Short report

Persistent visual hallucinations secondary to chronic solvent encephalopathy: case report and review of the literature

KS CHANNER, S STANLEY

From the Department of Neurology, Bristol Royal Infirmary and Department of Family Psychiatry, Bristol Children’s Hospital, Bristol, UK

SUMMARY A case of solvent encephalopathy presenting with persistent visual hallucinations, and characterised by a diffusely abnormal EEG and delayed visually evoked responses to checkerboard pattern reversal (VERs), is described. Abnormalities in the EEG and VERs showed minimal improvement over many months of abstinence from glue sniffing. The literature on the neurological sequelae of solvent abuse is reviewed.

Volatile complex hydrocarbons, in particular toluene, are the latest drugs to be abused for their euphoric effects. The commonest source of these chemicals are adhesives, although lacquer thinner, spot remover, dry cleaning substances, lighter fuel, aerosols and even marker pens have their advocates.1-7 The vapours are inhaled, entering the blood stream via the respiratory tract. The euphoric effects have been likened to those of alcohol and narcotics, although they are short lived and visual hallucinations feature predominantly.2 Physical dependence is thought not to occur2,4,8 but psychological dependence undoubtedly does. The first reports of this form of drug abuse appeared in the early 1960s and involved men who had first experienced the pleasurable effects by accident during industrial exposure.10-12 They had then purposefully abused the drug. Today’s sniffers or “huffers” are usually adolescent males who frequently initially experience the drugs in group sessions,5-6,11 although as many as 25% can be female.14 We report a case of persistent visual hallucinations from glue sniffing.

Case report

In January 1982 a 16-year-old boy was referred with a history of persistent visual hallucinations. He had first experienced bright coloured lights and zig-zag shapes in his visual fields when sniffing glue (usually puncture repair cement and Evostik) in July 1981. Initially the hallucinations had lasted for a few hours during the “high” but gradually, after each intoxication they persisted until they were constantly present. He had stopped partaking of the glue in September 1981, and claimed to have only sniffed glue for a total of three months.

At presentation the hallucinations consisted of a pattern of coloured shapes and lights which he likened to a film in front of his eyes. He was more aware of this phenomenon when the external lighting was low. There was no other significant history. General examination was normal and there were no focal neurological signs. Visual acuity (recorded at N4-5 both eyes) was normal, and colour vision and fundoscopy were normal. Visual evoked responses to checkerboard pattern reversal were delayed by 2-5 SD (standard deviations) on the right and 3 SD on the left. Electroretinograms and electro-oculograms were within normal limits. Liver function tests, serum electrolytes, urea and blood glucose were normal as was a CT brain scan. Cerebrospinal fluid (CSF) examination disclosed one lymphocyte/cmm, no red blood cells and no bacterial growth. CSF total protein was 0.52 g/l (normal up to 0.55 g/l) and CSF IgG was 0.04 g/l (normal up to 0.06 g/l). Repeated electroencephalograms over a three month period disclosed widespread generalised slow wave activity (3-4 Hz, 30-50 microvolts). Intermixed with this activity were theta waves of 4-7 Hz, 25-40 microvolts. Irregular alpha waves (8-10 Hz, 25 microvolts) were recorded posteriorly responding to eye opening.

At follow up four months after initial presentation and having not sniffed glue for eight months, the hallucinations remained, although they now consisted of a constant
looping pattern. The EEG was unchanged and the VERs were delayed 2 SD from normal.

Discussion

The practice of glue sniffing has become popular amongst adolescents over the last twenty years, and was initially thought to be harmless. Now the physical consequences of solvent abuse are emerging. Many case reports have appeared citing various toxic effects and it seems that these are dependent on the chemical abused. Proprietary cements and glues are mixtures of organic compounds and so an individual chemical cannot always be blamed. The table lists the documented acute reversible effects of intoxication with specific chemicals; the commonest abused is toluene. Neurological complications are most commonly reported from toluene intoxication and this is explained by the pharmacokinetics of the drug. The volatile hydrocarbon is lipid soluble, rapidly enters the blood stream from the lungs and saturates the high lipid content of the central nervous system which forms a reservoir of the chemical. After inhalation has ceased the hydrocarbon slowly returns to the blood from the lipid reservoir. Analysis of blood and brain samples always shows a higher concentration in the brain—often many times higher.

One of the effects of solvent intoxication is acute and subacute encephalopathy. King et al published a series of nineteen cases of acute encephalopathy in children—five presenting in coma, five with ataxia and dysarthria, three with convulsions and two with diplopia and behaviour disturbance. In five of the nineteen patients the diagnosis was not made historically but from toluene blood assay. Six of these children left hospital with neurological impairment and one patient seen one year later had persistent cerebellar signs. Chronic neurological damage from solvent abuse has been sporadically reported in patients who have abused solvents from one and a half to fourteen years and takes the form of dementia with cerebellar ataxia. Pathologically, in the one post-mortem analysis published, the most striking feature was diffuse demyelination with reactive gliosis. There was a 70% loss of cerebellar Purkinje cells and giant axonal degeneration in the posterior and lateral columns of the spinal cord.

