Misuse of ecstasy

EDITOR,—Ecstasy (3,4-methylenedioxymethylamphetamine, MDMA) has recently been associated with serious medical and psychiatric side effects.¹ We report the development of recurrent acute hepatitis associated with repeated use of ecstasy.

A 27 year old housewife was first admitted to hospital in August 1991 for investigation of her second episode of jaundice. She had experienced her first episode in January 1990. This had been investigated and managed at home by her general practitioner (SW). Non-A non-B viral hepatitis had been diagnosed provisionally, and she had recovered fully. She did not drink alcohol and did not have a history of recent contact with a jaundiced person or travel abroad. She had used intravenous drugs in the past but not since 1987.

On examination she was unwell and deeply jaundiced. Palmar erythema was present, but there were no other signs of chronic liver disease. The liver edge was palpable 3 cm below the costal margin. The bilirubin concentration was 471 µmol/l (normal 3-17 µmol/l), aspartate aminotransferase 1717 IU/l (10-42 IU/l), alkaline phosphatase 439 IU/l (80-250 IU/l), and γ glutamyl transpeptidase 50 IU/l (15-35 IU/l). Her serum albumin concentration, white cell count, and international normalised ratio were normal, and serological tests for hepatitis A, B, and C virus, Epstein-Barr virus, cytomegalovirus, Q fever, psittacosis, mycoplasma, and HIV all gave negative results. An autoantibody screen was negative, and an ultrasound scan of the liver was normal.

Liver biopsy showed a florid acute hepatitis with both portal and lobular components (figure). The infiltrating cells were predominantly mononuclear, but there were also appreciable numbers of eosinophils and neutrophils, especially in the portal tracts. The lobular inflammation was associated with lobular disarray and centrilobular

Florid hepatitis with inflammatory cell infiltrate and lobular disarray (arrow, also seen throughout field), centrilobular drop out of hepatocytes (arrow head), acidophilic cell formation (open arrow), and multinucleation of Kupffer cells (double arrow). (Haematoxylin and eosin)

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drop out of hepatocytes. Numerous acidophilic cells were seen, and some Kupffer cells and hepatocytes showed multinucleation.

The patient's condition resolved spontaneously, but in December she suffered a further episode of jaundice, which lasted for three weeks. The clinical picture was as before. In February 1992, prompted by a feature in the *Sunday Times* highlighting the physical dangers of misuse of ecstasy,² the patient presented to her general practitioner and admitted to having used ecstasy before each episode of jaundice. The interval between ingestion of the drug and the initial symptoms (eight to 15 days), together with the histological findings, suggested that she had had an idiosyncratic response to either ecstasy or a contaminant of the preparation.

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EDITOR,—John Henry draws attention to the dangers of ecstasy (3,4-methylenedioxymethylamphetamine, MDMA).¹ This increasingly popular drug of misuse is reputedly safe, although deaths have occurred after acute ingestion.^{2,4} We report a case of jaundice associated with recreational use of ecstasy.

A previously well 20 year old student presented with a 10 day history of jaundice and a three week history of nausea, anorexia, pale motions, and dark urine. He was not taking any prescribed drugs but had been taking one or two tablets of ecstasy at weekend parties for three months. He had ingested about 20 tablets over this period, the last tablet four weeks before presentation. He did not have a history of intravenous drug misuse or of use of any other illicit drugs. Alcohol consumption was moderate (four pints of beer a week), and he had not ingested paracetamol or travelled abroad recently.

On examination he was jaundiced but had a normal temperature and was otherwise well. Abdominal palpation showed no abnormality. Serum bilirubin concentration was 131 µmol/l, aspartate aminotransferase 2050 U/l, and alkaline phosphatase 201 U/l. Screening for hepatitis A, B, and C virus, cytomegalovirus, and Epstein-Barr virus yielded negative results. Serum copper and caeruloplasmin concentrations were normal, and antinuclear and smooth muscle antibody levels were negative. His blood count was normal

apart from a mild eosinophilia (8%). During the next week the bilirubin concentration rose to 330 µmol/l and the prothrombin time peaked at 21 s (international normalised ratio 1.5). Liver biopsy showed features of severe, current acute hepatitis with lobular disarray and areas of loss of hepatocytes, particularly in periportal sites. Inflammatory infiltrate consisted predominantly of plasma cells, lymphocytes, and, to a lesser extent, eosinophils. Over the next month the patient's jaundice resolved and serum liver biochemical variables returned to normal. Six months after his illness his serum liver biochemical variables remained normal and he was negative for antibody to hepatitis C virus.

Jaundice has not previously been reported as a complication of long term use of ecstasy. One woman who ingested a large dose of ecstasy, which led to hyperthermia and hypotension and necessitated cardiovascular and respiratory support, also developed hyperbilirubinaemia and raised aspartate aminotransferase during her acute illness.⁵ Our patient's hepatic illness developed many days after use of recreational doses of ecstasy. Although we cannot exclude harmful effects of possible contaminants in the "packaging" of the drug, we believe that hepatitis should be recognised as a complication of use of ecstasy.

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EDITOR,—Teenagers who take ecstasy and dance¹ may present to hospital emergency departments with acute severe chest pain.

Two normally fit male teenagers and one female teenager presented to us between 3 am and 6 am with severe central chest pain. They had all danced for some hours and admitted having taken ecstasy and alcohol. The pain was localised to the chest, and there was no associated dyspnoea. Anxiety and sweating were the only other prominent features. Pulse rate and blood pressure were mildly raised. Cardiorespiratory examination yielded otherwise unremarkable findings. Apart from the heart rate, their electrocardiograms were normal, as were their chest radiographs and results of routine blood tests. All three discharged themselves after observation and after learning that their pain was unlikely to be cardiac in origin: they did not want their parents to find out that they had taken ecstasy.

Though severe tachycardia and hypertension may induce angina, we think that the most likely cause of the pain in these cases was spasm of the intercostal muscles. Ingestion of ecstasy should be