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

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Campylobacter-like organisms and
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Serum angiotensin converting enzyme: a pos-
sible 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, malleolaloradeema, and
generalised weakness. Physical examination
showed subicetec 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 artery ileace. Bone and lymph node biopsy specimens confirmed the diagnosis of Lennert’s lymphoma.

The activity of SACE was 563 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 with
out 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 hypercalcaemia.

Combination chemotherapy with two
courses of MOPP (mustine-oncovin-
prodrisone) and procarbazine) and three of
ABVD (adriamycin-bleomycin-vincristine-
dacouzabine) was begun. After an inter-
ruption of three months an inguinal lymph
node relapsed and an additional course of
MOPP and ABVD was administered.

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
mography 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 diag-
nosed. 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 422 U/l.

Lennert's lymphoma is a haematological
malignancy characterised by the presence
of a high content of epitheliod 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
Dereme 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
epitheliod 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 charac-
terised 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 popula-
tion 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 infil-
trative myeloid metaplasia affecting the pre-
pyloric 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 chemother-
apy and associated with abdominal disten-
tion. A barium meal showed no gastric

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