

Case Reports on Acute Toluene Poisoning During Parquet Flooring

W P Tan,*^{MBBS}, E Seow,**^{FAMS, MBBS, FRCS (Edin)}

Abstract

We report two cases of acute toluene poisoning occurring during parquet flooring. Inhalation is the major route of absorption in the workplace, owing to the high volatility of the compound. Such patients should be monitored closely for respiratory distress, central nervous system (CNS) depression and cardiac arrhythmias. Both our patients manifested signs of CNS depression and one had bronchospasm aborted by salbutamol. Industries using toluene must ensure that precautions like adequate ventilation are taken to protect their workers.

Ann Acad Med Singapore 1997; 26:138-40

Key words: Glue sniffing, Inhalation, Occupational exposure, Solvent

Introduction

Toluene was thrown into the limelight in the mid-1980s when solvent abuse, known locally as “glue-sniffing” reached an epidemic proportion before Government legislation brought it under control. However, exposure to solvents like toluene still occurs in the occupational environment. Some of the main workplaces or work activities using solvents include automotive with spray painting, woodwork/furnishing, metal trades, printing, plastics/petrochemicals, dry cleaning, laboratories, building and agriculture.

We present two cases of acute toluene poisoning as a result of occupational exposure.

Case Reports

On 31 August 1995, two brothers, Patient A and Patient B were brought to the Accident & Emergency Department (A&E) by a Singapore Civil Defence Force (SCDF) ambulance team. They had been found dazed and unresponsive on the floor of an enclosed room where they had been laying parquet for two hours. The room was reported by the ambulance officer to have been enveloped in a strong smell of “thinner”. On examination, both patients had “cakes of glue” on their bodies and smelled strongly of thinner.

Considering the circumstances in which the patients had been found, the possibility of solvent abuse was ruled out.

Case 1

In the Emergency Department, Patient A, aged 23, was only able to respond to pain stimulus. The initial

blood pressure was 105/56 mmHg, pulse rate 100/minute, respiratory rate 23/minute and temperature 37.3°C. Both the conjunctivae were injected and both the pupils were 3 mm in diameter, equal and reactive. Neurological examination did not reveal any localising signs. Examination of the heart, lungs and abdomen was normal. The oxygen saturation was between 93% and 97%. The electrocardiogram and chest X-ray were normal, and the potassium level was 4.0 mmol/L. Arterial blood gas on high flow oxygen was pH 7.35, pCO₂ 44.6 mmHg, pO₂ 219.5 mmHg, base excess -1.7, standard bicarbonate (SBC) 23.8 mmol/L, and oxygen saturation 99.7%.

The patient was admitted to the Medical Intensive Care Unit. He had four episodes of vomiting. He needed one dose of nebulized salbutamol during his stay there. A repeat chest X-ray was normal. The cardiac enzymes and liver function tests were normal. Blood toxicology detected a toluene level of 16.6 ug/ml and a small, insignificant amount of xylene.

He was mentally alert 24 hours after admission.

Case 2

In the Emergency Department, Patient B, aged 21, was found to be conscious but disorientated. The initial blood pressure was 114/61 mmHg, pulse rate 85/minute, respiratory rate 24/minute and temperature 37.3°C. Both the pupils were 3 mm in diameter, equal and reactive. There were no abnormalities detected on physical examination. The electrocardiogram and chest X-ray were normal, and the potassium level was 3.9 mmol/L. Arterial blood gas on high flow oxygen was

* Medical Officer

** Consultant

Department of Emergency Medicine
Tan Tock Seng Hospital

Address for Reprints: Dr Eilayne Seow, Department of Emergency Medicine, Tan Tock Seng Hospital, Moulmein Road, Singapore 308433.

pH 7.34, pCO₂ 37.9 mmHg, pO₂ 224.3 mmHg, base excess 4.9, SBC 19.9 mmol/L, and oxygen saturation 99.7%.

He was admitted to the High Dependency Unit. He had one episode of vomiting during his admission. He was mentally alert on the night of admission. On questioning, he denied having auditory or visual hallucination before he "passed out" at work. A repeat chest X-ray was normal. The cardiac enzymes and liver function tests were normal. Blood toxicology detected a toluene level of 16 ug/ml.

On the second day of admission, both patients signed their own discharge citing financial difficulties as the reason. No follow-up on the patients was possible because they had decided to return to their home in Malaysia upon discharge.

