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.

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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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Myocardial infarction and primary ventricular fibrillation after glue sniffing

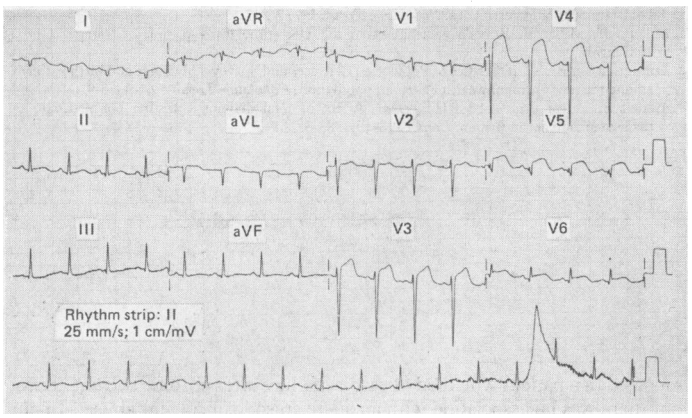
On average 80 deaths each year are associated with solvent abuse; about 27% of these are related to glue sniffing.[1] Most deaths are thought to be caused by ventricular arrhythmias, but this has been difficult to confirm owing to the sudden onset of symptoms outside hospital. Documentary evidence of 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

excess −8·9 mmol/l, sodium concentration 133 mmol/l, potassium 5·0 mmol/l, and urea 7·5 mmol/l. Serum osmolarity was normal. A chest x ray film showed no abnormality. An electrocardiogram showed acute anterior myocardial infarction (figure). Creatine kinase isoenzyme B activity was increased at 241 U/l (normal range 0-25 U/l). An echocardiogram showed an anteroapical aneurysm. A pyrophosphate scan showed an area of uptake in the anterolateral wall and apex of the left ventricle, and a gated blood pool scan indicated hypokinesia of the apex.



Electrocardiogram on day 1 showing acute anterolateral myocardial infarction.

The ejection fraction was 69%. He regained consciousness and made a full neurological recovery; he later declined cardiac catheterisation. He gave a history of heavy solvent abuse (4-6 litres of Evostick Timebond a week) and stated that before going swimming he had been sniffing glue. He denied alcohol consumption on that day and in general or intravenous drug abuse.

Comment

Various mechanisms have been postulated on how hydrocarbons cause death; they include medullary paralysis, respiratory failure, and cardiac arrhythmias.[2] Arrhythmias occurred in dogs allowed to breathe aerosols propelled with fluorinated hydrocarbons; the rhythm disturbances consisted mainly of slowing of the rate of the sinoatrial pacemaker and ultimate electrical asystole or ventricular fibrillation in some animals.[3] Taylor and Harris subjected mice to fluoroalkane and thereby produced brady-arrhythmias, tachyarrhythmias, and myocardial depression.[4] Myocardial infarction has been described in people using paint strippers containing methyl chloride in enclosed spaces.[5] In experimental animals inhalation of hydrocarbons has also resulted in depressed myocardial activity and reduced heart rate and stroke volume. Toluene, which is the solvent in Evostick, is known to cause ataxia, euphoria, vomiting, and chest tightness. Depression of the central nervous system, tachypnoea, and tremor leading to convulsions, coma, and ventricular arrhythmias have also been described.

We postulate that our patient developed coronary artery spasm which led to anterior myocardial infarction and primary ventricular fibrillation. An alternative but less likely hypothesis is that he suffered an episode of primary ventricular fibrillation after solvent abuse. Resuscitation was prolonged and possibly caused trauma, bruising, and oedema; this may have caused injury to the coronary circulation. A diagnosis of toxic myocarditis was considered but rejected because of the classical electrocardiographic changes of anterior myocardial infarction.

We believe that this is the first documented case of successful resuscitation outside hospital of a patient suffering ventricular fibrillation after solvent abuse. This highlights the ominous consequences of this form of drug abuse.

