Letters to the editor

Ultrasonographic findings in Crohn's disease

Editor,—We read with interest the paper by Gasche et al (Gut 1999;44:112–117) on the accuracy of transabdominal ultrasound in the detection of complications in Crohn's disease. The authors evaluated 33 patients with Crohn's disease who had resective bowel surgery. The results were impressive: 87% sensitivity with 90% specificity in the diagnosis of enterocutaneous fistulas; 100% sensitivity with 92% specificity in the diagnosis of intra-abdominal abscesses; and 100% sensitivity with 91% specificity in the diagnosis of strictures. However, these data are in contrast with those reported by Hata and colleagues (Gut 1998;41:1645–8), who found very low sensitivity (50%) with 95.5% specificity in ultrasound detection of enterocutaneous fistulas.

The difference in levels of sensitivity in these studies could be explained by the use of different standards and also, in our opinion, by varying definitions of fistulas. Gasche and colleagues considered fistulas to be any hypoechoic peri-intestinal lesion measuring less than 2 cm. However, although this arbitrary cut off point may be useful to differentiate between fistulas and abscesses, it does not allow for precise differentiation between fistulas and strictures, for which we usually do not allow for precise differentiation. Gasche et al defined bowel wall thickening of 4 mm or more to be abnormal. Hata and colleagues reported that the mean overall wall thickness of normal bowel specimens was 2.8 mm and that no normal specimens exceeded 4 mm in thickness. More recently other studies by Solvig, Van Oostayen, and even Gasche defined bowel wall thickening of 3 mm or more as pathological.1

Previously, we considered 4 mm to be the pathological value of bowel wall thickness in patients with inflammatory bowel disease, but we have now reduced this value to 3 mm or more, having excluded patients with portal hypertension, in which bowel wall thickness is due to an oedematous mimsification. Recently, we conducted a prospective study (unpublished data) in which bowel wall thickness was shown to have a prognostic value. We found that patients with Crohn's disease with a bowel wall thickness greater than 6 mm, who are in clinical remission, showed a significantly higher relapse rate (90%) in the subsequent 18 months, compared with patients with bowel wall thickness of less than 6 mm (40%).

In conclusion, the diagnostic accuracy of transabdominal ultrasound has improved progressively as more data are found in the literature are due principally to the introduction of new technologies, the level of experience of the operators, and the growing interest in the application of ultrasound to the study of the digestive tract.

V ARIENTI
I ZAMBONI
Department of Internal Medicine,
Ospedale Maggiore G.A. Pizzardi,
40133 Bologna, Italy

P GIONCHETTI
F RIZZELLO
M CAMPIERI
Department of Internal Medicine,
Policlinico S. Orsola,
40138 Bologna, Italy


Association between colon cancer and adenocarcinoma of the oesophagus

Editor,—Recently, Lagergren and Nyren (Gut 1999;44:819–821) concluded that results from a population based cohort study in Sweden did not support a common link between colon cancer and oesophageal adenocarcinoma. However, there is consistent evidence that low intake of dietary fibre is associated with both diseases. In an analysis of 13 case control studies involving more than 5000 colorectal cancer cases, Howe and colleagues reported an inverse association between fibre intake and colorectal cancer in 12 of the 13 studies, and an odds ratio of 0.53 (95% confidence interval 0.47 to 0.61) for the highest quintile of fibre intake compared with the lowest, in a pooled analysis.1 Similarly, four case control studies have reported a significant inverse association between fibre intake and adenocarcinoma of the oesophagus and gastric cardia (table 1).2

In contrast, two studies which included cases of squamous cell carcinoma found no significant link between fibre intake and squamous cell carcinoma of the oesophagus.3,4 Clearly, the dramatic increase in the incidence of adenocarcinoma of the oesophagus in the USA and parts of Europe over past decades cannot be explained by secular trends in dietary fibre consumption. A more plausible explanation links increased rates of the disease to increases in the prevalence of obesity.5 This view is supported by evidence from observational studies that suggests that both overweight and symptomatic gastro-oesophageal reflux are linked to increased risk of oesophageal adenocarcinoma.6 Possible mechanisms for the observed protective effect of dietary fibre include the mechanical cleaning effect of the lower oesophageal mucosa, increased motility of potential carcinoma across the gastro-oesophageal junc-

