Endoscopic findings and clinical patterns are not useful for distinguishing low from high grade MALT lymphoma

EDITOR,—Taal et al showed recently (Gut 1996; 39: 556–61) that both clinical parameters and endoscopic findings differ significantly between low and high grade gastric mucosa associated lymphoid tissue (MALT) lymphoma. In fact, low grade MALT lymphoma has often been interpreted endoscopically as a benign condition, whereas high grade MALT lymphoma often has neoplastic-like features. Weight loss and older age are more frequent in high grade MALT lymphoma.

In our experience the endoscopic findings and clinical parameters differ from the data reported by Taal et al.

Between 1993 and 1996 we diagnosed one high grade and three low grade MALT lymphomas. In the low grade MALT lymphomas, one 70 year old man and two women, one 65 and the other 56 years old, the endoscopic findings were a gastric ulcer in the main body of the stomach and a large antral mass with multiple ulcerations in the first and a large polypoid mass with pyloric obstruction in the second female patient. All of them had lost weight (6–10 kg.). The male patient and the female patient with multiple antral ulcerations were Helicobacter pylori positive. These patients were treated with an H pylori eradication regimen, and the H pylori negative patient was treated with polichemotherapy. In all cases the tumour regressed and the endoscopic pattern resolved. In the 67 year old woman with high grade MALT lymphoma the endoscopy showed a pinpoint stenosis of the pilorus, with a large mass ulcerated at the antrumus and weight loss (6 kg. in two months). Although H pylori was present, the patient was treated successfully with surgery (gastrectomy), followed by polichemotherapy. These data show that the clinical and endoscopic patterns are very similar in low and high grade MALT lymphoma, and that it is very difficult to determine the grade of the MALT lymphoma on the basis of the endoscopic or clinical, or both, patterns.

At the initial gastroscopy the male patient with the low grade MALT lymphoma had two adjacent ulcers on the posterior wall of the gastric corpus, was H pylori positive, and had histological and immunohistochemical evidence of low grade MALT lymphoma only in gastric mucosal specimens obtained from the ulcer margins. Anti-H pylori treatment resulted both in the regression of the ulcer and regression of the neoplasia, with resolution of the ulcers. After 18 months, the patient was reinfected by H pylori and endoscopy showed a diffuse thickening of gastric mucosal folds in all gastric sites, mimicking limitis plastica. Histological and immunohistochemical analyses showed a low grade MALT lymphoma. After the failure of two consecutive courses of anti-H pylori treatment, the patient was treated surgically due to progressive weight loss. Histological examination of the surgical specimens confirmed the diagnosis of low grade MALT lymphoma, stage E1.

This case shows clearly that the endoscopic pattern of low grade MALT lymphoma can change greatly even in the single patient, making it impossible to use the endoscopic or clinical pattern to distinguish between low and high grade MALT lymphoma.

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Reply

EDITOR,—The endoscopic pattern of gastric non-Hodgkin’s lymphoma (NHL) has been described by several authors. There are three main patterns: a tumour-like appearance with a polypoid mass (exophytic type); ulceration or multiple small erosions (infiltra-

itive type); or thickened, giant folds such as pseudo-polyps (endophytic type). These descriptions are not specific for gastric NHL. As gastric lymphoma is a rather rare disease in 52% of 51 patients with low grade NHL, the high grade malignancies are more frequent in high grade MALT lymphoma.

In our series of 114 patients with primary gastric lymphoma we evaluated the initial endoscopic diagnosis in relation to clinical findings and grade of NHL. As gastric lymphoma is a rather uncommon disease, one might imagine that the endoscopist is not always aware of the possibility of a lymphoma rather than a carcinoma or even the more frequently present gastritis.

In our series of 114 patients with primary gastric lymphoma we evaluated the initial endoscopic diagnosis in relation to clinical findings and grade of NHL. As gastric lymphoma is a rather uncommon disease, one might imagine that the endoscopist is not always aware of the possibility of a lymphoma rather than a carcinoma or even the more frequently present gastritis.

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Natural history of polypoid lesions of the gall bladder

EDITOR,—Moriguchi et al (Gut 1996; 39: 860–2) report that of 4543 patients, 109 had gall bladder polyps detected on ultrasound, most of which were benign. They follow this with an interesting brief review of the literature. From their comments and my own reading of the subject, the overwhelming majority of gall bladder polyp detections, although one of the 109 patients was found to have gall bladder cancer histologically, and this ties in with previous reports. Koga et al found carcinoma of the gall bladder presenting as polypoid lesions in three of four cases on ultrasound in their study and, surprisingly, by gall bladder wall thickening in only one of the four cases. Although the numbers are small and gall bladder cancer is relatively rare, it still comprises 3% of our patients with concurrently malignantancies and causes 6500 deaths annually in the USA. In addition, the spread and size of tumour are correlated. The patients who survive more than five years are those in whom the cancer is diagnosed histo-

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any conclusion from this study in terms of the prognosis of polypoid lesions of the gall bladder.

