SHORT REPORTS

Oesophageal pain exacerbated by propranolol

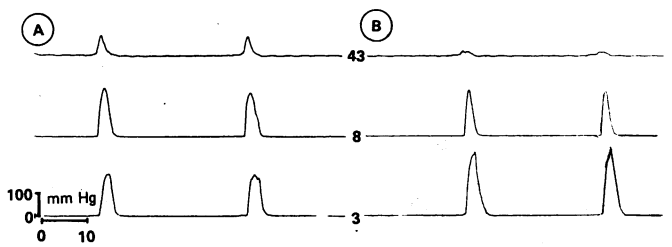
Chest pain suggestive of angina is characteristic of the nutcracker oesophagus, one of the commonest motor disorders of the oesophagus. The disorder is characterised by high amplitude peristaltic contractions and related to diffuse oesophageal spasm.[1,2] Distinguishing it clinically from ischaemic heart disease, especially in older patients, is often difficult. If antianginal drugs are given for the symptomatic relief of chest pain of uncertain origin they may not only fail to discriminate between an oesophageal and cardiac cause of the pain but also lead to an exacerbation of oesophageal symptoms. We report a case of chest pain of oesophageal origin that deteriorated when the patient was given propranolol.

Case report

A 68 year old man with mild hypertension well controlled with a diuretic was referred in October 1984 with a six month history of intermittent chest pain suggestive of angina. Previous investigations in a cardiology unit had failed to detect any abnormality, but he had been discharged with a diagnosis of probable angina. As the episodes of chest pain recurred and were prolonged propranolol 40 mg four times daily was started in September. A few days later he experienced an exacerbation of his chest pain and a crushing sensation in the mid-sternum. Cardiac examination yielded normal results, and he decided to stop taking the propranolol.

He was subsequently referred for further investigation when the only abnormality found on examination was obvious anxiety. Oesophageal manometry performed by standard techniques showed a lower oesophageal sphincter pressure of 22 mm Hg (normal range in our laboratory 10-35 mm Hg) with a normal pattern of relaxation.[1,2] The body of the oesophagus showed normal peristaltic activity after swallowing fluid, with a mean distal contraction amplitude of 200 mm Hg (normal mean amplitude 100 mm Hg, range 45-180 mm Hg). Some contractions showed peak pressures of up to 280 mm Hg. Nutcracker oesophagus was diagnosed.

Propranolol was suspected as the cause of the worsening symptoms, so he was rechallenged with the drug. Within 24 hours his chest pain recurred. Repeat manometric studies showed a lower oesophageal sphincter pressure of 20 mm Hg with normal relaxations. The mean amplitude of the distal oesophageal contractions was 295 mm Hg (figure) with peak values above 400 mm Hg. Propranolol was stopped and nifedipine 10 mg three times daily started. He subsequently had no further chest pain.



Manometric tracings before (A) and after (B) propranolol.

Comment

Under physiological conditions the body of the oesophagus seems to be under β adrenergic inhibitory influence, and it has been suggested that β receptors have an inhibitory effect on smooth muscle.[3-5] Propranolol (10 mg intravenously) has been shown to increase distal oesophageal contractions.[3] Given the close temporal relation between the exacerbation of our patient's chest pain and his taking propranolol we believe that the propranolol was responsible for the worsening of his symptoms.

This view is supported by the positive result of the rechallenge test. Propranolol along with other β adrenergic blockers may increase the amplitude of the oesophageal contractions in patients with nutcracker oesophagus, thereby increasing the sensation of pain.

Patients with chest pain suggestive of angina should be investigated carefully before treatment with β adrenergic blockers is given. In cases in which the diagnosis is in doubt it is probably advisable to start the patient on nifedipine, which reduces the amplitude of oesophageal contractions.[2]

We are indebted to Judy Dale-Etherington for reviewing the English version of the manuscript.

