

References

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Diarrhoea caused by collagenous colitis

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SUMMARY The first case of collagenous colitis in a child with protracted watery diarrhoea and abdominal pain is reported. Small bowel investigations and the macroscopic appearances were normal, but histological examination of the colon showed collagenous colitis. Steroids temporarily relieved the diarrhoea and induced transient dissolution of the subepithelial collagen band.

Colitis in children is normally associated with bloody diarrhoea with mucus, tenesmus, and abdominal pain, and is usually caused by ulcerative colitis or Crohn's disease.¹ Colitis caused by amoeba, campylobacter, antibiotics, food allergy, irradiation, or Hirschsprung's and Behçet's diseases are all uncommon. Recently microscopic and collagenous forms of colitis have been described in adults that cause profuse watery diarrhoea but remarkably occur without blood or mucus; thickening of the basement membrane is typical of this so called, 'collagenous' form.^{2,3} As far as we are aware no case has so far been reported in children, and we now report the first one.

Case report

A boy of 5 years old was referred to our unit with a four and a half year history of recurrent watery diarrhoea and abdominal pain, each episode lasting for about a week. During each episode his bowels opened up to 10 times a day, but he had otherwise grown and developed normally. His weight and height were on the 75th percentile, and between the 25th and the 50th percentiles, respectively. Examination showed no abnormal signs. Routine tests, including complete blood count, analysis of urine, plasma biochemistry, repeated stool culture

and examination for parasites, stool chromatography for sugars, sweat chloride excretion, faecal fat excretion, lactose and sucrose tolerance tests, small bowel biopsy, and barium follow through examination, were all normal; erythrocyte sedimentation rate was 29 mm in the first hour. Dietary exclusion of dairy products, eggs, fish, chocolate, sweets, fruit other than apples, and all processed foods, was of no benefit. Colonoscopy and ileoscopy showed no macroscopic abnormalities but histological examination of the biopsy specimens showed pronounced inflammation and thickening of the basement membrane.

HISTOPATHOLOGY

The initial biopsy specimens of large bowel mucosa were fixed in 10% formalin and embedded in paraffin; sections were then taken from several levels of each block and stained with haematoxylin and eosin, periodic acid-Schiff, van Gieson's, and Congo red. The most striking abnormality was the increase in the thickening of the basement membrane between crypts. This stained pale pink with periodic acid-Schiff, and red with van Gieson's, but did not show amyloid when stained with Congo red. One of us (GK) measured the basement membrane thickness using slides stained with haematoxylin and eosin and a Zeiss calibrated eye piece graticule. The thickness was estimated 30 times on each specimen in 25 adjacent intercrypt areas, and the mean thickness was 5.5 µm (range 3.7-9) (fig 1a). In addition the lamina propria was oedematous with an increased number of plasma cells and lymphocytes, particularly in the upper part of mucosa; there were no crypt abscesses or signs of ulceration.

In order to define the range of basement membrane thickness in children with other diarrhoeal diseases we retrospectively examined 543

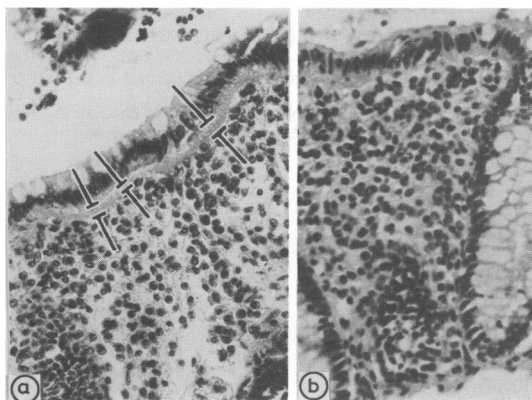


Figure (a): Rectal biopsy specimen taken before treatment showing prominent collagen band below the surface epithelium (—|—) that varies from 3.5–9.0 μm thick. Note infiltration by lymphocytes and plasma cells, and oedema of the lamina propria. (Haematoxylin and eosin $\times 160$.) (b) Rectal biopsy specimen taken in remission showing dissolution of the collagen band. Lesser inflammatory changes persist. (Haematoxylin and eosin $\times 280$.)

specimens of colonic mucosa from 303 children who had undergone colonoscopy for the following diseases: ulcerative colitis ($n=161$), Crohn's disease ($n=16$), chronic non-specific colitis ($n=80$), allergic colitis ($n=22$), campylobacter colitis ($n=11$), pseudomembranous colitis ($n=1$), Hirschsprung's colitis ($n=2$), and coeliac disease ($n=10$). Mean (SD) basement membrane thickness in the control biopsy specimens was 1.2 (0.6) μm confirming a highly significant increase in the thickness in the biopsy specimens from the patient ($p<0.01$).

