Solvent abuse: a case report and a review

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Abstract

The deliberate inhalation of solvents among children and adolescents "for kicks" is becoming more common in the West. It was generally regarded as a relatively harmless practice and consequently little attention had been paid to the isolation of the toxic agent from the variety of substances used. It is now well recognised that solvent abuse not only can result in sudden death but also cause pathological changes to the liver, kidney, brain, heart and lungs. A case of toluene associated death in Malaysia is discussed both from a medico-legal and pathological standpoint.

Key words: Solvent abuse, toluene, glue.

INTRODUCTION

"Glue sniffing" or solvent abuse as a habit was recognised in the United States round 1960 and a decade later attention was drawn in the United Kingdom to the problem. Commonly used solvents are shown in Table 1. Kupperstein and Susman (1968)' stated that "a number of communities in widely separated parts of the United States began reporting a high incidence of 'glue sniffing' around 1960."

In a study of 12 fatalities,2,3 8 deaths were due to suffocation by a plastic bag, and in two cases death was presumed to be caused by intoxication although not confirmed by analysis. In the United Kingdom between 1970 and 1978 only 45 deaths were reported from solvent abuse although the specific cause of death was only ascertained in 35 cases and only in 4 cases was death attributed to the toxic effects of vapour.4

The first report of a case of addiction to glue sniffing in Great Britain was given by Meny and Zachariadis in 1962.5 Over 100 people died from the effects of solvent sniffing in Japan in 1963 and in 1969 the total rose to 161. Twelve deaths occurred in Finland between 1968-1971 and sporadic reports of individual deaths from glue sniffing have also been reported in Canada and Scotland.6 No case of solvent abuse death appears to have been reported in Malaysia.

CASE REPORT

The decomposed body of an eighteen year old Malaysian male was recovered from a dry manhole. The deceased was a known "glue sniffer."

<table>
<thead>
<tr>
<th>Common solvents</th>
<th>Toxic constituents*</th>
</tr>
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<tbody>
<tr>
<td>Lighter fluid</td>
<td>Carbon tetrachloride</td>
</tr>
<tr>
<td></td>
<td>Naphtha (Petroleum origin)</td>
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<tr>
<td></td>
<td>Perchlorethylene</td>
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<tr>
<td></td>
<td>Trichlorethylene</td>
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<tr>
<td>Fingernail polish remover</td>
<td>Acetone</td>
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<tr>
<td></td>
<td>Alcohol</td>
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<tr>
<td></td>
<td>Aliphatic acetate</td>
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<td></td>
<td>Benzene</td>
</tr>
<tr>
<td>Lacquer thinner</td>
<td>Aliphatic acetate</td>
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<tr>
<td></td>
<td>Methyl, ethyl, propyl alcohol</td>
</tr>
<tr>
<td></td>
<td>Toluene</td>
</tr>
<tr>
<td>Cleaning fluid-spot removal</td>
<td>Carbon tetrachlorides</td>
</tr>
<tr>
<td></td>
<td>Trichlorethylene</td>
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<tr>
<td></td>
<td>Trichlorethanol</td>
</tr>
<tr>
<td>Household cements</td>
<td>Acetone</td>
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<tr>
<td></td>
<td>Isopropanol</td>
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<tr>
<td></td>
<td>Methyl ethyl ketone</td>
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<td></td>
<td>Methyl isobutyl ketone</td>
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<td></td>
<td>Toluene</td>
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<tr>
<td>Model cements</td>
<td>Acetone</td>
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<tr>
<td></td>
<td>Naphtha (Petroleum origin)</td>
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<td></td>
<td>Toluene</td>
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<tr>
<td>Plastic cements</td>
<td>Acetone</td>
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<tr>
<td></td>
<td>Aliphatic acetate</td>
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<tr>
<td></td>
<td>Cyclohexane</td>
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<td></td>
<td>Hexane</td>
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<td></td>
<td>Toluene</td>
</tr>
</tbody>
</table>

*Non-volatile and non-lethal constituents not listed.

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His occupation was that of a painter in a furniture shop. The deceased was missing from work from midday when he had stopped work for his midday meal and the body was recovered three days later. The clothing and body was splattered with paint. A tin of paint and "thinner" was also present alongside the body.

**Autopsy findings**

The body was that of a male measuring 170 cm in height and 64 kg in weight. There were no external injuries on his body. The postmortem changes were in keeping with the stated number of days since last seen. The internal organs showed autolytic changes confirmed by microscopy which was unhelpful and non-specific. Toxicological analysis confirmed the presence of ether and toluene in blood and brain. This was not quantified. No methane was isolated from his body fluids.