1 Narducci F, Bassotti G, Gaburri M, Morelli A. Transition from nutcracker esophagus to diffuse esophageal spasm. *Am J Gastroenterol* 1985;80:242-4.
2 Richter JE, Dalton CB, Buice RG, Castell DO. Nifedipine: a potent inhibitor of contractions in the body of the human esophagus. Studies in healthy volunteers and patients with the nutcracker esophagus. *Gastroenterology* 1985;89:549-54.
3 Lyrenäs E. Beta adrenergic influence on esophageal and colonic motility in man. *Scand J Gastroenterol* 1985;20(suppl 116):1-48.
4 Lyrenäs E, Abrahamsson H. Beta adrenergic influence on oesophageal peristalsis in man. *Gut* 1986;27:260-6.
5 Gillespie JS, Khoy MA. The site and receptors responsible for the inhibition by sympathetic nerves of intestinal smooth muscle and its parasympathetic motor nerve. *J Physiol* 1977;267:767-89.

(Accepted 6 March 1987)

Gastrointestinal Motility Laboratory, Gastrointestinal Unit, First Department of Internal Medicine, University of Perugia, Perugia, Italy

GABRIO BASSOTTI, MD, fellow in gastroenterology
MANUELA GABURRI, MD, fellow in gastroenterology
MARIA ANTONIETTA PELLI, MD, associate professor of digestive pathophysiology
ANTONIO MORELLI, MD, associate professor of gastroenterology

Correspondence to: Dr G Bassotti, Clinica Medica 1, Policlinico Monteluce, PO Box 41, 06100 Perugia, Italy.

Hypoglycaemia in insulin dependent diabetics: is advice heeded?

Euglycaemia remains the ultimate goal in the treatment of insulin dependent diabetes mellitus. It is not, however, without the inherent risks of hypoglycaemia and its associated morbidity and mortality.[1] Recognition of the early symptoms of hypoglycaemia by the patient and his relatives or colleagues is important because prompt ingestion of a rapidly absorbable carbohydrate (sugar lumps or glucose tablets) may prevent neuroglycopenia.

The British Diabetic Association recommends that insulin dependent diabetics should always (*a*) carry some form of easily ingestible sugar, (*b*) carry clear evidence that they take insulin, and (*c*) inform relatives or colleagues of the possibility of hypoglycaemic episodes.[2] We undertook a study to establish how well this advice is heeded.

Patients, methods, and results

Three hundred consecutive insulin dependent diabetics attending a diabetic clinic over two months were asked to complete an anonymous questionnaire covering the recommendations of the British Diabetic Association on hypoglycaemia. They were also asked about any episode of unconsciousness away from home due to hypoglycaemia (table). A total of 275 (91·7%) forms were returned and were suitable for analysis.

Eighty nine patients (32·4%) did not usually carry sugar with them, and 32 (23%) drivers did not have a readily accessible separate supply of sugar in their car. Ninety two patients (33·5%) did not carry evidence of their diabetes, although most (93·1%) had told their colleagues.

Sixty six patients (24·0%) had at one time experienced a severe hypoglycaemic episode away from home which required physical assistance or admission to hospital. Nearly half of these (25 patients) were not carrying sugar at the time, and most (17/25) thought that hypoglycaemia might have been prevented if sugar had

Clinical details of patients studied

	Men (n=156)	Women (n=110)
Mean age in years (range)	40·2 (14-75)	44·3 (11-81)
Mean duration of insulin treatment in years (range)	13·5 (1-54)	13·8 (1-51)
No (%) of patients who always carried identification	97 (62·2)	86 (72·3)
No (%) of patients who always carried a source of sugar	104 (67·7)	82 (68·9)
No of drivers	108	33
No (%) of drivers with a separate supply of sugar in the car	85 (78·7)	24 (72·7)
No (%) of patients who had collapsed away from home	46 (29·5)	20 (16·8)
No of patients who had been taken to hospital	32	13

been available. Loss of consciousness in those carrying sugar occurred either because hypoglycaemic symptoms were not recognised or because the onset of hypoglycaemia was particularly swift.

