community with strong ethnic organisations. They are therefore likely to remain susceptible and will be protected only by a policy aimed at national elimination or control of the disease.

The study in Leeds, Luton, and Manchester laboratories was funded by the Office of the Chief Scientist. We thank Miss Pauline Waight, Mr Peter Rushton, Dr Sheila Sheppard, and Mr Gerald Greene for help with data analysis.

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(Accepted 23 January 1987)

SHORT REPORTS

"Glue sniffer's" heart?

Heart failure is rare in British teenagers; solvent abuse is not.[1] We report on a 15 year old boy with a two year history of intermittent solvent abuse who presented with dilated cardiomyopathy.

Case report

A previously fit 15 year old white boy developed non-specific chest pains and shortness of breath. One week later he presented to his local hospital with continuing dyspnoea on minimal exertion. He had sniffed glue intermittently for two years: for the initial 12 months he had done so on a regular basis, then he had stopped for six months but subsequently restarted. Generally he used Bostick, in which the principal solvent is toluene. He also smoked both cannabis and tobacco. Two weeks before his presenting illness began he had had what he described as a "heavy session" of glue sniffing.

On admission he was unwell. His blood pressure was 120/80 mm Hg with a resting heart rate of 110 beats/minute. The jugular venous pressure was raised, but there was no ankle oedema. He had clinical biventricular dilatation and a pansystolic murmur, with a loud pulmonary second sound and a diastolic gallop. Haematological findings, urea and electrolyte concentrations, results of liver function tests, and cardiac enzyme activities were all within normal ranges throughout his illness. He responded to treatment with diuretics and nitrates and was transferred to this hospital.

A chest *x* ray film showed a slightly enlarged heart (cardiothoracic ratio 14/26), left atrial enlargement, blood diversion in the upper lobe, and Kerley B lines. Two dimensional echocardiography showed all four chambers to be dilated and hypokinetic. The left ventricular ejection fraction measured by first pass radionuclide angiography was 17% (normal range >50%). He underwent cardiac catheterisation primarily to exclude acute myocarditis. Myocardial biopsy showed chronic myocarditis, with interstitial fibrosis but no evidence of acute inflammatory responses. Findings on catheterisation were: pulmonary artery pressure 50/20 mm Hg (normal range 15-30/5-16 mm Hg), pulmonary artery capillary wedge pressure 24 mm Hg (6-15 mm Hg), left ventricular end diastolic pressure 32 mm Hg (4-12 mm Hg), and pulmonary vascular resistance 1·3 Wood units (1·0-1·8 Wood units).

He was allowed home taking digoxin, diuretics, nitrates, salbutamol, and enalapril and remained well for two months, when, after a hot bath, he sustained sudden loss of vision followed immediately by a transient loss of consciousness. His blood pressure was 90/65 mm Hg supine and 85/65 mm Hg standing, but after 10 deep knee bends it dropped to 50 mm Hg systolic and he again suffered loss of vision. His vision was restored when he lay down. He denied any recent solvent abuse. His diuretics were withdrawn, but this did not improve his clinical state; an intravenous infusion of dobutamine was required to maintain his blood pressure. Despite this his condition deteriorated and he became breathless even at rest. Within 10 days he underwent cardiac transplantation, and 18 months later he was leading a normal life.

The histological appearance of the excised heart was indistinguishable from that of healing myocarditis, although some patchy eosinophilic infiltration made it difficult to exclude a toxic drug reaction. None of the pathological features showed any difference from those of other dilated cardiomyopathies or healed myocarditides seen in the transplant programme.

Comment

The commonest cause of death in glue sniffers is asphyxiation,[2] but malignant cardiac arrhythmias have been reported[3] and are believed to be due to the "physical toxicity" of the dissolved small solvent molecules in the blood.[4] Acute myocarditis with very high cardiac enzyme activities was reported in a patient who died of cardiac failure after chronic solvent abuse, but there was no association with an acute episode of solvent abuse.[5] In our patient there was a clear history of chronic solvent abuse, with an acute episode of severe abuse two weeks before admission.

We are grateful to Dr P G I Stovin, consultant pathologist, Papworth Hospital, Cambridge, for his advice on the histological features of the excised heart.

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(Accepted 16 February 1987)

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Myocardial infarction and primary ventricular fibrillation after glue sniffing

On average 80 deaths each year are associated with solvent abuse; about 27% of these are related to glue sniffing.[1] Most deaths are thought to be caused by ventricular arrhythmias, but this has been difficult to confirm owing to the sudden onset of symptoms outside hospital. Documentary evidence of ventricular fibrillation as the primary arrhythmia is rare. We report a case of anterior infarction and primary ventricular fibrillation after prolonged inhalation of an adhesive containing toluene.

Case report

A previously healthy 16 year old boy, who was a strong swimmer, was seen to fall face down into the local swimming pool while wading in water 1 m deep. He was immediately taken to the edge of the pool, apparently without inhaling water. At first he was lucid and talking, but five minutes later he suddenly became unconscious. Cardiopulmonary resuscitation was started and a mobile coronary care unit called; on its arrival five minutes later the rhythm was that of ventricular fibrillation. After one direct current shock asystole occurred. Intravenous noradrenaline and calcium chloride resulted in ventricular fibrillation starting again. A further eight direct current shocks were required to restore sinus rhythm. Boluses of lignocaine 200 mg, mexiletine 200 mg, and amiodarone 200 mg plus an infusion of amiodarone 1500 mg over 24 hours were required to control the rhythm. He was intubated and transferred to intensive care.

On arrival he was unconscious and not breathing spontaneously. Astrup pH was 7·27, oxygen tension 14·0 kPa, carbon dioxide tension 6·7 kPa, base