Diffuse varioliform gastritis

R CAPORALI AND S LUCIANO

Divisione di Pediatra and Servizio di Radiologia, Ospedale di Bibbiena, Arezzo, Italy

SUMMARY Diffuse varioliform gastritis in a 10 year old girl is reported. The girl has been followed for four years. Biopsy specimens taken before and after three months' treatment with sodium cromoglycate showed a considerable fall in chronic inflammatory infiltrate. A rapid clinical improvement was also observed.

Diffuse varioliform gastritis is an uncommon type of chronic inflammation of the gastric mucosa, characterised by scattered erosions situated on discrete mucosal elevations, causing radiological and endoscopic ('varioliform') appearances. Immunohistochemical and clinical studies have provided evidence that this form of gastritis may have an allergic basis, or at least that type 1 hypersensitivity plays some role in its pathogenesis.1 2 This lesion of the gastric mucosa occasionally occurs after ingestion of anti-inflammatory drugs such as aspirin, indomethacin, phenylbutazone, and salazopyrin.

Case report

A 10 year old girl was referred to our department with epigastric pain, anorexia, and nausea, which had been increasing over a period of six months. Apart from evidence of recent weight loss (4 kg), there were no abnormal findings on physical examination. Antacid drugs and a three week course of cimetidine had been unsuccessful. No drugs were taken before the onset of the symptoms. The girl had been treated in hospital only once before, at the age of 4, for bronchial asthma. Her father suffered from duodenal ulceration. No other member of the family suffered from allergic disorders.

A double contrast barium examination of the stomach showed many radiolucent haloes with or without a central barium spot. The entire mucosa was involved (Fig. 1). The small intestine and colon were normal on barium examination.

At gastroscopy, erosions with a diameter of 3–5 mm surrounded by a ring elevation of mucosa (Fig. 2) were found in the antrum, in the body and the fundus of the stomach.

Histology of gastric biopsy specimens showed diffuse infiltration in the lamina propria by many lymphocytes, plasma cells, and some polymorphonuclear leucocytes. The glands showed hyperplasia without loss of specialised cells. No atypical cells were found.

Total serum protein and iron concentrations, sedimentation rate, and erythrocyte and leucocyte counts were normal. Serum eosinophil concentrations were 270×10⁶ cells/l. The serum IgG concentration was 18-2 g/l (normal 7–16-5 g/l), IgE 290 KIU/l (normal 48–120 KIU/l), IgA 1910 mg/l (normal 290–2700 mg/l), and IgM 1630 mg/l (normal 500–2600 mg/l). In the saliva the IgA concentration was 12-1 mg/l (normal 28–150 mg/l) and IgE 14-3 KIU/l. C₃ and C₄ concentrations were normal. The tests of cell mediated immunity and T and B lymphocyte populations in the blood yielded normal results, with the exception of T helper, which was slightly decreased ((OKT4) 39-7% (normal 54·5 ±6-5%).

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Correspondence to Dr A Hanukoglu, Department of Paediatrics, Edith Wolfson Hospital, PO Box 5, Holon, Israel

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Radioallergosorbent and skin prick tests to major food allergens yielded negative results. Radioallergosorbent tests to inhalant allergens were grade 1 positive to *Dermatophagoides pteronyssinus*. Skin prick tests to inhalants were positive to *D. farinae* (+++), *D. pteronyssimus* (++), *Tyrophagus putrescentiae* (+++), *Acarus siro* (+++), and *Glycyphagus domesticus* (+). Gastric basal acid output was 0.01 mEqH⁺/kg/h (normal 0.067 ±0.029 mEqH⁺/kg/h), maximum acid output 0.3 mEqH⁺/kg/h (normal 0.260 ±0.079 mEqH⁺/kg/h), and serum basal gastrin 59.82 pg/ml (normal 93 ±21.07 pg/ml). Circulating antigastric mucosal and antinuclear antibodies, immune complexes, and cytomegalovirus antibodies were absent.

Over the following four years treatment with sodium carbenoxolone was attempted several times, but no clinical improvement was observed. Serum IgE concentrations rose to 1100 KIU/l. There was no change in the diffuse chronic inflammatory infiltrate of the gastric mucosa.

Oral treatment with sodium cromoglycate 200 mg
Daily given in liquid form before meals and at bedtime was started. After three months’ treatment, biopsy specimens obtained from the gastric body and antrum showed the disappearance of the chronic inflammatory infiltrate (Fig. 3). There was concomitant clinical improvement with improvement in appetite and weight gain. Serum eosinophil concentrations returned to normal values.

Discussion

Diffuse varioliform gastritis, although occurring uncommonly in adults (0-5% of gastroscopic findings), has never been reported in children. The studies of André et al in adults contribute to the understanding of this lesion. These authors found large numbers of plasma cells containing IgE in the gastric mucosa of the affected subjects and an increased incidence of asthma, eczema, urticaria, eosinophilia, and raised serum IgE concentrations. They suggested that excessive histamine release, which gives rise to the gastric gland hyperplasia, has a central role in the pathogenesis of the disease.

The data emerging from examination of this patient excluded the more common forms of chronic gastritis, whereas the increase in total serum IgE and eosinophil concentrations, the low concentrations of IgA in the saliva, and the positive radioallergosorbent and skin prick tests to inhalant allergens suggested a type I allergic pathogenesis. In affected adults, Rosen et al observed an increased rate not only of positive food radioallergosorbent tests but also of positive inhalant radioallergosorbent tests.

There is, therefore, the possibility that in our patient, for reasons unknown, there is an IgE response in the gastric mucosa. In our patient, as in 10 adults studied by André, we found a normal stomach acid output.

In a randomised placebo controlled, double blind trial with sodium cromoglicate carried out in affected adults, André et al have stated that oral treatment with sodium cromoglicate showed clinical and endoscopic improvement with reduction of IgE containing cells in the gastric mucosa. Treatment with cimetidine, on the contrary, showed no appreciable improvement.

The efficacy of sodium cromoglicate might therefore reside in its ability to interrupt the vicious circle of macromolecule (allergen) absorption, mediator release, and increased mucosal permeability. Our report shows that a similar treatment may also be beneficial in children.

References


Correspondence to Dr R Caporali, Divisione di Pediatria, Via Turati, Bibbiena, Arezzo, Italy.

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Twenty year review of duodenal ulcer

J S A COLLINS, J F T GLASGOW, T G TROUTON, AND R J McFARLAND

Departments of Medicine and Child Health, The Queen’s University of Belfast and The Ulster Hospital, Belfast

SUMMARY Of 31 patients (18 male and 13 female) followed up 13-29 years after diagnosis, recurrent or persistent duodenal ulcer had occurred in four. In 22 (71%) gastrointestinal symptoms persisted into adult life, although only abdominal pain was significantly more frequent than in 126 controls.

Few long term studies of childhood duodenal ulcer have been published, although it has been suggested that there is a high rate of recurrence and that gastrointestinal symptoms often persist into adult life.1 This study reviewed patients from a group reported by Robb et al2 who had an ulcer diagnosed on barium meal examination between 1961 and 1970 and compared their gastrointestinal symptoms with those of apparently healthy adult controls.

Methods

A detailed questionnaire was administered that embraced various aspects of the patient’s life, including social habits, gastrointestinal symptoms
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R Caporali and S Luciano

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