**Case discussion**

The decomposed body of a habituated "sniffer" was recovered from a dry manhole. It is not known whether he was a lone "sniffer" or a "group" sniffer. Kupperstein and Susman state "that glue sniffing is usually an individual act, although some group sniffing has been observed. Many children and adolescents have admitted to sniffing in school in the lavatories, hallways, locker rooms and even in the classroom." "The use of glue outside the school context is very often, though certainly not in all cases, a form of social experience in which the participants share or attempt to identify with certain commonly felt values or feelings of boredom, rejection, social immaturity, insecurity, or fear of failure before one's parents or peers.""

"Into this group fall most of the middle-class youths living in bedroom suburbs of many of the large metropolitan areas whose activities have, on occasion, come to the attention of parents, law enforcement officials, school and social welfare personnel." Methane, a well known hazard to coal miners can also be found in sewers due to the action of putrefying bacteria that consume oxygen and generate methane. If the deceased was a lone "sniffer" and used a manhole for the prime purpose of solvent abuse, his body fluids might contain methane in addition to the solvent or solvents abused. No methane was isolated from the deceased's tissues. Thus it is reasonable to postulate that if he was a "group sniffer", that when death occurred he was deposited or dumped into the sewer along with the "solvent abuse paraphernalia." The manhole lid was considered too heavy for the deceased to lift and the manhole too small for "group sniffing."

If on the other hand, he was a lone "sniffer", it could be that when his body was discovered he was again deposited or dumped down the manhole. It is presumed that during lunch break he used the employer's backyard for his practice. No evidence to this was available. A visit to the scene is unlikely to be helpful as the "solvent abuse paraphernalia" is likely to have been cleared away by those arriving at the scene prior to the investigating authority.

On the other hand, there was no obvious ante-mortem or post-mortem laceration, it being difficult to exclude the occurrence of abrasions as post-mortem shedding of the cuticle had commenced. He should have sustained some post-mortem injuries if he was 'dumped' in the manhole.

Further, due to the advanced state of decomposition, it was not possible to state with any degree of certainty that the body had been moved from the initial spot where death occurred as post-mortem hypostasis was indistinct. There were no ante-mortem injuries to cause or contribute to death.

**REVIEW OF SOLVENT ABUSE**

**Absorption and excretion**

Inhaled vapours of these drugs pass rapidly from the alveoli into the blood and from the blood into the brain. It may enter the body by absorption through the skin, mucous membrane and gastrointestinal tract.

Excretion is the reverse of absorption, although it is somewhat slower because of the high solubility of hydrocarbons in brain tissue. Metabolic breakdown of the more popular hydrocarbons is small in comparison to excretion of the unchanged chemical in expired air. Some agents, such as trichloroethylene, are largely metabolised and the metabolites are excreted in the urine.

**Technique of administration**

The substance to be used is placed on a rag, piece of gauze or cloth which is held over the mouth and/or nose during inhalation. The cloth may be folded and the sniffer will inhale on it. There is usually no direct contact between the hydrocarbon and the nasal or oral mucosa because some of these chemicals are extremely irritant. Alternatively the gauze or cloth is placed in a paper or plastic bag and the opening of the bag is held
tightly over the mouth and/or the nose.

Higher concentrations of the hydrocarbon in the inspired air can be obtained when a bag is used. The maximum allowable concentration of toluene for industrial operations had been set by the American Conference of Government and Industrial Hygienists at 200 parts per million. The concentration of toluene achieved when inhaling directly from a paper bag containing gauze soaked with toluene from a tube of polystyrene cement is about 50 times this allowable concentration.¹

Tests conducted indicate that 3.6 mg of toluene can be recovered from 100 ml of air from such a bag.² A few deep breaths produce an effect which gradually dissipates over 35-45 minutes. The experienced user is able to maintain a "high" for up to 12 hours by periodic sniffs.⁹

Clinical effects

The immediate effect is one of pleasant exhilaration, euphoria and excitement, closely simulating the early effects of alcohol followed by ataxia and slurred speech. Diplopia and tinnitus are frequently experienced.

Acute intoxication is characterised by complaints of headache and feeling of fatigue and confusion. Some patients may act drunk. In addition, there may be nausea and vomiting as well as disturbances of equilibrium and co-ordination. Co-ordination may be sufficiently impaired to be potentially injurious.¹⁰ Unconsciousness have also been observed.

The initial euphoria is pleasant to the user and is the one sought after effect. There is also enjoyment of the drowsiness and a dream-like state in the later stages. Feelings of weakness are dispelled and during the "high," abusers often feel strong and invulnerable. These latter effects may demonstrate their invulnerability by self-mutilation."¹¹

Tolerance, habituation/dependence and "addiction"

In the initial stages of inhalation, prior to habit formation, a few whiffs of the vapours will produce a "jag" but tolerance to the solvents appears to develop as chronic users often have to 'take' the contents of as many as 5 tubes of 'cement' in order to experience the desired results. Tolerance tends to occur as early as 3 months with weekly usage.