RESPONSE TO TREATMENT

Sulphasalazine, cholestyramine, and loperamide each failed to control the diarrhoea, but a six week course of oral prednisolone (1 mg/kg/day) was effective in controlling his symptoms. Repeat examination of a colonic biopsy specimen eight weeks later showed that the collagen deposition was definitely reduced, although a microscopic colitis was still present (fig b). Eight months after the discontinuation of steroid treatment the patient relapsed with profuse watery diarrhoea and abdominal pain. Biopsy specimens from the colonic mucosa again showed subepithelial collagen deposition ranging from 3.0–5.0 μm thick. Steroids relieved the severity of diarrhoea, the stools became less watery, and the abdominal pain disappeared.

Discussion

Collagenous colitis has previously been described in adults as an uncommon cause of watery diarrhoea, usually accompanied by colicky pain.³ Barium enema examination, colonoscopy, and laboratory investigations of the chronic diarrhoea are all normal, and the histological hallmark is an abnormally thick band of collagen beneath the colonic epithelium; the cause of the excess collagen deposition is unknown.³ Lindstrom suggested that the thick collagen band may cause diarrhoea by interfering with water absorption,³ but perfusion studies would not support this hypothesis.⁴ No child with collagenous colitis has been described to our knowledge.

Microscopic colitis is another similar but equally uncommon cause of chronic watery diarrhoea in adults.² The diagnosis is based upon macroscopically normal colonic mucosa that shows microscopic mild, uniform, colonic mucosal inflammation.² Laboratory investigations of the chronic diarrhoea are also normal. Sanderson *et al* reported the first paediatric patient,⁵ and recently Levine *et al* suggested that microscopic colitis and collagenous colitis might be variants of the same disease.⁶ Both have the same clinical presentation, and radiographs and endoscopy of the large bowel, as well as routine laboratory tests, show no abnormalities. Indeed, the only histopathological distinction is the presence of subepithelial collagen deposition, and our observations are consistent with the suggestion that microscopic colitis might precede or follow collagenous colitis.^{2 3 6}

Our case, therefore, confirms the diagnostic value of colonic biopsy in children with diarrhoea.⁵ We also confirm that collagenous colitis does occur in children, and suggest that basement membrane thickening may parallel the clinical activity of colitis. Furthermore, though steroids may be of some symptomatic benefit, they do not seem to alter the natural history of the disease.

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Effect of fever on recurrence rate of febrile convulsions

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SUMMARY We studied 154 children admitted consecutively with their first febrile convulsion to assess the influence of the temperature on the recurrence rate of convulsions. Those with temperatures of 40°C or more were nine times less likely to have subsequent convulsions than those with temperatures of 38–38.9°C.

Factors associated with increased risk of recurrence of febrile convulsions are: the first febrile convulsion occurring before 12 months of age, a history of febrile convulsions in first degree relatives, and the presence of associated complex features of febrile convulsions.¹ Little information is available about factors associated with a reduced risk of recurrences. In a previous retrospective study with a limited number of children² we suggested that a high temperature during the initial febrile convulsions was associated with a decreased incidence of recurrent febrile convulsions. We have expanded the study by including 79 further children prospectively studied since January 1982.

Patients and methods

All children who presented to the paediatric department of this hospital with their first febrile convulsion between January 1980 and February 1986 were included in the study.

The study group comprised 173 children (91 boys and 82 girls). All except four had been born here, so their earlier records were available. Four infants were of low birth weight but had made normal neonatal progress, and the remainder were born at full term with birth weights within the normal range; they had also made uncomplicated postnatal progress.

A febrile convulsion was defined as a generalised convulsion associated with a temperature of more

than 38°C that occurred in a child aged 6 months to 5 years who had no pre-existing evidence of neurological abnormalities. All the children were managed consistently according to a routine protocol aimed at reducing body temperature. Paracetamol (10 mg/kg, every four hours) was prescribed for temperatures of over 38.5°C. The rectal temperature was recorded on admission and then every four hours. Routine investigations included a full blood count and cultures from throat, urine, and stool. Blood culture, chest radiograph, and lumbar puncture were not performed unless there were clinical indications. Antibiotics were used if there was evidence suggesting bacterial infection.

The children were divided into three groups according to the severity of the fever recorded on presentation to hospital. Group 1 had temperatures >40°C, group 2, 39–39.9°C, and group 3, 38–38.9°C.

Fifteen children were lost to follow up. Four children subsequently developed afebrile convulsions and were withdrawn from the study. The remaining 154 children were reviewed at three monthly intervals for a mean of 40 months (range 27 to 96).

Results

The groups were comparable with regard to age, sex, and family history of febrile convulsions.

Bacterial infection occurred in 24 patients (16%), 12 in group 1, 10 in group 2, and two in group 3. These infections were tonsillitis (n=2), shigella enteritis (n=4), salmonella enteritis (n=6), otitis media (n=8), pneumonia (n=2), and urinary tract infections (n=2). Antibiotics were not used to treat salmonella and shigella enteritis.

The recurrence rates of febrile convulsions are shown in the table. None of the 11 children who had their initial febrile convulsions after the age of 30 months had a recurrence. The average number of