MATTERS ARISING

New spiral bacterium in the gastric mucosa: Gastrospirillum hominis

We read with interest the paper by McNulty et al1 and note that they considered Gastrospirillum hominis to be responsible for the symptoms of only one of their five patients. We have seen two patients in whom we believe Gastrospirillum was a clinically important pathogen.

Case 1
A previously fit woman of 56 presented in March 1987 with a four month history of nausea, malaise, abdominal pain and weight loss. She did not smoke and drank 20 units of alcohol a week. Examination and routine blood tests gave normal results but endoscopy showed widespread submucosal haemorrhages and thickened yellow mucosal folds. Histological examination showed an active gastritis which was associated with many long tightly coiled spiral organisms. The patient's symptoms resolved spontaneously over six weeks and she refused further endoscopy.

Case 2
In February 1989 a 40 year old man presented with a six year history of recurrent burning epigastric pain and nausea. There were no abnormal signs and routine blood tests provided normal results, but endoscopy showed a mild antral gastritis. Histological examination showed a mild chronic gastritis with many long coiled spiral organisms adjacent to the mucosa. His symptoms worsened and he was treated with bismuth subcitrate (Denol) 1 g only for four weeks. After this he improved and both repeat endoscopy and subsequent histology were normal, with no gastric spiral organisms.

Both patients had a gastritis compatible with infection by Gastrospirillum hominis and bismuth subcitrate may possibly be an effective treatment. Clarification of this problem must await successful methods of culture.

R H LOGAN
K P POLSON
J H BARON
M M WALKER

Results of patients and control subjects

<table>
<thead>
<tr>
<th>Mean (SD) values</th>
<th>Patient</th>
<th>Control</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>IgE (U/ml)</td>
<td>132.36 (9.39)</td>
<td>42.94 (4.69)</td>
<td>p &lt; 0.01</td>
</tr>
<tr>
<td>Total eosinophil counts (mm&lt;sup&gt;3&lt;/sup&gt;)</td>
<td>2.56 (0.2)</td>
<td>2.67 (0.3)</td>
<td>p &lt; 0.05</td>
</tr>
<tr>
<td>Eosinophils in peripheral blood film</td>
<td>8.77 (0.89)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The diagnosis of Behçet’s disease was made according to the diagnostic criteria established by the Research Committee on Behçet’s disease.1 The patients studied had no evidence of a personal or family history of atopy. The stools of the patients and healthy subjects were examined microscopically three times over six months. Patients receiving treatment who had parasites in their stools were excluded from the study. All the patients were given a complete physical, ophthalmic, and dermatological examination in addition to the routine laboratory tests. Total number of eosinophils in the peripheral blood and peripheral blood film were counted. Serum IgE concentrations were measured with a radioimmunosorbent assay, using a slight modification of the Ceska and Link method.2

The table shows the total number of eosinophils in the peripheral blood; the peripheral blood film did not show any significant difference between patients with Behçet’s disease and controls (p > 0.05). Serum IgE concentrations were significantly higher in the peripheral blood film in patients with Behçet’s disease (p < 0.01). Duration of the disease varied from four to 18 years and there was a positive correlation between serum IgE concentrations and the duration of the disease (r = 0.85 ± 0.26; p < 0.05).

It has been reported that, thrombosis in the great veins and arteries can occur at any stage of Behcet’s disease.3 IgE mediated antigen response, probably by action on mast cells or basophils, can induce platelet activation and this activation results in platelet aggregates and perhaps arterial smooth muscle hyperplasia. Such evidence may suggest a reasonable biological pathway linking increased serum IgE concentrations and Behcet’s disease.

K CENGIZ
Ondokuz Mayis University School of Medicine, Department of Internal Medicine, Samsun, Turkey

2 Lehnert T, Batchelor JR. Classification and an immunogenic basis of Behçet's syndrome. In: Lehnert T, Barnes CG, eds. Behçet's syn-
4 Ceska M, Lundvist UA. A new and simple radioimmunoassay method for the deter-
mination of IgE. Immunology 1972; 9:1021-30.
5 Stathakis NE, Economopoulos TC, Papayannis AG, Thomopoulos D. Platelet function, blood coagulation and fibrinolysis in Behçet's syn-

Dr McNulty comments:
I do not agree with the conclusions drawn by Dr Logan and his colleagues from their two case histories of “Gastrospirillum hominis” in patients attending endoscopy for the investiga-
tion of abdominal pain and nausea. They suggest that Gastrospirillum hominis was clinically important in these patients—by this I assume they meant that the organism was responsible for their presenting symptoms. The case histories do not bear this out. The symptoms of case 1 resolved spontaneously over six weeks: we do not know whether the organism was present when the woman was asymomatic, but it is likely that they were as we and others (Heilmann KL, Borchart F. Gastric spiral bacteria. Second International symposium on Campylobacter pylori, Bad Nauheim, August 1989, to be published) have shown that the infection is chronic. The second patient’s symptoms improved with bismuth subcitrate in parallel with his gas-
tritis, suggesting that the organism, like C


Departments of Gastroenterology and Histology, St Mary's Hospital, London W2