LETTERS TO THE EDITOR

Cholecystectomy and bowel function

Editor,—I read with great interest the article by Hearing et al (Gut 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 bowel disturbance after 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.1

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. The therapy consisted of either conventional cholecystectomy or extracorporeal shock wave lithotripsy (ESWL), allocated at random. The second paper focused on surgery and reported on symptoms before and after conventional and laparoscopic cholecystectomy.2 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, in that diarrhoea before and after surgery did not differ. This is an important point to consider when comparing extracorporeal shock wave lithotripsy with conventional cholecystectomy. I agree with Hearing et al that postcholecystectomy diarrhoea is in fact an unproved entity. Given our and Hearing’s results, I doubt if more prospective studies are needed to solve this problem.

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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):1–5).

Statistical considerations which dictate that serum ferritin will always outrank transferrin, in turn 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.3 Statistical considerations also dictate acknowledgement of mean corpuscular haemoglobin (MCH) as a predictive entity in its own right following documentation of an MCH of <27 pg as superior to a mean corpuscular volume (MCV) of <77 fl in predicting serum ferritin levels of <20 µg/l.4 All low MCV values had low MCH values but nine haemoglobinopathic patients with low MCV had MCH within the normal range.

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References

2 Jonkers D, Gisbertz I, De Bruine A, et al. Recovery of 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 extraintestinal lesions regressed 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 extragastric MALT lymphomas can be given.

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In my study, comprising 201 subjects with iron deficiency (characterised by serum ferritin <18 μg/l), the MCH conferring 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 <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 pg compared with only four with an MCV >80 pg in the presence of an MCH <26 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%) <77 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.

Reply

EDITOR,—Suggesting both that transferrin saturation may help in the diagnosis and that ferritin is the most powerful test for iron deficiency anaemia in the elderly, Am J Med 1990;88:205–9.


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 Gusco, D Sarli, and the authors of A138 are L Sarli, R Costi, S Gobbi, C Pavliidis, L Roncoroni.

NOTES

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 gastrointestinal and hepatology so that an individual can acquire or develop new knowledge or a technical skill. This newly acquired knowledge or skill can 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 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.

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, and then be used as the springboard for the international meeting. The programme includes: Mari- lyn Katz, Secretariat, GI Malignancies, Target Tours, PO Box 29041, Tel Aviv 61290, Israel. Tel: +972 3 5175150; fax: +972 3 5175155; email: gij@targetconf.com.

Redefining Priorities in Gastroenterology

This conference will be held on 11–14 April 2001 in Monte Carlo, Italy. It will be chaired by Professor Massimo Crepaldi (Rome, Italy) and Professor Eammon Quigley (Cork, 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.
Cholecystectomy and bowel function

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