The results accord with the suggestion that *C pylori* is able to neutralise bactericidal gastric acid very rapidly by buffering H^+ ions. The rapid alkalinisation of the microenvironment around *C pylori* seems likely to be mediated by the great amount of active urease available in the outer membrane of the bacterium. Thus urease of *C pylori* is a highly active enzyme that may be associated with virulence by different means: first, the urease activity protects the bacterium from the gastric acid; and secondly, the generated ammonia may then cause cytopathic effects like cell damage and possibly inflammation.[5]

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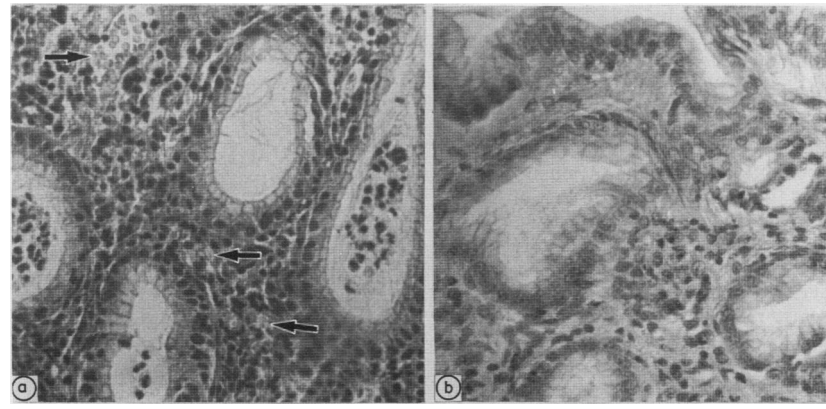

Figure *(a) Antral mucosa before treatment. Presence of a dense, diffuse inflammatory infiltrate, mainly composed of polymorphonuclear leucocytes in the lamina propria and inside glands. Congested vessels are present (arrows). (b) Antral mucosa after treatment. Presence of a few mononuclear cells in the lamina propria. (Haematoxylin and eosin.)*

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Campylobacter pylori associated acute gastritis in a child

The frequent association between *Campylobacter pylori* and chronic gastritis is well documented.[1] Marshal *et al* showed that this organism can cause acute gastritis in volunteers.[2] Marshal *et al* suggested that chronic gastritis associated with *C pylori* is preceded by an acute phase which is usually undiagnosed.[2] We could find only one such case.[3] In this case the micro-organism was not cultured and it was only shown by histological techniques. We report a case of *C pylori*-associated acute gastritis occurring in a child, in whom the diagnosis was confirmed by histological and microbiological methods.

An 8 year old boy was admitted to hospital with a three week history of epigastric pain and vomiting. The child was malnourished and there was no history of drug intake. A gastroduodenoscopy showed multiple erosions surrounded by red and friable mucosa in the antrum and body of the stomach. Biopsy specimens were taken from duodenum, stomach (fundus, body, and antrum), and oesophagus for histological assessment, immunoperoxidase staining,[4] and bacteriology.[5]

Histologically, the antral mucosa showed oedema, hyperaemia, and an intense polymorphonuclear neutrophyl leucocyte infiltration in the lamina propria, mucus layer, and inside the glands (fig 1a). The oxyntic mucosa showed mononuclear inflammatory cells in the lamina propria. Immunoperoxidase staining showed *C pylori* as dark brown, curved, or spiralled bodies within the gastric mucus, adherent to the epithelial cell surface and inside the antral glands. Brush smears of antral gastric mucosa showed curved and spiral Gram negative micro-organisms. Preformed urease tests were positive in biopsy specimens from fundus, body and antrum, and negative from oesophagus and duodenum. Culture of the biopsy specimens from gastric fundus, body and antrum showed a heavy growth of *C pylori*. The micro-organism had in vitro sensitivity to amoxycillin. No growth was observed in culture of specimens from oesophagus and duodenum. The patient was treated with oral amoxycillin (750 mg/day as three equal portions every eight hours, for one month). Within one week of treatment the symptoms disappeared. Biopsy specimens after treatment showed antral mild residual gastritis (fig 1b). *C pylori* was not shown by immunoperoxidase technique, Gram stain, preformed urease test, or by culture.

The patient presented a clinical, endoscopic, and histological picture of acute gastritis that was associated with *C pylori*. We believe that *C pylori* might be a more common causative agent of acute gastritis than previously thought.

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