Cholecystectomy and bowel function

EDITOR,—I read with great interest the article by Hearing et al (Gut 1999;45:889–94) 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.7 The study 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.6 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 assessments of therapy demonstrate new onset diarrhoea after cholecystectomy.1 I agree with Hearing et al that postcholecystectomy diarrhoea is in fact an unproved entity. Given our findings should be interpreted with caution. In contrast with other cases reported in the literature and cited by the authors, our patient suffered from concurrent gastric and colonic MALT lymphoma and had evidence of H pylori infection. Thus one cannot rule out the fact that antigenic shedding of H pylori from the stomach throughout the gastrointestinal tract or the presence of specific T cells alone was able to provide the colonic lesion with an antigenic drive needed for maintenance of the lymphoma. In this scenario, one would expect eradication of H pylori to lead to regression of the (still antigen and/or T cell dependent) lymphoma. The fact that various (apparently not H pylori related) intestinal as well as gastric lesions are reported with antibiotic treatment is indeed highly suggestive of an underlying infectious process but does not necessarily constitute proof of this assumption, as direct antiinflammatory 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 extragastric MALT lymphomas can be drawn.

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Guidelines for the management of iron deficiency anaemia

EDITOR,—It is 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):iv1–5). Statistical considerations which dictate that serum ferritin will always outtrack transferrin saturation and that 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 is superior to a mean corpuscular volume (MCV) of <77 fl in predicting serum ferritin levels of <20 µg/l.2 All low MCV values had low MCH values but nine hypoferritinaemic patients with low MCH had MCV within the normal range.

Reply

EDITOR,—Hopton Cann et al again point out the fact that Helicobacter pylori is not the sole cause of mucosa associated lymphoid tissue (MALT) lymphoma in some cases. They also suggest other, as yet undefined, bacterial/infectious causes in MALT lymphomas in extragastric sites in view of our case and the recent literature. While we believe this to be a valid point and agree with the already established notion of other contributing factors in addition to H pylori, we nevertheless advise that our findings should be interpreted with caution. In contrast with other cases reported in the literature and cited by the authors, our patient suffered from concurrent gastric and colonic MALT lymphoma and had evidence of H pylori infection. Thus one cannot rule out the fact that antigenic shedding of H pylori from the stomach throughout the gastrointestinal tract or the presence of specific T cells alone was able to provide the colonic lesion with an antigenic drive needed for maintenance of the lymphoma. In this scenario, one would expect eradication of H pylori to lead to regression of the (still antigen and/or T cell dependent) lymphoma. The fact that various (apparently not H pylori related) intestinal as well as gastric lesions are reported with antibiotic treatment is indeed highly suggestive of an underlying infectious process but does not necessarily constitute proof of this assumption, as direct antiinflammatory 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 extragastric MALT lymphomas can be drawn.

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LETTERS TO THE EDITOR

MALT lymphomas and Helicobacter pylori?

EDITOR,—Raderer et al (Gut 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, those 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 and delays bacte- rium, H. heilmannii, has also been found in association with gastric MALT lymphomas, including H pylori negative patients whose disease was still responsive to antibiotic treat- ment. Furthermore, other non-H pylori bacterial1 and protozoal1,2 flora have been observed in gastric lymphomas specific to involved 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 specula- tion how H pylori may induce antigenic stimulation of these lymphomas. Moreover, in the report by Inoue and Chiba,3 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 MALT lymphoma of nasal mucosa treated with antibi- otics.

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In my own study, comprising 201 subjects with iron deficiency (characterised by serum ferritin <18 µg/l), the MCH contrasting optimum trade off between sensitivity (65.2%) and specificity (65.9%) for iron deficiency was <24 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, characterised by sensitivities, specificities, and positive predictive values of 58%, 75%, and 62%, respectively.

CORRECTION

Two abstracts in Gut 2000;47(suppl III) had incomplete author lists. The authors of A136 are L Sarli, R Costi, S Gobbi, D Isusco, D Sarli, and the authors of A138 are L Sarli, R Costi, S Gobbi, C Pavlidis, L Roncoroni, Ireland. Further information: Maddalena Massaro, Project Leader, AISC-AIM Group, Via A Ristori 38, 00187 Rome, Italy. Tel: +39 06 809681; fax: +39 06 80968229; email: gastro2001@aisc.it.

NOTES

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 can 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, the capability of the 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: ACG, 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.

Replay

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.


Reply

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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

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 Beaufreux, Gastroenterology Department, Erasme Hospital, Route de Lennik 808; B-1070 Brussels. Tel: +32 02 555 49 00; fax: +32 02 555 49 01; email: beaufreux@ulb.ac.be


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