Comment

Although the true incidence of hypoglycaemia is unknown, up to 90% of adult insulin dependent patients have experienced hypoglycaemic symptoms.[3] Although great emphasis is placed on recognising symptoms and early self treatment, nearly a third of our patients were not aware of or did not follow the recommendations of the British Diabetic Association on hypoglycaemia. Serious hypoglycaemic episodes occur in up to 9% of insulin dependent diabetics each year.[4] If the British Diabetic Association's guidelines were followed unnecessary morbidity and mortality related to hypoglycaemia could be prevented.

The failure of 23·0% of the drivers in our study to carry a separate easily accessible supply of sugar in the car puts not only themselves but other road users at increased risk. Up to 40% of insulin dependent diabetics who drive experience hypoglycaemia while at the wheel of a car,[5] and a separate source of glucose in the vehicle is therefore essential as jackets and handbags are easily forgotten.

Those caring for diabetics need to educate their patients (particularly drivers) on the hazards of hypoglycaemia and should check that their advice is being heeded. The sound advice of the British Diabetic Association[2] is clearly not being taken by up to a third of our insulin dependent diabetics.

1 Tunbridge WMG. Factors contributing to deaths of diabetics under 50 years of age. *Lancet* 1981;ii:569-72.
2 Day JL. *The diabetes handbook (insulin dependent diabetes)*. Wellingborough: Thorsons Publications, 1986:88-94.
3 Malins JM. *Clinical diabetes*. London: Eyre and Spottiswoode, 1968.
4 Potter J, Clarke P, Gale EAM, Dave SH, Tattersall RB. Insulin induced hypoglycaemia in an accident and emergency department. The tip of an iceberg? *Br Med J* 1982;285:1180-2.
5 Clarke B, Ward JD, Enoch BA. Hypoglycaemia in insulin dependent diabetic drivers. *Br Med J* 1980;281:586.

(Accepted 26 March 1987)

Diabetic Unit, Leicester Royal Infirmary, Leicester LE1 5WW
P G McNALLY, BMEDSCI, MRCP, medical registrar
D H MacIVER, MB, MRCP, medical registrar
N I JOWETT, MD, MRCP, senior medical registrar
J R HEARNSHAW, BSC, FRCP, consultant physician

Correspondence to: Dr Jowett.

Possible reactivation of hepatitis D with chronic δ antigenaemia by human immunodeficiency virus

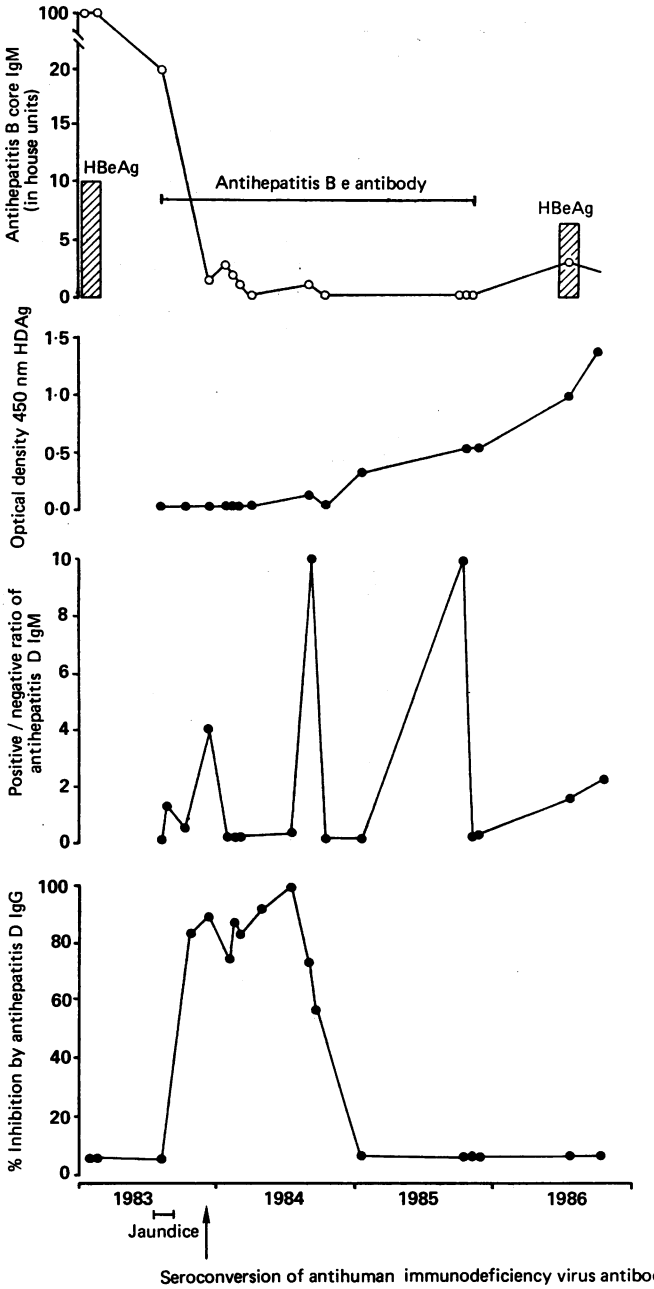
We describe a case of reactivation of infection with hepatitis D virus (δ) in an intravenous drug abuser who was a carrier of hepatitis B surface antigen (HBsAg). This condition was manifested by chronic δ antigenaemia of at least two years' standing and was probably induced by infection with human immunodeficiency virus.

