Campylobacter pylori gastritis

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SUMMARY *Campylobacter pylori* colonisation of the stomach is strongly associated with type B non-autoimmune gastritis in adults. In a retrospective study of 38 gastric biopsy specimens taken during upper gastrointestinal endoscopy in children attending this hospital we found *C pylori* in nine (24%). Ten biopsy specimens showed histological evidence of gastritis and *C pylori* was found in eight.

In 1983 Warren and Marshall described the presence of numerous S-shaped spiral bacteria on the antral epithelium of patients with chronic gastritis, and they successfully cultured the organism now called *Campylobacter pylori*.[1] Several reports since then have confirmed that gastric colonisation by *C pylori* is strongly associated with non-autoimmune type B gastritis and peptic ulcer in adults.[2] The histological recognition of *C pylori* by its characteristic curved shape in gastric biopsy specimens correlates well with bacteriological and serological diagnosis,[3] and allows retrospective diagnosis of *C pylori* colonisation.

We performed a retrospective study to determine the prevalance of *C pylori* in gastric biopsy specimens from children endoscoped for upper gastrointestinal symptoms, and we related the presence of clinical features to gastric histology.

Patients and methods

Between January 1981 and February 1987, 111 upper gastrointestinal endoscopies were performed. Suitable biopsy material was available from 38 (34%) patients. The age range of these patients was 1–16 (median 11 years), and there were 20 boys and 18 girls.

The patients' notes were reviewed for details of presentation and follow up. The histological slides were reviewed for presence and character of gastric inflammation and were stained for *C pylori* using a modified Giemsa stain (figure).[4]

Results

C pylori colonisation was detected on histological examination in nine of the 38 cases (24%), six of whom were boys. All patients were 10 years old or over. All 21 patients with histologically normal gastric mucosa were negative for *C pylori*. Ten patients showed histological evidence of chronic gastritis; eight were positive for *C pylori*. In those there was a typical diffuse chronic inflammatory cell infiltration of the lamina propria, two of these showed neutrophils within the glandular epithelium ('active' chronic gastritis) and in four there were mucosal lymphoid follicles. The histology of the two cases negative for *C pylori* but who had gastritis was different: one showed 'lymphocytic gastritis' and the other showed bile reflux gastritis. In seven patients the histology was normal apart from small focal aggregates of lymphocytes, often around a gland; only one of these showed *C pylori* colonisation. The clinical features of the study population are shown in the table. Duration of symptoms before endoscopy ranged from two months to 10 years (median 12 months). Epigastric pain was a distinctive feature of the patients who were positive for *C pylori*. It was present in seven of the nine patients; the presentation in the other two patients was anaemia in one

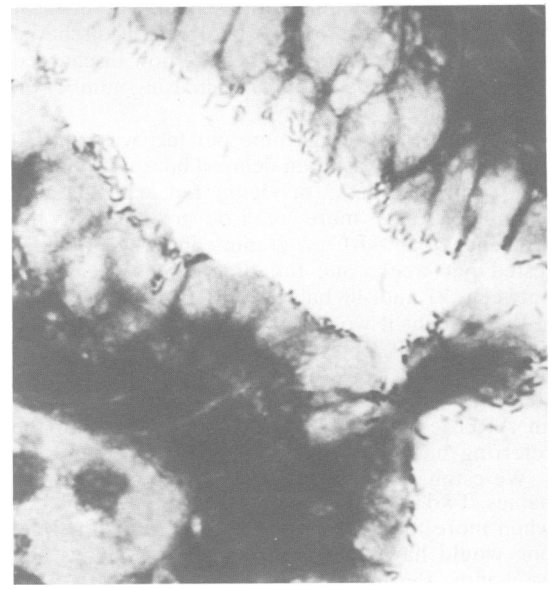

Figure *Antral mucosa from a case of antral gastritis showing colonisation by C pylori (modified Giemsa stain, original magnification × 300).*

Table *Clinical features of study population*

	Children positive for C pylori	*Children negative for C pylori*
Epigastric pain	7	3
Periumbilical pain	—	13
Vomiting	5	6
Anaemia	1	1
Gastrooesophageal reflux	—	5
Dysphagia	—	1
Duodenal ulcer	2	—
Family history of peptic ulcer	3	—

The duration of symptoms before endoscopy ranged from two months to 10 years (median 12 months).

and vomiting in the other. Three patients with no *C pylori* but who had epigastric pain had diagnoses of bile reflux gastritis, lymphocytic gastritis, and diverticulum of the duodenum, respectively. In contrast, periumbilical pain typical of recurrent abdominal pain of childhood was found only in the patients who were negative for *C pylori*. Vomiting often accompanied epigastric pain in the group who were positive for *C pylori*, but it was also found in the group without *C pylori*. Two patients had radiologically and endoscopically proved duodenal ulcer, and three patients (including the two with duodenal ulcer) had a family history of duodenal ulcer. Follow up information was available in seven of the nine patients who were positive for *C pylori* in a period ranging from one to seven years after biopsy. Four of these patients have remained symptomatic including the two with duodenal ulcer who have been treated with H_2 receptor blockers. Two became asymptomatic, and the seventh patient later developed a medullablastoma that has overshadowed her abdominal symptoms.

Discussion

Recurrent abdominal pain is common in childhood and the studies of the late John Apley show that an organic cause is found in only a few cases.[5] Chronic non-specific abdominal pain of childhood is typically central and periumbilical in location.[5] Epigastric pain is less common and usually leads to investigation for an organic cause. *C pylori* associated antral gastritis should be considered in those patients with epigastric symptoms. The diagnosis requires an antral biopsy specimen to be taken for histology or microbiology, or both, as the endoscopy appearance may be normal, although a micronodular appearance of the antrum is characteristic if found. Duodenal ulcer will be found in association with antral gastritis in a proportion of cases.

The role of *C pylori* in gastritis and the progression to peptic ulcer remains to be established. *C pylori* colonisation of the antrum is associated with active gastritis and active gastritis is associated with duodenal ulceration.[6] The emergence of *C pylori* represents another identifiable cause for recurrent abdominal pain in childhood and studies including follow up in childhood may help to elucidate the natural history of peptic ulcer disease.

References

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