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.

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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.

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Serum angiotensin converting enzyme: a possible marker in Lennert's lymphoma?

Data on raised activity of serum angiotensin converting enzyme (SACE) in patients with lymphoma are rare. High SACE activities were reported in Lennert's lymphoma,[1 2] and we report on two cases of Lennert's lymphoma in which SACE was monitored during the course of the disease and in which SACE reflected the activity of the tumour.

A 24 year old man was admitted with sustained fever, malleolaroedema, and generalised weakness. Physical examination showed subicteric sclerae, hepatosplenomegaly, and enlarged peripheral lymph nodes; pancytopenia was noted. A computed tomogram of the abdomen showed enlarged lymph nodes around the aorta, the vena cava, and the arteriae iliacae. Bone and lymph node biopsy specimens confirmed the diagnosis of Lennert's lymphoma.

The activity of SACE was 863 U/l (normal values 115–491 U/l).[3] Conventional chemotherapy was started; the patient went to partial remission after one course; SACE dropped to 287 U/l. Because of long standing leucopenia, treatment was changed to a less aggressive chemotherapeutic combination. Increased hepatosplenomegaly, fever without evidence of infection, and pancytopenia recurred. The SACE activity rose to 689 U/l, and the patient remained resistant to further chemotherapy.

A 21 year old man presented initially with renal colic, which an intravenous pyelogram showed to be due to an excluded right kidney. Because of coexisting splenomegaly, a computed tomography scan of the abdomen was performed and an extensive tumoral mass was found. The histological appearances of an abdominal lymph node and the spleen were first thought to be diagnostic of Hodgkin's disease. The patient developed a mild hypercalcemia. Combination chemotherapy with two courses of MOPP (mustine-oncovin-prednisone and procarbazine) and three of ABVD (adriamycin-bleomycin-vincristine-dacoubazine) was begun. After an interruption of three months an inguinal lymph node relapsed and an additional course of MOPP and ABVD was administrated.

Because of anorexia, vomiting, fever, icterus, hypercalcaemia and anaemia, the patient was referred to our hospital one and a half months later. Progression of abdominal lymphoma was noted on computed tomography and ultrasonography. Liver and bone biopsy specimens showed reactional histiocytosis and a haemophagocytic syndrome in association with malignant lymphoma, mostly closely resembling a T cell type. Lennert's lymphoma was diagnosed. SACE activity was 1004 U/l. The patient received one further course of chemotherapy. After some complications, the patient died of cardiac arrest. The SACE activity was 423 U/l.

Lennert's lymphoma is a haematological malignancy characterised by the presence of a high content of epitheloid histocytes, T cells, and rare Reed-Sternberg-like cells. In a study of sarcoidosis, Gaucher's disease, and other granulomatous disorders Lieberman *et al* found that three out of 11 patients with Lennert's lymphoma had raised SACE activities.

Reported cases of histiocytic medullary reticulosis have been associated with increased SACE activity. Grönhagen-Riska *et al* assumed that the increase in SACE activity reflected the monocytic line having reached the macrophage or histiocytic stage,[4] Deremee *et al* reported two patients with non-Hodgkin's lymphoma who showed increased SACE activities.[5] These authors suggested a direct role for SACE in the metabolism of vitamin D.

Lennert's lymphoma is possibly associated with an increase in SACE activity due to epitheloid proliferation. This can be helpful in differentiating Lennert's lymphoma from other malignancies. Tumour activity can also be evaluated by this marker during and after treatment.

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Infiltrative myeloid metaplasia: an unusual cause of gastric outlet obstruction

Myeloid metaplasia, histologically characterised by the simultaneous presence of all three bone marrow elements, is frequently found in myeloproliferative disorders. It is usually asymptomatic but may, on rare occasions, behave as a space occupying lesion or an aggressive infiltrate. In a population of 110 patients with myelofibrosis, aggressive infiltrates of myeloid metaplasia were found in six patients.[1] Infiltrative myeloid metaplasia has been reported in kidneys, ureters, breasts, small bowel, lungs, liver and spleen. We describe the first reported case (as far as we are aware) of gastric outlet obstruction caused by infiltrative myeloid metaplasia affecting the prepyloric region of the stomach.

In 1978 a 58 year old man was found to have essential thrombocythaemia (platelet count 790 × 10^9/l). His platelet count was successfully controlled by treatment. By 1980 he had become anaemic (haemoglobin concentration 10·9 g/dl) with a normal platelet and white cell count. His peripheral blood film showed features suggestive of myelofibrosis.

By 1982 acute myeloblastic leukaemia had supervened (peripheral white cell count 24·6 × 10^9/l with 27% blasts). With chemotherapy there was some improvement in his blood picture but he now developed intractable vomiting unrelated to chemotherapy and associated with abdominal distension. A barium meal showed no gastric