LETTERS TO THE EDITOR

Inflammation at the neo squamo-columnar junction in Barrett's oesophagus

EDITOR,—In the recent article entitled “Inflammation at the gastro-oesophageal junction (carditis) in patients with symptomatic gastro-oesophageal reflux disease: a prospective study” (Gut 1999;48:484–488), the authors determined that mucosal injury at the gastric cardia is highly localized to the region adjacent to the squamo-columnar junction in patients with gastro-oesophageal reflux disease (GORD). This is of particular interest to us in view of our recent work on the inflammatory response in Barrett’s oesophagus. We have shown that while the Barrett’s segment may be relatively devoid of inflammation, the neo squamo-columnar junction continues to excite an inflammatory reaction. These results were independent of patient medication (n=50, p<0.05), similar to the study by Lembo et al.

Lembo et al suggest that carditis may be due to “wear and tear” at the gastro-oesophageal junction as well as secondary to gastro-oesophageal reflux and Helicobacter pylori infection. Our similar findings in patients with Barrett’s oesophagus suggest that the gastric and intestinal types of epithelium (either in normal stomach or in metaplastic oesophagus) may represent an adaptation to frequent exposure to reflux. In contrast, the squamo-columnar junction is particularly susceptible to inflammation. It is interesting to speculate whether it is the potential squamous oesophagus or the distal oesophagus that are more susceptible to these epithelial reactions. In the study by Lembo et al, the biopsies containing squamous mucosa alone were not particularly inflamed; this suggests that it may be the interaction of cytokines generated from both the columnar and squamous epithelium in close proximity which are necessary to generate an inflammatory reaction. This may have implications for the strictures which occur in proximal Barrett’s oesophagus.

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Mother to child transmission of anti-S cerevisiae mannan antibodies (ASCA) in non-IBD families

EDITOR,—We enjoyed the recent study by Sutton and colleagues (Gut 2000;46:58–63) which confirmed our previous findings that elevated anti-S cerevisiae mannan antibodies (ASCA), are a familial trait in Crohn’s disease.1 The lack of concordance in marital pairs indicated that familiarity may be due to either a genetic factor or childhood environmental exposure. ASCA are present in 50–60% of patients with Crohn’s disease, in 10–15% of patients with ulcerative colitis, and in 2–5% of control subjects.2 To gain more information on concordance for ASCA among family members, we studied ASCA distribution in non-inflammatory bowel disease (IBD) families.

A total of 413 serum samples were collected from 94 diabetic families (table 1). One patient per family had type I diabetes. ASCA were detected by ELISA as previously described.3 Distribution of ASCA is given in fig 1 and table 1. Twenty three subjects (5.6%) were ASCA positive: six had diabetes and 14 were healthy. ASCA positive subjects were distributed within only 14 of 96 families. In seven of these families only one subject (parent) was positive for ASCA. The remaining 16 ASCA positive subjects clustered within seven families. All ASCA positive children were born of an ASCA positive mother. These results show that familiarity of ASCA occurs independently of Crohn’s disease and suggest vertical transmission of the marker from mother to child. Whether this is related to the observed higher risk of Crohn’s disease transmission from mother to child than from father to child is unknown. Further work is needed to assess if the presence of ASCA may predict an increased risk of Crohn’s disease in offspring.

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Table 1 Distribution of anti-S cerevisiae mannan antibodies (ASCA) in 94 diabetic families

<table>
<thead>
<tr>
<th>Families</th>
<th>Parents</th>
<th>Children</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total number ASCA</td>
<td>Father ASCA</td>
</tr>
<tr>
<td></td>
<td>22</td>
<td>44</td>
</tr>
<tr>
<td>1</td>
<td>30</td>
<td>60</td>
</tr>
<tr>
<td>2</td>
<td>34</td>
<td>58</td>
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<td>3</td>
<td>4</td>
<td>6</td>
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<td>4</td>
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<td>6</td>
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<td>1</td>
</tr>
<tr>
<td>7</td>
<td>12</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>94</td>
<td>188</td>
</tr>
</tbody>
</table>


Reply

EDITOR,—We were interested by the letter of Poullain and colleagues on our paper (Gut 2000;46:58–63) and their data on familiarity of this trait in non-inflammatory bowel disease (IBD) families, and evidence for mother to child transmission of anti-S cerevisiae mannan antibody (ASCA) expression. We have now re-analysed our own data set to our findings on this issue (table 1). We

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We have now re-analysed our own data set to our findings on this issue (table 1). We

observed a strong correlation for both the mother-child and father-child pairs. This differs from Poullain and colleagues who only observed vertical transmission from mother to child. The difference may relate to distinct subject populations. Poullain evaluated a non-IBD group (diabetic families) whereas we evaluated clinically unaffected relatives from families with Crohn’s disease (CD) probands. We also note that our analysis used statistical methods for quantitative data (antibody levels) which enhances statistical assessment, particularly when limited by small data sets. Poullain’s analysis compares qualitative data (seropositive or seronegative), and the small number of relevant pairs may be insufficient to support their conclusion statistically. We agree with them on the need to assess the possibility that ASCA may predict increased risk for CD in unaffected individuals.

