Irritable bowel syndrome—a safe diagnosis?

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Abstract

Seventy-seven of 84 surviving patients in whom the irritable bowel syndrome had been diagnosed at least six years previously were reviewed. A different diagnosis was made in only four cases. Forty-four patients remained symptomatic and 29 patients had no further bowel problems.

The irritable bowel syndrome is often a chronic, relapsing disorder, and further investigations are not necessary unless symptoms change considerably.

Introduction

The irritable bowel syndrome is probably the commonest diagnosis made in gastroenterology clinics. Unfortunately, the clinical features, though suggestive,¹ are not pathognomonic, and this diagnosis is acceptable only if organic gut disease has been excluded. The difficulty is deciding how extensive investigations should be before the diagnosis of the irritable bowel syndrome may be accepted with reasonable confidence. The fear of missing organic disease is understandable but exhaustive investigations are not always in the patient's interest.

When our combined medicosurgical gastroenterology clinic started in 1972 we accepted the diagnosis of the irritable bowel syndrome if the criteria detailed below were fulfilled. There have been comparatively few follow-up studies of this disorder, however, and to check the safety of these criteria we reviewed all cases of irritable bowel syndrome diagnosed from 1973 to 1975. In addition to assessing the correctness of the initial diagnosis, we also investigated the course of the patient's symptoms during the study period.

Patients and methods

The case records of all patients with the irritable bowel syndrome attending a combined medicosurgical gastroenterology outpatient clinic² from 1973 to 1975 were reviewed. Our criteria for the acceptance of this diagnosis were: recurrent abdominal pain associated with a disturbance of bowel rhythm (either constipation or diarrhoea) and sometimes the passage of mucus per rectum; normal findings on physical examination, sigmoidoscopy, and double-contrast barium enema; normal blood count, sedimentation rate, and serum protein concentrations; and negative results of faecal occult blood test.

Ninety-one patients fulfilled these criteria. Seven had died, seven had left the area and could not be contacted, and the remaining 77 (a follow-up rate of 85%) were recalled. The 77 patients (32 men and 45 women) were aged from 22 to 86 years (mean 56.7). A history was taken from all patients, including detailed questioning about gastrointestinal symptoms and general health: further physical examination and investigations were undertaken only if clinically indicated.

Results

Seven patients (four men and three women) had died since diagnosis. Two of the men died of myocardial infarction, one of cerebral arteriosclerosis, and one of bronchogenic carcinoma. One of the women died of myocardial infarction and one of bronchopneumonia. In the third case (an 81-year-old woman) gastric carcinoma was the certified cause of death, but there was no hospital record to substantiate this diagnosis, the general practitioner's records were not available, and no necropsy had been conducted.

A total of 44 of the 77 survivors (18 men and 26 women) still had symptoms of the irritable bowel syndrome, 29 had no bowel problems. Persistent symptoms had led to further investigation in another four cases, resulting in alternative diagnoses. One woman developed dyspepsia after many years of diarrhoea and passage of mucus per rectum; gastric ulceration was diagnosed. Another developed weight loss and a sore tongue; radiographs of the small intestine showed jejunal diverticula. The remaining two women were found to have thyroid dysfunction: one developed hyperthyroidism and improved after treatment with carbimazole, the other had hypothyroidism with diverticular disease of the colon.

Discussion

Most patients are referred to gastroenterology clinics with disorders of gastrointestinal function rather than organic disease. The irritable bowel syndrome is the most common diagnosis in these patients, but unfortunately is essentially a diagnosis of exclusion. Our findings confirm that this diagnosis may be made with an acceptable degree of safety provided the initial clinical assessment is thorough and includes sigmoidoscopy and blood screening: only two visits to hospital are required. One further visit is required for barium enema examination, which must be performed using a double-contrast technique.

Clinicians, understandably afraid of overlooking organic disease, tend to make the diagnosis of the irritable bowel syndrome with reluctance and may pursue investigations to extraordinary lengths.³ Follow-up studies have been comparatively rare^{4,5} and we believe our own 85% follow-up over six years provides ample justification for keeping investigations to a minimum. The fear of missing malignant disease of the gastrointestinal tract is an understandable worry,⁵ but in our series only one patient was later suspected of having gastric carcinoma and there was no radiological, histological, or necropsy evidence to support the certified cause of death.

The irritable bowel syndrome is generally accepted as being more common in the younger age group. The age range of patients in our follow-up study was older than we would have expected. This results from our insistence on a negative finding on double-contrast barium enema as one of the criteria for inclusion: we have been reluctant to carry out this investigation in younger patients in whom organic disease could be confidently excluded by careful clinical assessment, sigmoidoscopy, and blood investigations alone. A consequence of the age bias is that our patients would be theoretically more at risk from malignant disease. It is therefore reassuring that malignancy was diagnosed in only one case—and even then the diagnosis was doubtful. Nevertheless, we would not dispute that the irritable bowel syndrome should be diagnosed with some caution in patients of middle age or older who present with abdominal symptoms for the first time.

Two patients were found to have thyroid dysfunction, which has been noted more often in patients referred to the clinic

since the study period. In-vitro tests of thyroid function have become more specific and more available recently and we believe that these should now be included in the routine investigations whenever there is the slightest suspicion that hyperthyroidism or hypothyroidism might account for a patient's presenting bowel disorder.

