BRIEF REPORT

MENETRIER'S DISEASE IN A PATIENT WITH ILEOCOLONIC CROHN'S DISEASE

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Summary

A patient with Menetrier's disease and Crohn's disease is described. It is reported not only for its extreme rarity but because of the response of the Menetrier's disease to medical treatment.

Introduction

Menetrier's disease, first described in 1888, is a rare disorder characterised by gross hypertrophy of the gastric mucosa, hyposecretion of gastric acid and excess protein loss from the stomach. Its cause is unknown, it is rarely familial and it has no regular association with other diseases (Scharschmidt, 1977). We report a case of Menetrier's disease occurring in a patient with previously diagnosed Crohn's disease. This association does not appear to have been previously described.

Case Report

A 52 year old female first presented in 1973 with a 6 month history of passing 6 loose bowel motions per day which contained mucus but no blood. She stated that she had been diagnosed as suffering from ulcerative colitis 16 years previously for which she had been put on a low residue diet. She had suffered from varicose veins since the age of 14 years. She had no other relevant past history. Her father died at the age of 62 years from carcinoma of the stomach.

On examination she was a healthy looking woman. She had extensive varicose veins of both legs with varicose eczema. She had an area of cellulitis on the lower third of her left leg. Clinical examination was otherwise unremarkable. Sigmoidoscopy showed an oedemmatous rectal mucosa with excess contact bleeding. Histology revealed transmural chronic inflammation which was considered to be consistent with Crohn's disease. Barium follow-through and Barium enema showed the changes of Crohn's disease affecting the terminal ileum, ascending and descending colon. Barium meal showed nodular thickening of the gastric folds to the lower half of the body and the antrum of the stomach. Her cellulitis settled on treatment and the patient was discharged.

She first presented to our unit in 1976 with a 6 month history of diarrhoea which had contained bright red blood on 2 occasions. She also suffered from lower abdominal pain and epigastric discomfort. There had been no weight loss. On examination she had bilateral ankle oedema and periorbital oedema; clinical examination was otherwise unremarkable.

Laboratory investigations at that time revealed a haemoglobin of 12 g/dl, E.S.R. 3 mm in 1st hour, total serum proteins 50 g/l, serum albumin 28 g/l and seromucoids 129 mg/dl. Urea and electrolytes were normal as were liver function tests; there was no proteinuria. Further barium studies and rectal biopsy confirmed the diagnosis of Crohn's disease. Repeat endoscopy again showed multiple hypertrophic gastric folds. The patient was commenced on Sulphasalazine but shortly afterwards developed an allergic type rash and the drug was discontinued.

Over the next few years she became progressively less well and developed massive oedema with 'weeping' of her legs. She had persistent diarrhoea, crampy abdominal pain and epigastric discomfort. Her total serum proteins fell to 30 g/l with serum albumin of 16 g/l. Haemoglobin was 9 g/dl. She had a platelet count of 900 x 10^9/l; her serum B12 and serum folate levels were normal; serum iron and T.I.B.C. were both reduced. Jejunal biopsy was normal. Further endoscopy and barium studies (Figure) confirmed the presence of giant rugal hypertrophy of the stomach. We felt that her protein loss might be from her stomach: 1.5% of intravenously given radioactive labelled albumin was aspirated from her stomach over the first 24 hours following injection (the gastric aspirate was negative on testing for haemoglobin). Faeces contained 0.3% of the radioactive labelled albumin on the first day, 2.6% on the second day, 4.7% on the third day, 3.3% on the fourth day, and 2.1% on the fifth day. These findings were interpreted as consistent with the major protein loss from the stomach. Fasting serum gastrin levels were normal but post...
prandial serum gastrin was moderately elevated at 280 ng/l.

The patient was commenced on azathioprine 50 mg daily and cimetidine 200 mg three times daily and 400 mg at night. Marked clinical and biochemical improvement followed over the next few months. Her abdominal pain and discomfort settled and her ankle oedema became greatly reduced. In 1981 her total serum proteins were 77 g/l with serum albumin of 46 g/l. Her haemoglobin was 14.8 g/dl, and her platelet count was normal. Today (September, 1982) she feels well and is symptom free and has no ankle oedema. Her haemoglobin is 14.4 g/l, serum albumin 43 g/l and seromucoids 117 mg/dl. Endoscopic and radiological (Figure) appearances of the stomach have virtually returned to normal. Over the years endoscopic gastric biopsies have all shown increased round cell infiltration with diminution in gastric glands but have otherwise been normal.

**Discussion**

The association of gastric mucosal hypertrophy, hypoprotein-anaemia, protein loss from the stomach and oedema in this patient with its resolution whilst on treatment over a four year period makes the diagnosis of Menetrier's disease very likely. This rare disease has not, to our knowledge, been previously described in a patient with Crohn's disease. A review of 120 cases was published by Scharschmidt in 1977: our patient exhibits many of the features of the disease as described in that paper. Whilst it is more common in men than women upper abdominal pain and discomfort was the most frequent symptom described and diarrhoea was included as a non specific complaint. Whilst oedema was reported in less than one fourth of the patients the serum albumin levels were reduced in nearly all. Allergic manifestations have been reported several times; our patient exhibited a presumed allergic response to Sulphasalazine. Thrombocytosis was described in one of Scharschmidt's patients although in his cases it was a preterminal event.

Two thirds of all patients in that survey had either a partial or total gastrectomy for persistent distressing symptoms and for the possibility of malignant disease. Operative intervention did...