Table 1 Correlation coefficients for IgG anti-S cerevisiae mannann antibodies (ASCA) among 33 first degree relatives without evidence of Crohn’s disease

<table>
<thead>
<tr>
<th>Type of pairs</th>
<th>IgG ASCA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mother-daughter</td>
<td>0.135</td>
</tr>
<tr>
<td>Mother-son</td>
<td>0.483</td>
</tr>
<tr>
<td>Father-daughter</td>
<td>0.423</td>
</tr>
<tr>
<td>Father-daughter</td>
<td>0.404</td>
</tr>
</tbody>
</table>

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BOOK REVIEW


Many specialist registrars embarking on a period of research are faced with the task of writing grant proposals requiring a comprehensive understanding of a subject, but often begin from a point of relative ignorance. The first port of call for many will be to delve into MEDLINE, but bibliographic databases organised in a similar format, sparing the reader from many hours of fruitless searching. The opening chapter provides a brief historical introduction as to how MEDLINE was conceived and the way in which data are indexed, a key to understanding the most efficient way of extracting relevant information.

The novice user of MEDLINE will in most cases begin searching using text or keywords, often generating a list of references that is simply too long to read. Chapter two takes the reader through the different fields or indexes that can be used when searching and details how the long list of references obtained can be systematically fine tuned using combinations of these. Particular emphasis is placed on the usefulness of Medical Subject Headings (MeSH), an underlying theme of the book, as an initial means of searching, and useful hints are given to direct the reader to the MeSH headings most appropriate to the information they require. The book concludes by setting out some standard approaches to effective searching using information learned in an initial search strategy to direct subsequent avenues of enquiry. In addition it gives useful hints on the framing of questions in order to get results most appropriate to the topic of enquiry.

MEDLINE contains a vast array of information and perhaps Brian Katcher’s most useful tip is to start with books, pencil, and paper. His book is an informative read, most useful to those who are relative novices to using MEDLINE. It is presented in an easy style and without doubt will guide the reader to more “effective searching” of bibliographic databases.

J A TIBBLE

NOTES

Sir Francis Avery Jones British Society of Gastroenterology Research Award 2001

Applications are invited by the Education Committee of the British Society of Gastroenterology who will recommend to the Council the recipient of the 2001 Award. Applications (TEN COPIES) should include:

- A manuscript (2 A4 pages ONLY) describing the work conducted
- A bibliography of relevant personal publications
- An outline of the proposed content of the lecture, including title
- A written statement confirming that all or a substantial part of the work has been personally conducted in the UK or Eire.

Entrants must be 40 years old or less on 31 December 2000 but need not be a member of the Society. The recipient will be required to deliver a 30 minute lecture at the Annual meeting of the Society in Glasgow in March 2001. Applications (TWENTY COPIES) should be made to the Honorary Secretary, British Society of Gastroenterology, 3 St Andrews Place, London NW1 4LB by 1 December 2000.

British Society of Gastroenterology Hopkins Endoscopy Prize 2001

Applications are invited by the Endoscopy Committee of the British Society of Gastroenterology who will recommend to the Council the recipient of the 2001 Award. Applications (TEN COPIES) should include:

- A manuscript (2 A4 pages ONLY) describing the work conducted
- A bibliography of relevant personal publications
- An outline of the proposed content of the lecture, including title
- A written statement confirming that all or a substantial part of the work has been personally conducted in the UK or Eire.

An applicant need not be a member of the Society. The recipient will be required to deliver a 20 minute lecture at the Annual meeting of the Society in Glasgow in March 2001. Applications (TEN COPIES) should be made to the Endoscopy Section Secretary, British Society of Gastroenterology, 3 St Andrews Place, London NW1 4LB by 1 December 2000.

American College of Gastroenterology 2001 International GI Training Grants Programme

The ACG International GI Training (IGT) Grant Programme provides funding for clinical or research training in gastroenterology and hepatology so that an individual can acquire or develop new cognitive knowledge or a technical skill. This newly acquired knowledge or skill would then be used to improve patient care in the applicant’s geographic area. Physicians outside of the United States and Canada are eligible to apply. At least one fellowship with a maximum of $10 000 per IGT fellowship will be awarded during 2001, for a training period of not less than six months. Awards will be made by a special committee of the ACG and will be based upon the applicant’s credentials, the merit of the proposed training by the selected host training centre and the potential for enhancing the field of gastroenterology in the applicant’s home country. Application forms can be obtained from the ACG administrative office, 4900B South 31st Street, Arlington, Virginia 22206–1656, Tel: +1 703 820 7400; fax: +1 703 931 4520; website: www.acg.org. Deadline for submission of application is 1 April 2001.

3rd European Federation of Autonomic Societies (EFAS)

The third European Federation of Autonomic Societies (EFAS) meeting in conjunction with the annual meeting of the sections “Autonomic nervous system” of the German Neurological Society, “Diabetes and Nervous System” of the German Diabetes Association, and “Autonomic Nervous System” at the University of Erlangen-Nuremberg, Germany, will be held in Erlangen, Germany on 26–28 April 2001. Abstract deadline: 20 December 2000. Further information: Professor Dr M J Hilz, Department of Neurology, University or Erlangen-Nuremberg, Schwabachanlage 6, D-91054 Erlangen, Germany; Tel: +49 09131 8534444; fax: +49 0131 8534328; website: www.gutjnl.com
CORRECTION

The authors of Guidelines for the management of iron deficiency anaemia (Gut 2000;46(suppl IV)) would like to correct an error they made. In section 2.2, second paragraph, the wrong unit was used for ferritin. Instead of µg/dl, it should have been µg/l (or ng/ml). The authors apologise for any confusion this may have caused.