Alcohol: matters of life and death

Most of us enjoy alcohol and drink moderately, but some 7 million people in Britain drink more than the old 'safe' levels of alcohol — 21 and 14 units a week for men and women respectively. This represents one in eight of the population, or one in six if you exclude children under 15 years of age, difficult to justify these days given the amount of drinking by young children.

These levels were set in the late 1980s by professional consensus; when it became clear that alcohol could prevent deaths from ischaemic heart disease, the government, with minimal medical consultation, upped the quantities to 3–4 units a day for men and 2–3 units for women, although experimental evidence suggested that as little as 1–2 units three times a week are protective.

Some 4 million people are heavy drinkers, and nearly a million will suffer problems as a result. However, alarming as these figures may appear, 'only' one in four is seriously harmed. The selectivity of alcohol damage is one of the unsolved riddles of alcohol misuse; the two case reports in this issue shed some light on why only a minority of heavy drinkers succumb.

MORBIDITY AND MORTALITY

Doctors, concerned as they are with disease, do not always appreciate that most of the damage from alcohol is social and economic: examples are unemployment, financial difficulties, homelessness, criminal offences, and disruption of families by divorce, violence, and abuse of children. Only 10–20% suffer physical harm, although alcohol has the potential to damage every organ in the body.

While doctors are familiar with morbidity from cirrhosis, cardiomyopathy, pancreatitis and brain damage (all relatively uncommon), they may not recognize an association with, for example, hypertension, cardiac arrhythmias (see below), chest infections such as pneumonia and tuberculosis, gastrointestinal complaints, anxiety and depression, gynaecological problems and infertility. These are only a few of the possibilities, and since heavy drinking is widespread and reducing consumption could be beneficial, doctors in all specialties need to make connections by taking a proper alcohol history in every patient.

We are on stony ground when it comes to assessing mortality, because underreporting of alcohol use, failure to recognize its role in illness and injury, lack of comprehensive surveys and difficulties in defining 'alcohol-related' make it almost impossible to come up with accurate figures. The Office for National Statistics (1998), for example, reported nearly 4500 deaths in England and Wales in 1996, based on International Classification of Disease (ICD) codes, but it is widely accepted that alcohol could be responsible, directly or indirectly, for as many as 28–33 000 deaths a year (Godfrey and Maynard, 1992).

Half the deaths are the result of accidents and violence, including suicide; the remainder are made up of liver disease, pancreatitis, cancer, brain damage, 'heart attacks' (probably arrhythmias), and stroke. They are often premature — the average age at death from cirrhosis, for example, is 55 years — and women die earlier in their drinking career than men. Binge drinking, mostly in young social drinkers (defined as 10 or more units for men and 7 or more for women at any one time), and drinking bouts in dependent drinkers, especially when they take place on an empty stomach, are particularly dangerous, because of metabolic disturbances, cardiac arrhythmia or inhalation of vomit.

INTOXICATION

The case reports in this issue illustrate two of these life-threatening complications which, but for the authors' expertise, might well have proved fatal.

The first patient (p. 210) illuminates another aspect of alcohol misuse which is still not sufficiently recognized by doctors. Atrial fibrillation in social drinkers after a binge was reported in England by Thornton as long ago as 1984; in America it had been dubbed 'holiday heart' because it was a common cause of hospital admission at weekends, especially in people unaccustomed to large quantities of alcohol (Ettinger et al, 1978). Since then a variety of potentially life-threatening cardiac arrhythmias, especially in chronic heavy drinkers, have been reported (Koskinen and Kupari, 1992). These occur in the absence of cardiomyopathy, consist of ventricular tachycardias, and are associated with deficiencies of potassium and magnesium as in the present patient. Day et al (1993) noted prolonged QT intervals in patients with alcoholic cirrhosis, and demonstrated that they were a predictor of sudden death, especially when the prolongation was pronounced.

Anyone reading the clinical history of the second patient (p. 212) might be forgiven for making a diagnosis of severe alcohol intoxication with well-recognized metabolic disturbances: hypoglycaemia (a particular danger in intoxicated children) from depleted liver glycogen stores and inhibition of gluconeogenesis, and deficiencies of electrolytes such as potassium and magnesium. Indeed the case report is reminiscent of alcoholic ketoacidosis, in which a period of heavy drinking

without food is followed by abdominal pain and vomiting, stupor and sometimes sudden death. This was first reported in England by Thompson et al (1986), although it was already well known in the United States. Oxidation of the excess alcohol is thought to result in increased ketogenesis and inhibition of gluconeogenesis, and response to intravenous fluids, dextrose and electrolytes is usually rapid.

The striking finding in the present patient, however, was the presence of an inherited multiple acyl-CoA dehydrogenase deficiency (MADD — not to be confused with a similar acronym for Mothers Against Drink Driving), which responded to treatment with carnitine. Since dehydrogenases are responsible for the metabolism of alcohol, a deficiency of coenzymes would be expected to produce a marked sensitivity to alcohol, and it would be interesting to know the patient's previous alcohol history, and whether she had abstained since the attack. The presence of a proximal myopathy may also be relevant because it brings to mind another rare syndrome, acute rhabdomyolysis with myoglobinuria, in which a drinking bout is followed by necrosis of muscles and renal failure (Hudgson, 1984). Detailed biochemical investigation along the lines of the present report would be of great interest in this and other complications of alcohol misuse.

The enigma of physical damage resulting from alcohol is that only a minority of individuals are affected, for instance, probably as few as one in ten heavy drinkers will develop cirrhosis. Could it be that such individuals have a genetic predisposition to specific organ damage? These case reports of unusual complications of alcohol misuse suggest that detailed investigation by modern techniques, especially of biochemistry and molecular biology, might provide us with an answer. HM

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KEY POINTS

- Physical damage plays a small but important part in the total harm caused by misuse of alcohol.
- While alcohol has the potential to damage almost every organ in the body, only a minority of heavy drinkers are physically harmed.
- Newer biochemistry and molecular biology techniques might be able to resolve the paradox of selective organ damage by alcohol.

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