain aluminium zirconium tetrachlorohydrex gly and aluminium chloride hydrate, propylene glycol, triclosan and stearine. Breathing large amounts of aluminium dusts can result in lung problems, such as coughing or changes so significant as to show up in chest X-rays. Systemic exposure to aluminum is associated with central nervous system (CNS) toxicity [5] and chronic exposure with an increased incidence of Alzheimer’s disease [6]. Aluminum exposure has also been linked to decreased performance in certain tests reflecting nervous system functions [7-9].

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Repeated use of antiperspirants containing triclosan is also a potential health risk. There is evidence of high acute toxicity via inhalation. A repeated dose inhalation toxicity study found that triclosan is a respiratory irritant in rats [7]. For the inhalation exposure calculation, the concentration of triclosan in body spray products is taken as 0.3%, and the duration per application is assumed to be 15 minutes [7]. In addition to adverse effects on the respiratory tract, triclosan has an adverse effect on muscle contraction, thus may affect heart and skeletal muscles [8,9].

There are many different household items that can be misused. Adhesives are a readily abused product because they often consist of toluene, naptha (a mixture of liquid hydrocarbons), xylene, hexane, and/or benzene. Spray paints can also contain the same compounds as adhesives, depending on the brand and colour. Other substances, which are frequently misused include solvents containing acetone (acrylic nail remover and nail polish remover) and mothballs.

There are several methods used when inhaling aerosols, and these depend on the particular product being misused. For instance, the nozzle of nitrous oxide canisters or compressed gas dusters may be aimed directly into the mouth, opened, and inhaled (a procedure known as “dusting”). Other products, like paints and adhesives, can be sprayed or placed into a bag, and the gaseous mixture within inhaled. This is referred to as “bagging”. In addition to that, some fuels, solvents, and adhesives like butane, acetone, and glue can be inhaled directly from their containers in a method referred to as “sniffing” whilst the term “huffing” is used when a substance is applied to a cloth and placed over the nose and mouth (conf. “waterboarding”) [10].

Clues to misuse are often subtle and may include the patient’s proximity to an aerosol or inhalation paraphernalia when found intoxicated, or the presence of skin burns, blisters, pigments, or rashes, and chemical odour on the patient. Treatment of intoxication begins with management of the patient’s airway, breathing, and circulation. Exogenous catecholamine should be avoided if possible because of the potentially “sensitized” and irritable myocardium. In the case of ventricular dysrhythmias, direct current defibrillation and/or beta-adrenergic receptor antagonism should be utilized [11].

Cases
Case 1

Patient A is a 21-year-old young man diagnosed with alcohol and cocaine dependence. He started using cocaine and alcohol daily from the age of 15. Eventually, the drug abuse and stress caused by traumatic events evolved into an acute form of a psychotic mental state. Patient A was, by judicial order, compulsorily admitted to a rehabilitation facility for young adults.

During his inpatient treatment, Patient A was diagnosed with a post-traumatic stress disorder, dysthymia, an intellectual disability and a personality disorder with paranoid, borderline and antisocial features. After having been admitted to the facility. While being admitted, because of the lack of other psychoactive substances, Patient A inhaled substantial quantities of deodorant. After he was discharged from the rehabilitation unit, Patient A went home and inhaled seventeen cans of deodorant. After this, he had a cardiac arrest due to ventricular fibrillation and resuscitation was necessary. Defibrillation was used several times to stabilize his cardiac condition and thereafter he was held in a medically induced coma. A decision was made to sedate, intubate and cool Patient A for 24 hours. Due to aspiration they started with antibiotic therapy using amoxicillin/clavulanic acid. Screening for toxins was negative. After 24 hours the cooling phase was ended and body temperature was actively increased. No neurological deficit had occurred and Patient A’s respiratory and circulatory systems were stable. Following his uncomplicated recovery a full cardiac investigation was performed. Echocardiography showed normal dimensions for all heart chambers, normal left and right ventricular function, all valves normal and functioning correctly, and no signs of pulmonary hypertension. Coronary angiography showed normal coronary arteries. As there was the possibility of electrical heart disease, an Ajmalin-test was performed to exclude Brugada Syndrome, and this showed nothing abnormal. Finally, bicycle test results were normal, and in particular there were no signs of catecholaminergic polymorphic ventricular tachycardia.

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Case 2

Patient B is a 16-year-old boy with a history of cannabis dependence and ecstasy (MDMA) abuse. At the age of 13, Patient B started smoking 12 cigarettes a day and began using cannabis. He said “this is the only way I can feel calm and happy and forget about my problems”. Around the age of 14, he started using cannabis daily, smoking around eight joints a day, every day of the week. At around this time, Patient B also started using ecstasy pills. In the two years prior to his involvement in this study, he reported consuming two to three ecstasy pills every four day period. He also experimented with amphetamine and nitrous oxide.