Discussion

Toluene, together with xylene and benzene, belong to a class of compounds known as aromatic hydrocarbons. It has a high affinity for lipid-rich neural tissues. As such, higher concentration of toluene has been found in the brain of forensic specimens, compared to other organs like the kidney and liver.¹ Experiments in animals after toluene inhalation have shown toluene concentration in fatty tissues 80 times more than in blood.² The central nervous system (CNS) effects of toluene—easily confused with those caused by alcoholic intoxication—consist of an initial excitation stage of euphoria, exhilaration and giddiness, followed by CNS depression characterised by headache, dizziness, fatigue, drowsiness, confusion, vertigo and decreased reaction time.

Due to the high lipid-dissolving property of toluene, the time interval between exposure to toluene and sampling is important when interpreting results. Blood concentration of toluene may be biphasic; an initial peak is followed by a trough reflecting lipid binding by the CNS, and a subsequent peak may appear as toluene is slowly released into the blood.³ Blood test for toluene was done for our patients shortly after they reached the A&E Department. They manifested CNS signs and symptoms at levels of 16.6 and 16.0 ug/ml respectively. In a study by Miyazaki et al⁴ on the correlation between "on admission" blood toluene concentrations and presence or absence of signs and symptoms in solvent abusers, the toluene concentrations in the blood ranged from 0.3 to 22.8 ug/g. In another local study on glue-sniffing deaths, blood toluene levels were found to be between 0.2 to 92 ug/ml. Among the 33 deaths, only two, with blood toluene levels of 92 and 49 ug/ml, were directly attributed to acute toluene toxicity. The rest were caused by trauma.⁵ Traumatic deaths as a result of depression of the CNS, rather than from the direct toxic

effects of toluene, have been reported by other workers.¹

Acute inhalation of toluene also exerts direct adverse effects on the lungs, resulting in bronchial and laryngeal irritation, acute bronchitis, bronchospasm, pulmonary oedema, chemical aspiration pneumonitis and asphyxia from narcosis. Both our patients did not have obvious signs of the above complications and their arterial blood gas taken at the A&E Department were normal. However, in the Intensive Care Unit, Patient A probably developed an episode of bronchospasm, aborted by one dose of nebulised salbutamol.

Other systems affected by acute toluene inhalation include the cardiovascular system, gastrointestinal tract and the acid-base and fluid-electrolyte balance of the body. Cardiovascular system effects include arrhythmias, myocardial infarction and myocarditis. Toluene inhalation also causes nausea, vomiting and abdominal pain. Metabolic acidosis occurs as a result of tissue hypoxia and the accumulation of the metabolites of toluene, namely benzoic acid and hippuric acid. Hypokalaemia, hypophosphataemia and, less frequently, hypercalcaemia and hypouricaemia are also found in toluene poisoning.

Therefore, patients suffering from acute inhalation should be monitored closely for respiratory distress and CNS depression. As there is a possibility of a lowered myocardial threshold to the arrhythmogenic effects of adrenaline and other catecholamines, they should be used with caution.

In Singapore, the Department of Industrial Health, Ministry of Labour, has confirmed 4 cases of toluene poisoning since 1983 (one in 1983 and three in 1991). Whereas cases of industrial toluene poisoning have been reported in rust-preventive painting inside a bridge pier, car and industrial spray painting, and printing,^{1,6,7} there have been no cases reported occurring during the process of parquet flooring. Owing to the nature of the work and chemicals involved, workers laying parquet floors are evidently at risk of acute toluene poisoning, especially if their work environment is poorly ventilated and no protective masks and gloves are used.

Inhalation is the major route of absorption in the workplace, owing to the high volatility of the compound. Measurement of toluene concentration in mixed exhaled air of volunteers indicates that, at the apparent steady state, pulmonary retention is about 50%.⁸ After inhalation, the blood concentration of toluene increases with increased environmental contamination. Environmental toluene concentration is measured in parts per million (ppm) which means parts of toluene per million parts of air.

The American Conference of Governmental Industrial Hygienist (ACGIH) recognises the potential toxic

effects of toluene and has developed three categories of threshold limit values (TLVs) for toluene.⁹ TLV is defined as the airborne concentration of a substance that represent conditions under which it is believed that nearly all workers may be exposed day after day without adverse effects.

The three categories are:

- (1) time-weighted average (TWA), a value for a normal 8-hour workday and 40 hour workweek;
- (2) short-term exposure limit (STEL), a value for a short period of time, usually 15 minutes;
- (3) ceiling (TLV-C), a value which should not be exceeded even briefly.