Case report

Detailed serological tests for hepatitis B and D viruses were carried out on a series of specimens from a 21 year old male drug abuser in whom acute hepatitis B had been diagnosed in February 1983. At that time HBsAg was detected by commercial radioimmunoassay and enzyme immunoassay kits (Abbott); hepatitis B e antigen (HBeAg) and antihepatitis B core IgM were detected by in house enzyme immunoassays.[1] There was no jaundice and his liver enzyme activities were slightly raised. Jaundice of three weeks' duration occurred six months later, however, in August 1983.

Antihepatitis D IgG was detected in October 1983 by enzyme immunoassay (Deltassay B, Noctech). Antihepatitis D IgM was also detected by an in house enzyme linked immunosorbent assay (ELISA) using hepatitis D antigen (HDAg) derived from serum, which indicated that his hepatitis in August had been caused by a superinfection with hepatitis D virus. Antihepatitis D IgG persisted throughout 1984.

Reactivation of infection with hepatitis D virus was first detected in July 1984, when transient HDAg appeared resulting in seroconversion to antihepatitis D IgM. Reactivation was again detected in January 1985, after which he showed increasing amounts of serum HDAg (detected by enzyme immunoassay Deltassay, Noctech) with loss of antihepatitis D IgG and fluctuating amounts of antihepatitis D IgM.



Temporal changes in results of detailed serological tests for hepatitis B and D viruses in 21 year old drug abuser.

Tests for antibody to human immunodeficiency virus were carried out retrospectively. Enzyme immunoassay gave an equivocal result at first, but a Western blot analysis (using DuPont preprepared nitrocellulose strips) produced a positive result in his specimen of October 1983; all subsequent tests (Wellcome enzyme immunoassay) remained positive. The activities of his liver enzymes were persistently raised and variable throughout, and a liver biopsy in October 1984 indicated the presence of chronic active hepatitis. In October 1986 he had lymphadenopathy but had not returned for follow up at the time of writing.

Comment

The serological results strongly suggest that our patient acquired superinfection with hepatitis D virus and human immunodeficiency virus about six months after acute infection with hepatitis B virus. The unusual serological pattern and the rising concentration of HDAg in serum strongly suggest reactivation of replication of hepatitis D virus. Furthermore, his most recent specimens showed reversion from antihepatitis B e antibody to HBeAg, which indicates reactivation of hepatitis B virus as well. Unfortunately the patient has been lost to follow up and the importance of this reactivation of hepatitis D virus is therefore unknown. Although this is the first reported occurrence of reactivation of infection with hepatitis D virus by human immunodeficiency virus, the large numbers of drug abusers