A total of 57% of the surviving patients (18 men and 26 women) still had their symptoms at follow up, though most had learnt to live with the problem and confirmed that reassurance and explanation were particularly important in treatment. This confirms the conclusions of previous studies that the irritable bowel syndrome is often a chronic, relapsing disorder^{4,6} and further investigations are not necessary unless there is an appreciable change in the symptom pattern.

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SHORT REPORTS

Bromocriptine-associated hyponatraemia in cirrhosis

Bromocriptine (2-bromo- α -ergocryptine) is a dopaminergic agonist that has been used to treat chronic portosystemic encephalopathy in selected patients.¹ We report on such a patient in whom use of bromocriptine resulted in profound hyponatraemia.

Case report

A 60-year-old man with inactive cirrhosis was admitted to hospital after sustaining a variceal haemorrhage. He had never abused alcohol. On examination he had hepatosplenomegaly, moderate ascites, and features of portosystemic encephalopathy. Serum sodium concentration was 130 mmol (mEq)/l (normal 136-148 mmol/l) and urinary sodium excretion 1 mmol/l (normal 50-120 mmol/l) (figure); serum and urinary osmolalities were appropriate at 261 and 209 mmol (mosmol)/kg.

Changes occurring in serum sodium concentration, urinary sodium excretion, and weight during treatment with bromocriptine and subsequent challenge with the drug. Dietary sodium intake was constant throughout and fluid intake never more than 1 l daily.

Conversion: SI to traditional units—Sodium: 1 mmol/l = 1 mEq/l.

The variceal haemorrhage stopped without vasopressin. Fluid retention was treated by restricting fluids and sodium and administering spironolactone, up to 200 mg daily. His encephalopathy responded initially to protein restriction and lactulose, but later bromocriptine was added to a final dose of 15 mg daily. Seven weeks later his mental state had deteriorated and he was confused. The serum sodium concentration was 116 mmol/l (figure) with normal blood urea and serum potassium concentrations. Spironolactone was stopped and fluid intake restricted to 500 ml daily, but there was no improvement. Three days later, when the serum sodium concentration was 113 mmol/l,

urinary sodium excretion 47 mmol/l, and serum and urinary osmolalities 242 and 639 mmol/kg, the bromocriptine was stopped. Within 24 hours urinary sodium excretion was 3 mmol/l and osmolality 234 mmol/kg. Over the next two weeks his clinical condition and electrolyte abnormalities improved; he had a moderate diuresis and lost 2 kg in weight.

The development of hyponatraemia with continued renal excretion of sodium during treatment with bromocriptine suggested inappropriate secretion of antidiuretic hormone. He was restabilised taking 20 mmol sodium and 1 l fluid daily. Plasma cortisol concentrations were normal. Plasma renin activity was 10.4 nmol/l/h (13.5 ng/ml/h) (normal 0.8-3.5 nmol/l/h; 1.0-4.5 ng/ml/h) and plasma aldosterone concentration 13.5 nmol/l (0.49 μ g/100 ml) (normal 0.3-1.1 nmol/l; 0.01-0.04 μ g/100 ml), both measured with the patient supine. The rate of urinary excretion of antidiuretic hormone was 144 fmol/min (156 pg/min) (normal 40-90 fmol/min; 43-98 pg/min).

With the patient's consent bromocriptine was reintroduced at a dose of 7.5 mg daily. Within five days the serum sodium concentration had fallen to 118 mmol/l and urinary sodium increased to 21 mmol/l; serum and urinary osmolalities were 268 and 433 mmol/kg. Plasma renin activity (9.2 nmol/l/h; 12.0 ng/ml/h), plasma aldosterone concentration (9.0 nmol/l; 0.33 μ g/100 ml) and the rate of urinary excretion of antidiuretic hormone (163 fmol/min; 177 pg/min) did not change appreciably. Bromocriptine was stopped with prompt improvement in the electrolyte abnormalities.

Comment

Hyponatraemia is common in cirrhosis; the mechanism is unclear since total body sodium is often increased. Bromocriptine contributed substantially to the hyponatraemia in this patient, although how it did so is necessarily speculative. Several features suggested that the drug induced inappropriate secretion of antidiuretic hormone. Urinary sodium excretion continued despite profound hyponatraemia, and the urinary osmolality was inappropriately higher than the serum osmolality; the patient was not dehydrated and had normal renal and adrenal function. Pretreatment urinary excretion of antidiuretic hormone was, however, high, a feature often observed in patients with cirrhosis,² and it did not change substantially when the drug was reintroduced. This does not exclude inappropriate secretion of antidiuretic hormone since plasma concentrations of the hormone were not measured; indeed, bromocriptine stimulates secretion of antidiuretic hormone in normal subjects.³ It may possibly also alter renal responsiveness to circulating antidiuretic hormone; as a result expansion of the circulating fluid volume and secondary natriuresis might follow. Bromocriptine can produce modest natriuresis either from a direct effect on the kidney or because of dopaminergic inhibition of release of aldosterone.⁴ The latter mechanism is unlikely to have been important in this patient as plasma renin activity and aldosterone concentrations were little affected by bromocriptine. The high pretreatment values reflected the patient's secondary aldosteronism. Bromocriptine consistently lowers serum prolactin concentrations, but prolactin appears to have only a minor role in regulating fluid and electrolyte balance in mammals.⁵ We suggest that bromocriptine should be used with extreme caution in patients with cirrhosis and ascites.

We thank Professor Dame Sheila Sherlock for allowing us to study a patient under her care.