Each year, the ACGIH meets to consider new information and revises TLVs when warranted by new data. The presently recommended TLV-TWA of 100 ppm (377 mg/m³) with a STEL of 150 ppm (565 mg/m³) is based on preventing acute depression of the central nervous system. A proposed reduction of TLV-TWA to 50 ppm (147mg/m³) with a skin notation but no STEL, was placed on the Notice of Intended Change in 1991.¹⁰

In our cases, we were unable to obtain the size of the room the workers had been working in or how much toluene had already been applied onto the floor. Thus, we were unable to calculate the concentration of toluene in the environment. In any case, since toluene vapour is heavier than air, laying parquet at floor level would have exposed our patients to a higher concentration of toluene than the general room concentration.

Besides TLV, the ACGIH has also recommended biological exposure indices (BEI) for toluene in venous blood, toluene in end-exhaled air and hippuric acid in urine, sampled at the end of a shift.¹⁰ Measurements of toluene in venous blood and in end-exhaled air are specific indicators of toluene exposure, whereas hippuric acid in urine is a non-specific indicator because it is also a metabolite of ethyl benzene, benzoic acid and possibly styrene. However, the timing of sample collection for measurement of toluene in venous blood and end-exhaled air is so critical that these tests are unsuitable for evaluation of the degree of exposure. They are, nevertheless, valuable as qualitative confirmatory tests whenever the origin of hippuric acid is in question. As a result of occupational exposure at 100 ppm of toluene, blood concentrations at the end of the shift are expected to be about 1 ug/ml and 16 hours later (prior to the next shift)

about 0.05 ug/ml; concentrations in end-exhaled air collected at the end of the shift should be below 40 ppm and 16 hours later about 1 ppm. The value of 2.5 g of hippuric acid in urine per gram of creatinine is also recommended as a BEI. Without taking into account the timing of sample collection, the venous blood concentrations of toluene in our cases have exceeded the recommended BEI.

Conclusion

We recommend that workers who are exposed to toluene in their work, be made aware of the signs and symptoms of toluene poisoning. Industries should ensure that precautions are taken to provide adequate ventilation when activities such as parquet flooring are carried out.

Acknowledgement

We would like to thank Dr Gan Siok Lin and the staff of the Department of Industrial Health, Ministry of Labour for their help in providing statistics and information on occupational toluene poisoning.

REFERENCES

1. Takeichi S, Yamada T, Shikata I. Acute toluene poisoning during painting. *Forensic Sci Int* 1986; 32:109-15.
2. Carlsson A, Lindqvist T. Exposure of animals and man to toluene. *Scand J Work Environ Health* 1977; 3:135-43.
3. King M D, Day R E, Oliver J S, Lush M, Watson J M. Solvent encephalopathy. *BMJ* 1981; 283:663-5.
4. Miyazaki T, Kojima T, Yashiki M, Chikasue F, Tsukue I. Correlation between 'on admission' blood toluene concentrations and the presence or absence of signs and symptoms in solvent abusers. *Forensic Sci Int* 1989; 44:169-77.
5. Chao T C, Lo D S T, Koh J, Ting T C, Quek L M, Koh T H, et al. Glue-sniffing deaths in Singapore-volatile aromatic hydrocarbons in postmortem blood by headspace gas chromatography. *Med Sci Law* 1993; 33(3):253-60.
6. Elofsson S A, Gamberale F, Hindmarsh T, Iregren A, Isaksson A, Johnsson I, et al. Exposure to organic solvents: a cross-sectional epidemiologic investigation on occupationally exposed car and industrial spray painters with special reference to the nervous system. *Scand J Work Environ Health* 1976; 2:240-55.
7. Chia S E, Tan K T, Kwok S K. A study on the health hazard of toluene in the polythene printing industry in Singapore. *Ann Acad Med Singapore* 1987; 16:294-9.
8. Cohr K H, Stockholm J. Toluene, a toxicological review. *Scand J Work Environ Health* 1979; 5:71-90.
9. Amdur M O, Doull J, Klaassen C D, editors. *Casarett and Doull's Toxicology: The basic science of poisons*. 4th ed. New York: Pergamon Press, 1991.
10. Documentation of TLV's and BEI's. Volume III. 6th ed. Cincinnati, Ohio: American Conference of Governmental Industrial Hygienist, Inc, 1991.