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Review

Medico legal investigations into sudden sniffing deaths linked with trichloroethylene

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ABSTRACT

Sudden deaths attributed to sniffing trichloroethylene are caused by the abuse of this solvent which produces pleasant inebriating effects with rapid dissipation. In the event of repeated cycles of inhalation, a dangerous and uncontrolled systemic accumulation of trichloroethylene may occur, followed by central nervous system depression, coma and lethal cardiorespiratory arrest.

Sometimes death occurs outside the hospital environment, without medical intervention or witnesses and without specific necroscopic signs.

Medico legal investigations into sudden sniffing deaths associated with trichloroethylene demand careful analysis of the death scene and related circumstances, a detailed understanding of the deceased's medical history and background of substance abuse and an accurate evaluation of all autopsy and laboratory data, with close cooperation between the judiciary, coroners and toxicologists.

1. Introduction

Trichloroethylene is a chlorinated hydrocarbon compound classified as a halogenated industrial solvent, which is volatile at room temperature, and which was used as an inhalational general anaesthetic until the 1970s.

It is a non-inflammable clear, colourless liquid with a sweet smell over the odour threshold at approximately 100 parts per million in air. It produces irritation of the eyes and lungs at over 1000–1200 parts per million in air and causes drowsiness and lethargy within a few minutes at 2000–3000 parts per million, just like inhalation agents used in general anaesthesia.[1–3]

Its main use in the past was as a degreaser for metal parts in the automotive or aeronautics industries and as a catalyst for the extraction of vegetable oils from plants and in coffee decaffeination. Nowadays it is found in a variety of consumer products such as spot removers, dry-cleaning agents, degreasers, paint removers and typewriter correction fluids.[1–5]

Trichloroethylene is often used for volatile substance abuse (VSA) in which volatile compounds are intentionally inhaled in order to produce dose-related effects on the central nervous system, which alter the level of consciousness, and cause feelings of euphoria and disinhibition, resulting in an intense, pleasurable sensory experience which dissipates rapidly.[4]

Proportional to the alveolar fraction, trichloroethylene, like many other volatile substances, is quickly absorbed through the alveolo-capillary membranes and produces neurological effects similar to the early stages of inhalational general anaesthesia.

Acute intoxication, after an initial stimulating rush with disinhibition, excitation, visual hallucinations and impulsive behavior, lasts for a few minutes and is followed by light-headedness, drowsiness and sleep. Repeated cycles of inhalation may cause a significant accumulation of trichloroethylene in brain and fat tissues which is then followed by a slower clearance generally lasting from 1 to 6 h.[4–7]

Flanagan et al. reported that the mechanism of death, produced by a sizeable and rapid increase in the inspiratory fraction of the inhaled halogenated solvent compounds, is due to fatal cardiac arrest after dysrhythmias or vasovagal reflexes, or after hypoxia and hypercapnia following central respiratory depression.[8]

Demise may occur during inhalation itself (the phase involving compartmental distribution from alveolar to blood and brain

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districts) or during the few hours following an acute cardiorespiratory crisis (the phase involving relatively slow dissipation from lipid-rich cell membranes of the brain to the blood and alveolar districts).[4–12]

When the deliberate inhaling of vapours is repeated over a brief period of several minutes it may produce a high and uncontrollable inspiratory alveolar concentration of the compound and may cause sudden death due acute toxicological effects such as central nervous system depression, coma and cardiorespiratory arrest.[13–15]

The procedures mainly used by volatile substance abusers are:

a) the direct inhalation of a compound from its original container through the mouth or nose (sniffing or snorting);
b) with a saturated rag placed over the nose and mouth or stuffed inside the mouth (huffing);
c) the placing of a plastic bag, containing the substance or a saturated rag, against the face or over the head (bagging) which dangerously increases the concentration of vapours and produces a rebreathing mechanism as in closed circuit general anaesthesia;
d) the spraying of aerosol products directly into the nose or mouth (glading);
e) the drinking or squirting of substances directly into the mouth is less frequent.[4,9,14,16]

Sudden sniffing deaths, due to solvents or trichloroethylene abuse, are fatal events which generally leave non-specific signs in a post-mortem examination (e.g. acute cardiorespiratory failure, post-anoxic brain damage, acute hepatic or renal failure), with the result that the exact cause of death cannot be identified, leading forensic pathologists to conclude that death was due to cardiorespiratory decompensation of unclear origins.[3,16–18]

