LETTERS TO THE EDITOR

Cholecystectomy and bowel function

EDITOR,—I read with great interest the article by Hearing et al (1999;45:889–894) on the effect of cholecystectomy on bowel function. In this elegant publication, however, the authors mistakenly assume that published estimates of the incidence of postcholecystectomy diarrhoea derive from retrospective or uncontrolled data only. In this context I would like to draw attention to earlier publications derived from the Rotterdam Gallstone Study.

In the first paper the results are discussed of a prospective analysis of biliary and gastrointestinal symptoms (including diarrhoea) prior to and up to two years after gall stone therapy. This therapy consisted of either conventional cholecystectomy or extracorporeal shock wave lithotripsy (ESWL), allocated randomly. The second paper focused on surgery and reported on symptoms before and after conventional and laparoscopic cholecystectomy. This study was based on the same concept, and treatment depended on the availability of a laparoscopic set. Generally, we found that the reported incidence of diarrhoea before and after surgery did not change. In fact, there was no difference in the reported incidence of diarrhoea at any time between cholecystectomy and gall bladder preserving therapy (that is, ESWL). We also found that there were no differences in the reported incidence or severity of diarrhoea between laparoscopic and conventional cholecystectomy at any time.

Although the study design of our two studies differed largely from that of Hearing’s, the results and conclusions are in agreement, that clinical diarrhoea seldom develops after cholecystectomy. O’Donnell is correct that objective diarrhoea is in fact an unproved entity. Given our findings, it is likely that the increased numbers of patients with diarrhoea who are treated with antibiotic treatment is indeed highly suggestive of an underlying infectious process but does not necessarily constitute proof of this assumption, as direct antiproliferative mechanisms of various antibiotics, including clarithromycin, have been reported in different settings. Further investigations are needed before definite recommendations for (as yet empirical) antibiotic therapy in patients with extra-gastric MALT lymphomas can be given.

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MALT lymphomas and Helicobacter pylori

EDITOR,—Raderer et al (2000;46:133–5) present an interesting case report of a patient with a mucosa associated lymphoid tissue (MALT) lymphoma of the stomach and descending colon. Their report adds to the growing literature of gastrointestinal MALT lymphomas that respond to antibiotic treatment. In addition to the numerous reports on antibiotic sensitive gastric lymphomas of the small intestine, salivary glands, nasal mucosa, and colon have recently been reported.

Although Helicobacter pylori is generally implicated as the inducing agent, this does not always appear to be the case. Erdmann, has also been found in association with gastric MALT lymphomas, including H pylori negative patients whose disease was still responsive to antibiologic treatment. Furthermore, other non-H pylori bacterial and protozoal flora have been observed in gastric lymphomas specific to infected regions. In the report by Raderer et al, and in several of the cases previously mentioned, H pylori was not identified in the extragastric lesions, leaving it open to speculation how H pylori may induce antigenic stimulation of these lymphomas. Moreover, in the report by Inoue and Chiba, not only was the rectal lesion H pylori negative but upper gastrointestinal endoscopy was normal. Their patient was seronegative for H pylori and had a negative rapid urease test, culture, and histological examination.

In light of this evidence, it seems that although H pylori may be the most common cause of many gastrointestinal MALT lymphomas, it is not the only causative organism. This is an important consideration when confronted with patients diagnosed with H pylori negative MALT lymphomas.

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Guidelines for the management of iron deficiency anaemia

EDITOR,—It somewhat self contradictory to suggest that “a transferrin saturation of <30% may help the diagnosis” if there is still doubt about validation of iron deficiency after receipt of the serum ferritin result, the authors having previously acknowledged that the latter is “the most powerful test for iron deficiency” (Gut 2000;46[suppl IV]:1–5). Statistical considerations which dictate that serum ferritin will always outrank transferrin as a predictive power have their basis in the comparison between the receiver operating characteristic (ROC) curves for serum ferritin versus transferrin saturation, yielding values of 0.91 versus 0.71 (p<0.001) for the area under the curve.1 Statistical considerations also dictate acknowledgement of mean corpuscular haemoglobin (MCH) as a predictive entity in its own right following documentation that an MCH of <27 pg was superior to mean corpuscular volume (MCV) of <77 fl in predicting serum ferritin levels of <20 µg/l. All low MCV values had low MCH values but this has not been validated.