Habituation also occurs. It was the impression of Press and Done² that the habituation or dependence pertained not to the specific chemical itself, but rather to the state of intoxication. Whether physical dependence and objective withdrawal signs develop is less clear and questionable. It has been claimed that some develop tremors, become irritable, anxious and have difficulty in falling asleep after withdrawal.¹² Baker¹² reported a lack of withdrawal symptoms except for possible lethargy and depression.

Pathology

The volatile organic solvents contained in glues used by "sniffers" can be toxic to organ systems such as the heart, liver, kidney, brain and bone marrow.

Cardiac deaths in solvent abuse include fatal arrhythmias, believed to be due directly to the solvent affecting the myocardium, and myocardial infarction and dilated cardiomyopathy.¹³ Baerg and Kimberg⁶ have noted the occurrence of centrilobular hepatic necrosis and acute renal failure in "solvent sniffers." Luric also comments on hepatic and renal damage following acute toluene poisoning.¹⁴ No histological confirmation was possible in our case as the tissues were autolysed.

Baker and Tichy¹⁵ studied the effects of toluene on the nervous system in acute and chronic exposure. Under acute conditions, scattered abnormalities of neurones were seen in brain and spinal cord. An experient with chronic exposure revealed more definitive nervous abnormalities consisting of increased pigmentation and neuronal shrinkage. Patchy loss of myelin, particularly in perivascular areas, was also seen. The cerebellar folia were considered to be abnormal with a decrease in the number of Purkinje cells as well as degenerative changes.

The effects on the EEG, of inhalation of vapourized lacquer thinner – a mixture of butylacetate, toluene and ethyl alcohol – was studied on rabbits, normal human subjects and epileptics. Slight slowing of the normal waves was recorded from the brains of rabbits only on exposure to doses approaching lethal levels and was always preceded by other signs of intoxication. No EEG changes were noted in the human subjects.¹⁶

There is little evidence in the literature that a single acute exposure of toluene intoxication alters the blood picture in any way, and even a small exposure over a short period produces nothing more alarming than a relative lymphocytosis.¹⁴ Bone marrow depression⁹ and hepatic enlargement have been reported.¹⁷ Wilson, in 1948 reported bone marrow depression
Medico-legal aspects

Sudden death is a recognised hazard of volatile substance abuse. In addition, some of these deaths may be the result of some secondary event such as trauma. Deaths may also occur months or years after exposure and still be related to the magnitude and duration of the exposure.

Specific post-mortem features have not as yet been identified either macroscopically or microscopically in volatile substance abuse deaths. Toxicological analysis for volatile substances is hence necessary for the identification of the cause of death. Four pathophysiological mechanisms for acute volatile solvent abuse deaths have been postulated: anoxia, vagal inhibition, respiratory depression and cardiac arrhythmia. Of these, cardiac arrhythmia due to "sensitization" of the heart to adrenaline is probably the most common. The triad of hypoxia, a volatile substance and adrenaline was found to be more toxic than the combination of volatile substance and adrenaline. There is a case of successful resuscitation from ventricular fibrillation after toluene inhalation in a 16-year-old boy.

A particularly dangerous practice is the use of a plastic bag in the administration of the agent. In the first place, rebreathing air contained in a bag will sharply decreased pO2 and increased pCO2 of arterial blood and aggravate any tendency towards cardiac arrhythmia caused by hydrocarbons, thus inducing anoxia in a sensitized myocardium. Further, the "sniffer" may become unconscious while sniffing and the plastic bag can cause suffocation.

Conclusion

When a victim of solvent abuse is discovered at an unusual site the possibility of the body being moved should always be considered. When such a body is recovered from a sewer or disused mine the toxicologist should be requested to identify methane in addition to the identification of agents used for "sniffing." Sudden death results from solvent abuse both in the inexperienced novice and the habituated abuser. Delayed deaths have also occurred from liver and renal failure.

Those physical effects experienced by the abuser which simulates the effects of alcohol may result in a traumatic death e.g. motor vehicle accident. This makes it mandatory for the pathologist to forward samples from victims of vehicle accidents particularly pedestrians, not only for the determination of alcohol levels but also for the presence and estimation of volatile substance of abuse.

The exhibits that should be forwarded in suspected cases of solvent abuse are subcutaneous fat and brain. These should be forwarded as quickly as possible in glass containers with metal screw top for analysis.

ACKNOWLEDGEMENTS

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REFERENCES