At the age of 16, Patient B was admitted to an inpatient rehabilitation facility for young adults with substance related disorders. Besides the dependence, he was diagnosed with an intellectual disability and problems within his primary support group. Due to his limited mental capabilities, he seemed to be highly overstimulated from a young age. During treatment he suffered from hallucinations and multiple seizures. During these seizures, his muscles where partially tensed and he didn't respond to verbal communication. His blood pressure was systolic = 130 and diastolic = 75 with 105 irregular beats per minute. Urine tests did not detect any drugs of abuse. Patient B didn't report having a history of epilepsy but admitted inhaling deodorant over the past two months by using a towel to prevent the gas from evaporating while inhaling it. Inhaling deodorant gave him an intoxicated and numb feeling in his head, which made him feel calm and relaxed. The addiction medicine physician considered referring him to a neurologist but Patient B did not want this as then his parents would find out. After confessing to inhaling deodorant and having been warned about the risk of seizures as a result of inhaling such substances, Patient B agreed to being more closely monitored by the medical staff.

Discussion

Here we presented two cases of life threatening incidents after abusing deodorant aerosol sprays, with both patients unaware of the dangers of doing so. In this discussion we apparently know the most for toluene and want to limit ourselves to this in the context of this cases.

When volatile organic substances are inhaled they easily enter the body via the lungs, moving quickly and efficiently across the alveolar-capillary membrane. Once inside the body, they easily dissolve in the blood and are carried to other bodily tissues. Given their high lipophilicity, they are distributed initially to tissues with high lipid content like the nervous system, liver, kidneys, and adipose tissue [12]. In the cardiology literature, there are only a few case reports relating to aerosol abuse as IAS and acute cardiac death is a rare condition for cardiologists to encounter. Aerosol substances sensitize the heart for circulating catecholamines in such a way that sudden alarm or exercise may precipitate sudden cardiac death [13]. The main mechanism of cardiac arrest is cardiac (ventricular) arrhythmia, but anoxia, respiratory depression and vagal stimulation along with aspiration of vomit or trauma may be contributing factors in the sudden death. Timely resuscitation is often impossible, since the majority of deaths are unwitnessed and therefore never reach the hospital for further critical care. Differential diagnosis of intoxication by IAS should include coronary artery disease as well as catecholamnergic polymorphic ventricular tachycardia, Brugada Syndrome and of course cardiomyopathies which could be causing malignant ventricular arrhythmias.

Aerosol sprays are misused for their rapid onset CNS effects. While their exact mechanisms of action are not known, many in vitro studies have been undertaken and these identified potential receptor-mediated mechanisms. Specific targets perceived to be responsible for clinical effects include increases in N-methyl-D-aspartic acid (NMDA), gamma-aminobutyric acid A (GABAA), glycine, nicotine, dopamine, and serotonin receptors as well as nerve membrane ion channels [14]. Toluene, for example, inhibits NMDA receptor-mediated currents in a concentration-dependent manner [15]. Toluene increases dopamine levels in several parts of the brain including the striatum, prefrontal cortex, and ventral tegmental area [16,17]. Increased dopamine excreted from the mesolimbic dopamine neurons is considered to contribute to the psychological dependence of toluene.

Few clinical laboratories are able to analyze for specific aerosols. In acute toxicity, a high degree of suspicion of IAS is required to rapidly diagnose and stabilize the patient. Patient history and physical examination can guide clinicians to suspect intoxication by IAS. For example, the discovery of IAS-related paraphernalia at the scene may indicate volatile substance abuse to clinicians; specific findings

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during clinical examination such as paint pigment seen on the patient’s face or hands; hydrofluorocarbons, such as dusters, may result in frostbite to the mouth or hands; or the smell of fuel or solvents may be detected on the patient’s clothes or body [11]. For outpatients, when IAS is suspected, urine and blood samples may be sent in air tight containers for testing in reference laboratories where gas chromatography, headspace gas chromatography, or atomic absorption spectroscopy (AAS)-coupled chromatography can be used to detect volatile substances and their metabolites. Care must be taken when collecting and transporting the samples given the volatile nature of the substances. Additional ancillary findings that may aid in diagnosis will be discussed in regards to that particular substance [11].

Some controversial research has suggested the development of physical dependence after long-lasting inhalant abuse leading to a withdrawal syndrome upon abstinence [18]. A case of severe withdrawal syndrome manifested by tremor, hypertonia, ataxia, irritability, and deteriorated altered mental state was reported in a person who misused toluene for many years [19].

Conclusion

The two cases reported here illustrate that misuse of aerosol spray, particularly deodorant spray, can lead to serious life threatening complications. As differential diagnosis of intoxication by IAS is challenging with other cardiomyopathies, it seems important to educate addiction specialists and acute care clinicians about the misuse of aerosol sprays in order that they take rapid action to help prevent serious complications.

Although there is evidence demonstrating the addictive potential of IAS, evidence- and practice-based treatment is lacking.

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