Letters to the Editor

Campylobacter-like organisms in Meckel's diverticulum?

Campylobacter pylori is known to have a special affinity for antral mucosa and has increasingly become incriminated in the pathogenesis of type B gastritis and peptic ulcer. The reason for the affinity for antral mucosa is at present not known: the presence of certain growth factors and nutrients has been implicated.

Meckel's diverticula can contain several gastrointestinal mucosa types, among which is antral mucosa. Foci of antral mucosa in Meckel's diverticula showing signs of inflammation and ulceration may occasionally be seen. Might *C pylori* be found in such conditions in normal antral mucosa? There are conflicting data about the occurrence of Campylobacter-like micro-organisms in Meckel's diverticula.[1-3]

To investigate this question we retrieved 36 cases of Meckel's diverticula from our files. All diverticula were removed over 17 years in our hospital. Presence of gastric epithelium was noted and signs of inflammation were scored according to Whitehead (grades 0 to 1, normal histological features; grade 2, increase of mononuclear cells and polymorphonuclear cells present; grade 3, increase of mononuclear and polymorphonuclear cells with intraepithelial invasion of polymorphonuclear cells).

All material was recut and stained according to the modified Giemsa stain for the histological detection of Campylobacter-like organisms. Six diverticula contained both antral and body type mucosa; only one showed antral type mucosa. Four diverticula had signs of gastritis (grade 2 (n = 3); grade 3 (n = 1), which in all cases was confined to the gastric mucosa. No case of generalised diverticulitis was found. Ulceration was seen in one diverticulum. In none of the four cases of gastritis, nor in the three cases of normal gastric mucosa were Campylobacter-like micro-organisms observed. In a recent report Campylobacter-like organisms were reported in four out of 13 Meckel's diverticuli containing heterotopic gastric mucosa.[3] Unfortunately, no data were provided as to the age of the patients under study. In our study those patients with heterotopic gastric mucosa were significantly younger (mean age 15 years, SD 17) than those without gastric mucosa (mean age 35 (24) years). It is tempting to speculate that this circumstance may explain the discrepancy between these data as it is known that the occurrence of *C pylori* significantly increases with age.

This observation does not support the suggestion that *C pylori* is a major factor in the genesis of inflammation of heterotopic gastric mucosa of Meckel's diverticula.

RJLF LOFFELD*
BCAJ LOFFELD*
JW ARENDS
*Departments of *Internal Medicine and Pathology, University Hospital Maastricht, PO Box 1918, 6201BX Maastricht, The Netherlands*

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Duodenal gastric heterotopia and *Campylobacter pylori*: an exception to the rule?

Campylobacter pylori infection in gastric antral mucosa is strongly associated with the presence of type B gastritis and peptic ulceration. Evidence is accumulating that its presence may be a key factor in the tendency of duodenal ulcers to relapse.[1]

The organism is found only in gastric type epithelium and its relevance to duodenal ulceration has been explained by the common finding of gastric metaplasia within the duodenal bulb in this condition.[2] Gastric epithelium can also be found in the duodenum in another group of patients who have gastric heterotopia at this site, believed to be of developmental origin.

We retrospectively examined duodenal biopsy specimens using haematoxylin and eosin and modified silver stains to look for *C pylori* in 20 heterotopic gastric mucosa in the duodenum, and we failed to find the organism in any case including the three cases in which duodenitis was present in the adjacent mucosa. From the known age related prevalence of the organism in our area[3] we would have expected six or seven of these patients to harbour it in their gastric antrum. The standardised incidence ratio[4] was thus zero with 95% confidence limits 0 to 0·55, indicating that our finding was unlikely to be due to chance. The organism was also apparently not found in four cases of gastric heterotopia mentioned incidentally in the report of Wyatt *et al.*[2]

The possibility arises, therefore, that heterotopic gastric mucosa within the duodenum is relatively resistant to infection with *C pylori*, though why this should be so in unknown. If correct, it may explain why such patients are not known to be at particular risk of duodenal ulcer disease or are even relatively immune to its development.

In view of our findings we were interested to read that in four out of 13 patients gastric mucosa in Meckel's diverticulum was colonised with *C pylori*.[6] If it has a role in peptic ulceration then drug treatment to eradicate the infection, such as colloidal bismuth combined with an antibiotic,[1] might provide an alternative to surgery in the prevention of bleeding in patients with Meckel's diverticulum. Not all the patients in this series with ulceration in the heterotopic mucosa, however, were apparently infected with *C pylori*.

We are in the process of prospectively examining patients with duodenal gastric heterotopia to see if a proportion do, indeed, harbour *C pylori* in their gastric antra without the gastric epithelium in the duodenum being affected.

NY HABOUBI
GE WILSON
DF MARTIN
EB FARAGHER
C GODDARD
JP MILLER
Department of Histopathology, University Hospital of South Manchester, Nell Lane, West Didsbury, Manchester M20 8LR

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