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Ectopic pregnancy in Finland 1967-83: a massive increase

The incidence of ectopic pregnancy is increasing[1]; this rise has been exceptionally rapid in Finland and is a cause of alarm because of the morbidity of ectopic pregnancy.

Subjects, methods, and results

I report the annual rates of ectopic pregnancy in Finland, expressed per 100 deliveries, per 100 pregnancies, and per 100 000 women aged 15-44 (table).

Annual number of ectopic pregnancies per 100 deliveries, 100 observed pregnancies, and 100 000 women aged 15-44 in Finland from 1967 to 1983

Year	No of ectopic pregnancies	No/100 deliveries	No/100 observed pregnancies	No/100 000 women aged 15-44
1967	587	0·8	0·7	45
1968	549	0·7	0·7	54
1969	464	0·6	0·6	46
1970	502	0·8	0·6	50
1971	506	0·8	0·6	50
1972	562	1·0	0·6	54
1973	598	1·1	0·7	57
1974	798	1·3	0·9	76
1975	859	1·3	0·9	81
1976	938	1·4	1·0	88
1977	1070	1·6	1·2	100
1978	1165	1·8	1·3	108
1979	1274	2·0	1·5	118
1980	1351	2·2	1·6	124
1981	1477	2·3	1·7	135
1982	1581	2·4	1·8	143
1983	1736	2·6	2·0	156

The data include all ectopic pregnancies, legal induced abortions, miscarriages, and deliveries coded according to the International Classification of Diseases of 1969. Each year since 1967 the National Board of Health has registered the diagnosis for all patients attending any hospital in Finland. The statistics of the Population Register Centre of Finland were used for demographic data.

From 1967 to 1983 there were 16 017 ectopic pregnancies in Finland. During 1967-73 the rates were stable; from 1974 all the rates increased in a linear fashion. From 1973 to 1983 the number of ectopic pregnancies per 100 000 women increased 2·7-fold. During the whole study period the rates in women aged 15-19 and 40-44 did not change. A sharp increase was first seen in 1970 in women aged 20-24, then in 1972, 1973, and 1976 in women aged 25-29, 30-34, and 35-39, respectively. All of these rates increased until 1983 except in women aged 20-24, in whom they levelled off from 1978. There was a significant increase in ectopic pregnancy between 1969-73 and 1974-8 ($p<0·0005$, one way analysis of variance). In the five years 1979-83 the increase continued ($p<0·0025$, one way analysis of variance).

Comment

It has been maintained that the recent increase in the incidence of ectopic pregnancy is not as large as some reports would lead us to believe.[2] This has been assumed to be due to biased methods of reporting. Indeed, the collection of data on the incidence of ectopic pregnancy often suffers from methodological flaws.[3] If interpopulation and intrapopulation comparisons of rates are to be reliable women aged 15-44 should be used for reference.[4] All the rates in 1983 presented here are higher than those in any nationwide studies previously reported.[1-5] The rate per 100 deliveries is even close to the highest such rates ever reported, in hospital studies from Saigon, Jamaica, and Taiwan.[1] The current figures from Finland show a severe national epidemic of ectopic pregnancy.

Why the incidence of ectopic pregnancy is increasing to such an extent is not known.[1-4] Further questions are for how long the rise will continue and at what level it will stop. The number of ectopic pregnancies in any population is related to the prevalence of fertile women exposed to the chance of pregnancy and to the distribution of risk factors for ectopic pregnancy among such women; risk factors include increased age, tubal damage after infection and postoperatively, other tubal lesions, and contraceptive use.[5] In Finland the number of women in their 30s—an age at high risk—has increased considerably since the early 1970s. Moreover, the use of intrauterine contraceptive devices became popular at this time; pelvic (venereal) infections were common then; but the use of sensitive diagnostic tools for ectopic pregnancy did not become routine until the late 1970s. The sharp rise in the incidence of ectopic pregnancy per 100 000 women suggests