Persistent peripheral neuropathy occurs with N-hexane, methyl-ethyl ketone and methyl-buty1 ketone intoxication but not with toluene. Characteristically the neuropathy is predominantly motor and progresses slowly over a few months. It usually continues to progress despite abstinence but gradually recovers, although not always fully, in months. Pathologically large myelinated nerve fibres are most affected with axonal swelling by neurofilamentous masses, degeneration and secondary demyelination.

Damage to the visual pathway has been reported in a Mexican who for three years habitually inhaled a metallic copper aerosol spray paint containing

<table>
<thead>
<tr>
<th>Chemical</th>
<th>Systemic effect</th>
<th>No of cases</th>
<th>Authors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Toluene</td>
<td>Hepatic damage</td>
<td>1</td>
<td>O'Brian et al 1971</td>
</tr>
<tr>
<td></td>
<td>Renal damage</td>
<td>1</td>
<td>Will, McLaren 1981</td>
</tr>
<tr>
<td></td>
<td>Distal renal tubular acidosis</td>
<td>1</td>
<td>Moss et al 1980</td>
</tr>
<tr>
<td></td>
<td>Recurrent urinary calculi</td>
<td>2</td>
<td>Bennett 1980</td>
</tr>
<tr>
<td></td>
<td>Convulsions</td>
<td>1</td>
<td>Taher 1974</td>
</tr>
<tr>
<td></td>
<td>Encephalopathy</td>
<td>19</td>
<td>Kroeger et al 1980</td>
</tr>
<tr>
<td></td>
<td>Optic neuropathy</td>
<td>1</td>
<td>Allister et al 1981</td>
</tr>
<tr>
<td></td>
<td>Aplastic anaemia</td>
<td>3</td>
<td>Helliwell (letter) 1979</td>
</tr>
<tr>
<td></td>
<td>Aplastic crises in sickle cell anaemia</td>
<td>1</td>
<td>King et al 1981</td>
</tr>
<tr>
<td>Chlorinated hydrocarbons</td>
<td>Hepatic crises</td>
<td>3</td>
<td>Keane 1978</td>
</tr>
<tr>
<td></td>
<td>Renal damage</td>
<td>5</td>
<td>Powars 1965</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2</td>
<td>Wilson 1943 (industrial exposure)</td>
</tr>
<tr>
<td>N-hexane</td>
<td>Peripheral neuropathy</td>
<td>4</td>
<td>Baerg, Kinberg 1970</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3</td>
<td>Litt, Cohen 1969</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2</td>
<td>Knox, Nelson 1966</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2</td>
<td>Baerg, Kinberg 1970</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2</td>
<td>Litt, Cohen 1969</td>
</tr>
<tr>
<td>Methyl-ethyl ketone</td>
<td>Retrobulbar neuritis</td>
<td>1</td>
<td>Goto et al 1974</td>
</tr>
<tr>
<td></td>
<td>Peripheral neuropathy</td>
<td>18</td>
<td>Herskowitz et al 1971</td>
</tr>
<tr>
<td></td>
<td></td>
<td>7</td>
<td>Towsfghi et al 1976</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2</td>
<td>Prokop et al 1974</td>
</tr>
<tr>
<td></td>
<td></td>
<td>18</td>
<td>Altenkirch et al 1977</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2</td>
<td>Shirabe et al 1974</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>Korobkin et al 1975</td>
</tr>
<tr>
<td></td>
<td></td>
<td>18</td>
<td>Berg 1971</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Altenkirch et al 1977</td>
</tr>
</tbody>
</table>
Persistent visual hallucinations secondary to chronic solvent encephalopathy

85
toluene.25 He presented with deteriorating vision, cerebellar ataxia and nystagmus. The visually evoked responses to checkerboard were abnormal and remained so despite clinical improvement with abstinence. Other abusers have reported visual impairment as part of a generalised chronic encephalopathy.38 The case reported here is the first recorded of persistent visual hallucinations symptomatic of a solvent encephalopathy. It is unusual also in that the encephalopathy has occurred with a short exposure (3 months).

Besides chronic neurological damage, suicidal and accidental death during intoxication from, for example, drowning,24 and suffocation with plastic bags used to raise the partial pressure of vapour in the inhaled air, have been reported.43 A large series of 110 sudden, presumed cardiac deaths in the USA were attributed to the inhalation of aerosol solvents and propellants.43-45 A recent paper quoted research identifying 117 deaths associated with abuse of volatile substances in the UK from 1970–81.46

We thank Dr MJ Campbell for permission to publish this case and Mrs M Miles and Miss E Trapp for typing the manuscript.

References