2. Epidemiology

Different authors have reported, both recently and over the last few decades, an increase in volatile substance abuse (VSA) in various areas of the world, involving children, adolescents and adults who frequently live in impoverished social conditions, and who have a history of drug dependence or who are experimenting with drugs or attempting to come off drugs. Users of volatile substances are generally described as chronic inhalant abusers (people who predominantly abuse inhalants), or polydrug intermittent users (subjects who abuse inhalants when their drugs of choice are unavailable) or experimenters (generally adolescents who are experimenting with different substances such as cigarettes, alcohol or marijuana).[4,6,7]

Volatile substance abuse (VSA) often occurs against a background of poverty, in both urban and rural settings, delinquency, incarceration, antisocial attitudes, and family disorganization or conflict. Among adults it may be encouraged by the low cost of the products or by particular occupations which give easy access to a variety of abusable compounds such as volatile solvents, glues, adhesives, correction fluids, fuels, air fresheners, lighter refills, halocarbon fire estinguishers, aerosol propellants or anaesthetics.[4,5,8–10,13]

Many industrial, commercial or household products contain a mixture of compounds including not only chlorinated hydrocarbons like trichloroethylene but also aliphatic hydrocabons, aromatic hydrocarbons, ketones or others.[1,2,5,14]

In the earliest reports on sudden sniffing deaths linked with trichloroethylene, the substances used were stain-removers (Clearifield, 1970), cleaning agents and paint thinners (Alha, 1973) and typewriter corrections fluids (King, 1985 and Troutmann, 1988).[3,5,19,20]

The most common products containing trichloroethylene are degreasers, spot removers and typewriter correction fluids, which are cheap and readily accessible household products, paint removers and industrial solvents. In some degreasers or spot removers the trichloroethylene may be mixed with other halogenated substances (such as 1,2-dichloropropane; 1,2-dichloroethane; 1,1,1 trichloroethane; 1,1,2,2 tetrachloroethane; 1,2-dichloroethylene; tetrachloroethylene) in order to strengthen its chemical action. However, these can produce synergic toxicological effects when inhaled.[1,2,5,14]

Ramon et al. reported a frequency of 16% in the use of halogenated solvents among compounds employed for volatile substance abuse (VSA) while Williams et al. reported US data indicating that the majority of deaths due to volatile substance abuse involve the use of volatile halogenated solvents (included trichloroethylene), fuels and anaesthetics.[4,13]

Troutman reported three cases of death associated with the intentional inhalation of typewriter correction fluid containing two volatile halogenated hydrocarbons (trichloroethylene and 1,1,1 trichloroethane) which underline the lethal synergistic effects of products containing mixtures of different solvents.[5]

3. Acute toxicity

Products containing trichloroethylene are frequently used by solvent abusers because of the rapid onset of pleasant feelings of inebriation preceding the depressive neurological effects, given that the central nervous system is the main target organ for trichloroethylene in humans.[1,3,4,6]

As is typical of all organic solvents, the cerebral effects derive from the lipophilic interactions of trichloroethylene with the neuronal membranes, which allow the compound to gain rapid access to the brain. This occurs after crossing the alveolo-capillary membranes and achieving a steady state concentration in alveolar, blood, muscle, fat and neuronal regions, thus generating neurological effects similar to those occurring during the induction phase of general inhalational anaesthesia.[1,3,4,6]

After the interruption of trichloroethylene inhalation the neurological effects persist so long as the alveolar-blood-brain steady state maintains the chemical interactions of the compound with the neuronal membranes through aspecific or specific receptorial contacts, followed by an initial recovery, due to the intervention of pulmonary ventilation and subsequent liver metabolism and renal excretion. Within a few minutes of exposure at 2000 parts per million or over, the subject experiences headache, nausea, vomiting, abdominal pain, dizziness, visual disturbances, hallucinations, light-headedness, confusion and ataxia, which are generally followed by stupor and lethargy.[1,3,4,6]

Systemic symptoms due to brief inhalational exposure generally resolve almost completely within a few hours of exposure but may last for longer after repeated cycles of inhalation.[1,3,4,6]

Fatal coma, respiratory depression and cardiac malignant arrhythmias with acute heart failure and pulmonary oedema may occur due to prolonged (more than 5 min) and high-level exposure (over 2000 parts per million). Acute liver and renal failure and haematological disorders (decreased platelet count, diffuse intravascular coagulation causing splanchnic thrombosis and extensive aspecific abdominal pain) may also occur during the hours following exposure.[1,3,4,6]

Other indirect acute causes leading to sudden sniffing death are the result of altered sensorium, such as suffocation (from a loss of control of the upper airways or the use of plastic bags) and aspiration of vomitus (loss of airway reflexes) or endulging in dangerous behaviour which results in trauma, motor vehicle