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(Accepted 31 May 1985)

Department of Pediatrics, Tel Aviv Medical Center, Rokah Hospital, Tel Aviv, Israel

I MELAMED, MD, paediatrician
Y BUJANOVER, MD, consultant gastroenterologist
Z SPIRER, MD, professor and head of department

Microbiology Laboratory, Sackler Faculty of Medicine, Tel Aviv University, Israel

D SCHWARTZ, MSC, microbiologist
N CONFORTY, MSC, head of laboratory

Correspondence to: Professor Spirer.

Campylobacter colitis with intestinal aphthous ulceration mimicking obstruction

We report what we believe to be the first case of aphthous ulceration of the intestine in a child with colitis due to campylobacter. This child had the characteristic mucosal lesions of herpetic gingivostomatitis,

X ray film showing numerous aphthous ulcers (arrowed) present in colon in campylobacter colitis.

which also have not been recorded before with this particular bowel infection. The stools did not contain blood or mucus, and the initial tentative diagnosis was of bowel obstruction.

Case report

A 7 year old boy presented with a five day history of intermittent periumbilical pain and was referred to us because of suspected obstruction. Two other children attending his school and using the same dining room had acute enteritis. Within this cluster one child was eventually found to have campylobacter in his stool. Our patient had severe vomiting and became more

than 10% dehydrated. A swinging fever with a peak at 40°C was observed. The stools were loose but at no time contained blood or mucus; they were characterised by being malodorous. In addition he had glossitis and gingivitis. He did not have ocular or genital signs of inflammation to suggest Behçet's disease.

When he was admitted to hospital his diarrhoea, which had been a prominent feature, resolved and a stool could not readily be obtained for culture. To avoid a delay in establishing the diagnosis, however, a rectal swab was cultured and produced a heavy growth of campylobacter. Other pathogens known to be associated with colitis, such as amoebas, salmonella, shigella, and yersinia, were not recovered.

The results of investigations were: haemoglobin concentration 131 g/l; white cell count $8.2 \times 10^9/l$; erythrocyte sedimentation rate 5 mm in the first hour; negative results of a Widal test; amylase activity 195 U/l (normal 70-300 U/l); sodium concentration 145 mmol(mEq)/l; results of amoebic complement fixation test 1/16, of Paul-Bunnell (Monospot) test negative, of cytomegalovirus complement fixation test <1 in 2, and of toxoplasma fluorescence antibody test negative; total protein concentration 61 g/l (normal 60-80 g/l); albumin concentration 49 g/l (normal 32-50 g/l); gamma-globulin concentration 20 g/l (normal 20-40 g/l); and Coxsackie B virus titres not significant. A nose swab yielded *Haemophilus influenzae* and *Staphylococcus aureus*, and a throat swab yielded normal flora. No virus was seen on electron microscopy of stool, and a midstream specimen of urine yielded no cells or growth. A radiograph of the abdomen showed distended bowel with several fluid levels in the colon. Because the pain, vomiting, and fever persisted a double contrast barium enema was performed (figure), and this showed extensive fine aphthous ulceration of the large bowel. We were not given permission to carry out sigmoidoscopy and rectal biopsy or small bowel radiological and histological studies to exclude Crohn's disease (discrete (or aphthoid) ulcers are a characteristic and common finding in Crohn's disease of the colon).¹

He responded rapidly and completely to erythromycin 20 mg/kg/day. As the colitis resolved clinically so did the oral problem.

Comment

Aphthous ulceration of the colon in childhood has not been reported before, although campylobacter colitis has been described.² Moreover, glossitis and gingivitis have not previously been noted in campylobacter colitis. Ellis *et al*, however, reported a mouth ulcer in an adult with the disease.³ Our patient showed that campylobacter colitis can present with some features suggesting bowel obstruction rather than colitis, and in such cases there is a risk of unnecessary laparotomy, particularly if a patient is referred directly to a surgeon. Although taking a rectal swab is often impugned as a diagnostic method,⁴ it is worth while in avoiding delay in establishing the diagnosis in the rare cases in which a stool cannot be obtained.

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(Accepted 13 May 1985)

Hammersmith Hospital, London W12 0HS

D BENTLEY, MSC, MRCP, honorary consultant paediatrician
J LYNN, MS, FRCS, consultant endocrine surgeon

King's College Hospital, London SE5 9RS

J W LAWS, FRCP, FRCP, director of radiology

Correspondence to: Dr Bentley.

Topical rubefacient ointment: studies on haemostasis

Rubefacient ointments are widely used for the treatment of musculo-skeletal pain, often with concurrent oral analgesia. These compounds generally consist of salicylate esters, heparinoids, and nicotinic acid esters, a combination designed to enhance the anti-inflammatory and analgesic effects of the individual substances.¹

Systemic absorption of any of the active ingredients may disturb the haemostatic mechanism,²⁻⁴ and the concurrent use of aspirin would aggravate any effect. Animal studies show systemic effects and detect-