Table 1 Dietary fibre intake and adenocarcinoma of the oesophagus and gastric cardia

<table>
<thead>
<tr>
<th>Reference</th>
<th>Country</th>
<th>Sites</th>
<th>Comparison</th>
<th>Odd ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 USA</td>
<td>OGC</td>
<td>Highest of lowest quintile</td>
<td>0.3</td>
<td>0.1 to 0.7</td>
<td></td>
</tr>
<tr>
<td>3 USA</td>
<td>OGC</td>
<td>Highest of lowest quintile</td>
<td>0.3</td>
<td>0.1 to 0.8</td>
<td></td>
</tr>
<tr>
<td>4 USA</td>
<td>O</td>
<td>Highest of lowest quintile</td>
<td>0.4*</td>
<td>Not stated</td>
<td></td>
</tr>
<tr>
<td>5 Greece</td>
<td>O</td>
<td>Marginal quintile</td>
<td>0.74</td>
<td>0.55 to 0.99</td>
<td></td>
</tr>
</tbody>
</table>

Odds ratios adjusted for alcohol and tobacco use.

*95% confidence interval (CI) does not include 1.0. †Multiple logistic regression model.

OGC, oesophagus and gastric cardia; O, oesophagus.


10 Epidemiology of squamous cell carcinoma found no significant link between fibre intake and squamous cell carcinoma of the oesophagus.3,4

11 Clearly, the dramatic increase in the incidence of adenocarcinoma of the oesophagus in the USA and parts of Europe over past decades cannot be explained by secular trends in dietary fibre consumption. A more plausible explanation links increased rates of the disease to increases in the prevalence of obesity.5 This view is supported by evidence from observational studies that suggests that both overweight and symptomatic gastro-oesophageal reflux are linked to increased risk of oesophageal adenocarcinoma.6
The primary hypothesis of our population based cohort study was not that colon cancer would subsequently develop into oesophageal cancer, but rather that there might be a common underlying link between the occurrence to these two tumours, independent of the time latency in their Development. Hence, the individual follow up latency after colon cancer diagnosis was of minor importance. Therefore, it would seem reasonable to analyse differences between exposure to the critical underlying factors, for example, insufficient dietary intake of fibre, and the development of oesophageal or colonic adenocarcinoma. Therefore, as long as selection or ascertainment biases are deemed to be small, the time period that follows immediately after diagnosis of colon cancer is the most informative. The total number of person years was more critical, and we were able to follow up more than 500 000 person years in our study. The rarity of oesophageal adenocarcinoma is a problem in any study of the aetiology of this tumour in any country, particularly if the studied exposure is relatively recent. This problem explains our limited power to exclude a weak association. Nevertheless, we were able to identify more than 100 000 people with verified colon cancer and to follow them for subsequent cancer. Oesophagus is a substantial number of exposed people. We agree that case control studies are generally more efficient than cohort studies when rare outcomes are to be investigated. However, in the case of our register based retrospective cohort study, a case control approach would not entail any advantage, as our cohort contained all individuals exposed to colon cancer in Sweden during the same period. A case control study conducted in Sweden during this period would, at best, include the same number of exposed oesophageal adenocarcinoma cases as in our cohort study. Thus, the problem with low statistical power is not owing to study design, but that the study base (all residents of Sweden 1958–1992) was too small for sufficient number of individuals with the combination of colon cancer and oesophageal adenocarcinoma.

J LAGERGREN
P NYREN
Division of Surgery,
Karolinska Institutet,
Danderyd Hospital,
S-182 88 Danderyd, Sweden

Replay

EDITOR,—The influence of dietary fibre on the risk of adenocarcinoma of the oesophagus and gastric cardia is certainly interesting, but further and larger studies are needed before a link between the two can be confirmed. Although several case control studies have reported a relation between colon cancer and fibre intake, others have failed to identify such a link.1 Hence, the suggested link between fibre intake and both colon cancer and oesophageal cancer is debatable.

