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

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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