Clinical Toxicology

Fatal fulminant hepatic failure in a 'solvent abuser'

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Summary: The case of a 17 year old abuser of butane aerosols who developed fulminant hepatic failure after taking a proprietary engine or carburettor cleaner is described. Fatalities as a result of liver failure due to volatile hydrocarbons or solvents have not previously been reported. The likely toxins included isopropyl alcohol, methyl amyl alcohol, butylated hydroxytoluene as well as petroleum products, and evidence for their toxicity is reviewed. The possibility of increased susceptibility to hepatotoxins after enzyme induction by abuse of volatile agents is discussed. Hepatic dysfunction should be sought in patients who abuse solvents or volatile hydrocarbons and supportive measures instituted promptly when required.

Introduction

The recognized causes of death from solvent abuse include anoxia, vagal inhibition, respiratory depression, cardiac arrhythmias, trauma and suicide.¹ The number of deaths attributable to the inhalation of fuel gases and pressurized aerosols is increasing in the United Kingdom and over the last few years has nearly doubled in males aged 10–19 years.² We report a case of solvent abuse resulting in fatal fulminant hepatic failure.

Case report

A 17 year old male Caucasian was admitted to a surgical ward with a 10 h history of abdominal pain and vomiting. He was flushed but afebrile and had a fetor with a mildly tender abdomen especially on the right. Full blood count was normal. The pain settled after 24 h observation. A barium meal to investigate the vomiting revealed situs inversus partialis, but was otherwise normal.

During the next day he became progressively more agitated and showed bizarre behaviour. He developed hepatomegaly, polyuria and jaundice. Inquiries revealed that the patient had been abusing butane aerosols (5 to 10 cans per day) for 3 years. The patient admitted taking a proprietary engine or carburettor cleaner (Table I) 2 days prior to admission and denied drug overdose, alcohol or intravenous drug abuse. No contacts had had jaundice and he had never had a blood transfusion.

Investigations revealed a hepatocellular jaundice (alanine transferase 12,850 U/l, alkaline phosphatase 8,851 U/l, bilirubin 117 mmol/l, prothrombin time 46/13 s), with renal impairment (urea 13 mmol/l, creatinine 336 mmol/l).

Hepatitis A IgM and IgG and hepatitis B surface antigen were negative. Urine testing was negative for paracetamol and aspirin. He was treated with lactulose, magnesium sulphate enemas, vitamin K and B vitamins. Blood sugars and acid–base status remained stable.

Over the subsequent 24 h his encephalopathy progressed rapidly. This did not respond to mannitol infusion. He became anuric and developed hyponatraemia (116 mmol/l). Following 3 grand mal fits he became unresponsive to pain. Computed tomographic scan showed severe cerebral oedema with evidence of coning. Post mortem confirmed the findings of cerebral oedema with cerebellar and brain stem coning. The liver showed extensive necrosis of acinar zones 2 and 3.

Discussion

Death due to fulminant hepatic failure following solvent abuse has not to our knowledge previously been reported. The clinical course and classical post mortem findings were typical of toxin-induced fulminant hepatic failure.³ Solvents contain a variety of substances which might be toxic. Known hepatotoxins such as carbon tetrachloride³ are also frequently nephrotoxic.⁴ Chronic low-dose industrial exposure to related chlorinated hydrocarbons and benzol derivatives causes mild abnormalities of serum aminotransferases and

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Accepted: 18 July 1991

Table I Contents of the carburettor and engine cleaner

<i>Carburettor cleaner/conditioner</i>	
isopropyl alcohol	40–50%
mineral oil	40–50%
calcium petroleum sulphanate	not stated
butylated hydroxytoluene	not stated
<i>Engine cleaner</i>	
aromatic petroleum distillates	40–50%
water	20–30%
butyl cellulose	< 10%
methyl amyl alcohol	
petroleum oil	
oleic acid	
calcium petroleum sulphonate	1–5%
aqueous ammonia	1–5%

occasionally a mild reactive hepatitis on biopsy.⁵ Toluene has been implicated in hepatorenal damage in one ‘glue sniffer’ who required dialysis, though hepatic damage was not severe. Hepatic and renal function returned to near normal values within a few days.⁶

Several of the compounds in the engine or carburettor cleaner might have been involved in the toxic reaction including alcohols and toluene derivatives. Isopropyl alcohol appears to be more toxic than ethanol and toxicity has been reported with ingestion of as little as 20 ml.⁷ It undergoes hepatic metabolism to acetone. As with many industrial compounds⁸ there is little information (Medline & Toxline literature searches) on the excretion and toxicology of the other compounds

in the cleaners, though oleic acid is unlikely to be the cause. Encephalopathy but not hepatotoxicity has been reported with petrol inhalation.^{9,10}

Enzyme induction is known to predispose to the toxic effect of carbon tetrachloride on the liver,³ and alcohol probably enhances renal damage due to chlorinated hydrocarbons.⁴ Our patient may have had microsomal enzyme induction as a result of longstanding and heavy abuse of butane and this may have resulted in the rapid and severe nature of the liver damage. A total of 30–45% of an inhaled dose of butane is absorbed and though most is excreted unchanged via the lungs a small amount is probably metabolized in the liver.¹¹

The predominance of abdominal pain in this patient’s symptomology resulted in admission to a surgical ward for observation. Abdominal pain may be a feature of hepatitis² and was a marked feature of ‘Epping jaundice’, a hepatotoxic reaction to an aromatic amine dissolved in butyrolactone.¹² Clinicians should remain alert to the possibility of toxin or drug ingestion as a cause for non-specific symptoms including pain and vomiting. This case demonstrates that hepatic dysfunction with solvent abuse can be severe or life threatening and may present in rather unusual ways. Clinical suspicion and early investigation (e.g. prothrombin time) should permit early recognition and the prompt instigation of supportive measures.

Acknowledgements

We thank those in the Drug Information Centre, City Hospital, Nottingham for their help.

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