We agree that changes in dietary fibre consumption cannot explain the increasing incidence of oesophageal adenocarcinoma. The increasing prevalence of obesity is a possible reason for this rise, but some seemingly inconsistent observations need to be conciled before this hypothesis can be verified.1 The apparently sudden deflection of the incidence curve for oesophageal adenocarcinoma,1 the rapidity of the increase,1 and the noticeable (6–8 fold) increases which do not entirely support this interpretation.


2 Kabat GC, Ng SK, Wynder EL. Tobacco, alcohol, and cigarette smoking as risk factors for oesophageal and gastric cancer. Cancer Causes Control 1993;4:123–32.


Quality of life of parents of children on home parenteral nutrition

EDITOR,—Jeppesen and colleagues (Gut 1999;44:814–852) evaluated the non-disease specific sickness impact profile (SIP) and the disease specific inflammatory bowel disease questionnaire (IBDQ) to assess the quality of life of 49 patients receiving home parenteral nutrition (HPN). They found a significant reduction in the quality of life of these patients compared with patients with anatomically or functionally short bowel not receiving HPN.

S L JOWETT
T COBDEN
Department of Gastroenterology,
North Tyneside General Hospital,
Rake Lane,
North Shields, Tyne and Wear NE29 8NH, UK
We showed recently that having a child on HPN has a major impact on the quality of life of the parents. We studied 11 parents of children with chronic intestinal failure requiring HPN. Following an initial focus group meeting to identify important issues, semi-structured interviews were held with the parents. The General Health Questionnaire (GHQ-28) and a questionnaire developed for the British Artificial Nutrition Survey (BANS) were also administered. A control group of 11 parents with age matched healthy children also answered the BANS questionnaire.

The GHQ-28 showed that seven of the 11 parents with children on HPN exceeded the threshold for psychiatric morbidity. The BANS described a significant deterioration before and after the child’s illness for social life (p<0.007), family life (p<0.007), sex life (p<0.003), and work (p<0.004) in these parents compared with controls. Parents caring for children on HPN were also more likely to be physically tired and to have difficulties in taking holidays, going shopping and spending time with their partners. Many of them admitted to feeling frustrated, annoyed, stressed, and having problems sleeping.

With the advent of HPN, increasing numbers of children with chronic intestinal failure are now being managed at home. Although HPN has given life to many of these children who would otherwise have died, the burden of care on these parents is enormous and could have a significant impact on their quality of life. Health care professionals should be aware of this problem and endeavour to offer the necessary support for families who provide this demanding type of care. The services of a dedicated community nutrition support team is recommended.

NOTES


5th World Congress on Trauma, Shock, Inflammation, and Sepsis

The 5th World Congress on Trauma, Shock, Inflammation, and Sepsis will be held in Munich, Germany, from 29 February to 4 March 2000. Further information from: Prof Eugen Faist, Department of Surgery, Ludwig Maximilians University Munich, Klinikum Grosshadern, Marchioninistrasse 15, 81377 Munich, Germany. Tel: +49 89 7095 5461/2461; fax: +49 89 7095 2460; email: faist@gc.med.uni-muenchen.de

International Hepato-Pancreato-Biliary Association 4th World Congress

The International Hepato-Pancreato-Biliary Association 4th World Congress will be held in Brisbane, Australia, from 28 May to 1 June 2000. Further information from: International Hepato-Pancreato-Biliary Association, PO Box 1280 (Intermedia House, 11/97 Castlemaine Street), Milton, Queensland 4064, Australia. Tel: +61 (07) 3369 0477; fax: +61 (07) 3369 5152; email: hpb2000@im.com.au
Association between colon cancer and adenocarcinoma of the oesophagus

R MARIC and K K CHENG

Gut 2000 46: 293
doi: 10.1136/gut.46.2.293a