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References


In my study, comparing 201 subjects with iron deficiency (characterised by serum ferritin <18 µg/l), the MCH characterised by optimum trade-off between sensitivity (65.2%) and specificity (65.9%) for iron deficiency was <23 pg, and this yielded a positive predictive value of 70%. By contrast, for MCV, optimum trade-off between sensitivity (61.7%) and specificity (59.1%) was obtained with a cut-off level of <77 fl, giving a positive predictive value of 65%. There were 31 patients with an MCH <26 pg in the presence of an MCV >80 fl compared with only four with an MCV <80 fl in the presence of an MCH >24 pg and, among these, four had an MCH <24 pg in the presence of an MCV >77 fl in contrast with only one with an MCV <77 fl in the presence of an MCH >24 pg. In my study, the most stringent cut-off diagnostic level for iron deficiency was a serum ferritin level <10 µg/l found in a subgroup of 145 subjects. At this level, the MCH characterised by optimum trade-off between sensitivity (65%) and specificity (66%) was <76 fl (identical with the cut-off level in the guidelines), and this yielded a positive predictive value of 55%. Correspondingly, the optimum MCH was either <24 pg, characterised by sensitivities, specificities, and positive predictive values of 74%, 59%, and 80%, respectively, or <23 pg, based on sensitivities, specificities, and positive predictive values of 58%, 75%, and 62%, respectively.

Reply

Editor—Suggesting both that transferrin saturation may help in the diagnosis and that ferritin is the most powerful test for iron deficiency anaemia (IDA) is not contradictory. Being the most powerful test does not mean it is always reliable. For example, in inflammatory conditions such as rheumatoid arthritis, ferritin may be normal even if there is iron deficiency. We find the reference to the greater reliability of mean corpuscular haemoglobin (MCH) compared with mean corpuscular volume (MCV) in diagnosing IDA interesting. We agree that MCH can be useful in the diagnosis of iron deficiency. However, none of the papers quoted takes account of the red cell distribution width (RDW). We wonder if Dr Jolobe would still be able to demonstrate the superiority of MCH compared with MCV if anaemic patients with a normal MCV but raised RDW were excluded. We explain in our guidelines that combined deficiency (that is, iron deficiency together with B12 and/or folate deficiency) may be associated with a normal MCV and may be recognised by a raised RDW.

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Two abstracts in Gut 2000;47(suppl III) had incomplete author lists. The authors of A136 are L Sarli, R Costi, S Gobbi, D Iusco, L Sarli, and the authors of A138 are L Sarli, R Costi, S Gobbi, C Pavlidis, L Roncoroni, and this yielded a positive predictive value of 74%

American College of Gastroenterology 2001 International GI Training Grants Programme
The ACG International GI Training (IGT) Grant Programme provides funding for clinical or clinical 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 should 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 selection 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.gi.org Deadline for submission of application is 1 April 2001.

Cleveland Clinic Florida’s Gastroenterology Update 2001
Cleveland Clinic Florida will be sponsoring a postgraduate course entitled “Gastroenterology Update 2001” to be held on 10–11 February 2001 in Fort Lauderdale, Florida, USA. Further information: Sally Jagelman, Manager of Continuing Medical Education, Cleveland Clinic Florida, 3000 West Cypress Creek Road, Fort Lauderdale, FL 33309, USA. Tel: +1 954 978 5539; fax: +1 954 978 5056; email: jagelman@ccf.org

GI malignancies can be prevented and treated: from the bench to the bedside
This international meeting will be held on 14–17 February 2001 in Jerusalem and the Dead Sea, Israel. Further information: Dr Massimo Crespi, Tel Aviv University, Tel Aviv, Israel. Tel: +972 3 5175150; fax: +972 3 5175155; email: gi@targetconf.com

Gastroenterology and Endotherapy: XIXth European Workshop
This course, to introduce the experienced gastroenterologist to the growing field of therapeutic endoscopy, will be held on 18–20 June 2001 in Brussels, Belgium. Further information: Mrs Nancy Beauprez, Gastroenterology Department, Erasme Hospital, Route de Lennik 808; B-1070 Brussels. Tel: +32 2 055 49 00; fax: +32 2 055 49 01; email: beauprez@ulb.ac.be

Summer Abdominal Imaging Conference
A five day course designed for the practising radiologist with a primary interest in abdominal imaging, emphasising the most recent advances in helical CT, MRI, US, and gastrointestinal imaging. It will be held on 23–27 July 2001 in Banff Springs, Canadian Rockies. Twenty-five category 1 credit hours. Further information: Janice Ford, Benner, University of Pennsylvania Medical Center (Radiology), 3400 Spruce Street, 1 Silverstein Building, Philadelphia, PA 19104, USA. Tel: +1 215 662 6904; fax: +1 215 349 5925.