Loss of duodenal folds allows diagnosis of unsuspected coeliac disease

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Abstract
We report three patients with coeliac disease who presented without the classic features of malabsorption and who underwent biopsy and were diagnosed only because of the endoscopic finding of the disappearance of Kerckring's folds in the descending duodenum. This sign constitutes a new and valid aid for the identification of patients with otherwise unsuspected coeliac disease.

We recently described the loss or appreciable reduction in Kerckring's folds in the descending duodenum of patients with coeliac disease.1 This new endoscopic sign proved to have a sensitivity of 88% and a specificity of 83%, and allowed us to identify four patients with coeliac disease which was so mild that it would otherwise have been diagnosed. Adult coeliac disease is largely underdiagnosed,2 mainly because most patients present without the classic features of malabsorption.3 Any new aid to diagnosis would therefore be welcome and we have suggested4 that a systematic search for the easily recognisable sign we described could lead to the diagnosis of coeliac patients with minimal, transient, or unrelated symptoms. We now report three additional patients in whom duodenal biopsies, performed during upper gastrointestinal endoscopy, showed a flat duodenal mucosa indicative of a previously unsuspected coeliac disease.

Case reports

PATIENT 1
A 48 year old man presented in July 1988 with weakness accompanied by dyspepsia and pyrosis. His previous medical history showed no diarrhoea or weight loss. Investigations were normal (Table). He underwent upper gastrointestinal endoscopy which showed a small sliding hiatus hernia and the complete loss of Kerckring's folds in the descending duodenum, explored after maximal insufflation (Figure). Duodenal biopsy specimens showed subtotal villous atrophy, which resolved appreciably after eight months of gluten-free diet. The patient's HLA phenotype was DR3/7, DQw2 and antigliadin antibodies were found to be positive.

PATIENT 2
A 60 year old unmarried woman presented in July 1988 with persistent dysphagia. Her previous history showed episodes of diarrhoea in early childhood only. At the age of 23 she underwent splenectomy for haemolytic anaemia, and only then did she begin to menstruate. At the age of 35 the menopause began and osteoporosis was subsequently diagnosed and treated. On presentation, her bowel habit was normal and there was no considerable weight loss. Upper gastrointestinal endoscopy showed a normal oesophagus, chronic diffuse gastritis, and a noticeable fold reduction in the second stage of the duodenum. Duodenal biopsy specimens showed subtotal villous atrophy due to coeliac disease. After diagnosis, the patient underwent a blood xylose test which was positive (Table). Her HLA phenotype was DR3/7, DQw2, and antigliadin antibodies were positive. The patient's symptoms improved after gluten-free diet.

PATIENT 3
A 51 year old woman presented in December 1988 after a persistent weight loss of 15 kg. She claimed to have no prior symptoms. Her previous medical history was negative without previous weight loss. Investigations showed a weight loss of 15 kg (Table). Her HLA phenotype was DR3/7, DQw2, and antigliadin antibodies were positive. After gluten-free diet, she started to gain weight without any other symptoms of coeliac disease.
Endoscopic associated with coeliac virus were not disease. coeliac laralities without anaemia 3),6 (patient which features iron deficiency anaemia, recurrent aphthous ulceration, dermatitis herpetiformis, recurrent episodes of colicky biliary pain caused by gall stones, and overweight that was still evident (Table). In 1982, she was admitted to another hospital for chest pain preceded by an influenza-like illness. Pericarditis was diagnosed but after treatment with a course of steroids this never recurred. With regard to her gastrointestinal problems, endoscopy showed only a reduction in the height and number of folds in the second stage of the duodenum and biopsy specimens showed subtotal villous atrophy with areas of severe partial villous atrophy. There was regrowth of the villi after six months of gluten-free diet. The patient’s HLA phenotype was DR4/5, DQw3 and antigliadin antibodies were found to be positive.

Discussion
Because none of the three patients reported here had diarrhoea or were malnourished they were not suspected of having coeliac disease. They were not identified through family studies nor were ‘warning conditions’ such as dermatitis herpetiformis, recurrent aphthous stomatitis, iron deficiency anaemia, and short stature present. Although patients 2 and 3 had some features which have been reported as being associated with coeliac disease – macrocytosis without anaemia (patient 3), menstrual irregularities (patients 2 and 3), and pericarditis (patient 3), these did not arouse suspicion of coeliac disease. Blood xylose, HLA typing, and antigliadin antibody investigations were always performed after a diagnosis was made and were therefore useless for this purpose. All three patients had symptoms that required upper gastrointestinal endoscopy, and the absence or appreciable reduction in duodenal folds found during the procedure was then the crucial factor leading to diagnosis. The report of these three new patients, who presented over the short period of six months, reinforces our previous statement that in all patients who undergo upper gastrointestinal endoscopy for any reason, the descending duodenum should always be explored and biopsy specimens taken in the case of loss or reduction of duodenal folds. The clinical relevance of this procedure is confirmed by the present paper, representing as it does an aid for the diagnosis of coeliac disease at an early stage. Treatment with gluten-free diet, which a recent study has shown to be protective against malignancy in coeliac disease,7 can thus be started more promptly.