Prospective studies are needed to determine whether polypoid lesions develop into gall bladder carcinoma. Progression from adenoma to carcinoma takes time, and therefore these patients should be followed long term.

**Reply**

**Editor,**—We are grateful to Dr Johnson for his interest in our recent paper. As we described in the title, we studied the natural history of polypoid lesions in the gall bladder. The study by Yang et al cited by Dr Johnson, however, showed the prevalence of gall bladder carcinoma in patients who had undergone cholecystectomy. In their paper, the prognosis of polypoid lesions of the gall bladder is not described. To our knowledge, there are no papers that describe the natural history of polypoid lesions in the gall bladder in the literature. Therefore, it is difficult to say whether our study underestimates the risk of malignancy. A large study does not necessarily result in the correct conclusion, but we do feel that our study would have benefited from a larger series of patients.

All of the patients presented to the outpatient with heterogeneous abdominal complaints, but were asymptomatic during follow up, differing in this regard from the patients studied by Yang et al. In our opinion patients with right upper quadrant symptoms should not be followed for five years without treatment.

Finally, we agree with Dr Johnson that patients with gallstones should be monitored carefully. However, none of our patients with gallstones despite the close association between gall bladder carcinoma and gallstones.

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**Polypoid lesions of the gall bladder**

**Editor,**—The paper by Moriguchi et al (Gut 1996; 39: 860–2) raises a number of questions. The authors state that this is the first study to describe the natural history of these lesions, but in our opinion, the conclusion that most polypoid lesions of the gall bladder detected by ultrasonography are benign, is not new. Indeed, they seem to underestimate the risk of malignancy compared with other larger series.

Yang et al study 182 patients who underwent cholecystectomy and who had an ultrasonographic or pathological diagnosis of polypoid lesions of the gall bladder. There were 10 false positive ultrasound diagnoses. All the lesions less than 1 cm in diameter were benign. There were 13 malignant lesions, 11 of which were greater than 1.5 cm in diameter. All the malignant lesions were solitary. All but two of the 182 patients underwent cholecystectomy and the calculated sensitivity of ultrasonography was 90.1% and specificity was 93.9%.

These findings were in agreement with our study in terms of the incidence of symptoms. Moriguchi et al do not indicate how their patients were selected, nor whether they were symptomatic. However, it seems likely that most were asymptomatic as only four patients underwent cholecystectomy during a five year follow up period. It may be that differences in selection explain their much lower incidence of polyps greater than 1 cm (6.4% v 19.8%) and of malignant tumours (1.8% v 7.6%).

While most gall bladder polypoid lesions may indeed be benign, those which exceed 1 cm in diameter and are single have a high risk of malignancy and should be removed surgically, especially if the patient is over 50 years old. Unfortunately, Moriguchi et al’s study does not give us any information about the relationship between symptoms and gall bladder polypoid lesions. It seems reasonable to recommend that cholecystectomy be performed in symptomatic patients, particularly if gallstones are also present.

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**Bone mineral density in Crohn’s disease**

**Editor,**—We were interested to read the recent article by Jahnssen et al (Gut 1997; 40: 313–9). We have just published a similar study investigating the bone mineral content (BMC) of children with inflammatory bowel disease (IBD), and have also found that BMC was reduced in those with Crohn’s disease but not in those with ulcerative colitis.

No relation could be found between BMC and disease duration, disease activity, or biochemical markers of bone metabolism. There was, however, a significant relation between reduction in bone mineralisation and steroid usage, as also noted by Jahnssen et al. Although BMC was significantly lower in the children treated with steroids, there was no association with magnitude of steroid use in either dosage or duration. We postulated that the steroid effect was an all or nothing effect, or that the use of steroids acts as a marker for other variables, such as worsening disease activity.

We agree that screening of bone mineral status in an important aspect of continuing care in patients with IBD and would like to emphasise that children are not immune to the osteopenic side effects of IBD.

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**Letters, Book review, Notes**


**E Kyriacou**

London


Incidence of persistent symptoms after laparoscopic cholecystectomy

EDITOR,—I read with great interest the article by Luman et al (Gut 1996; 39: 863–6) on the incidence of persistent symptoms after laparoscopic cholecystectomy. I would like to point out that their statement that no study has analysed prospectively symptoms before and after laparoscopic cholecystectomy is not quite true. Although in a much smaller patient population, we published a paper on the same subject in 1995. In our study, we found that cholecystectomy significantly improved quality of life, and cured nausea, fatty food upset, abdominal distension, and biliary pain. We also found that laparoscopic cholecystectomy improved quality of life and symptomatology at an earlier stage than conventional cholecystectomy.

Furthermore, I would like to stress that, although Luman et al’s study provides us with lots of interesting data and recommendations, it also leaves us with many unanswered questions. How many patients were excluded from the study because of planned open cholecystectomy and inability to answer the questionnaire? What were the reasons for treating patients by open cholecystectomy? How many patients underwent preoperative endoscopic retrograde cholangiopancreatography (ERCP) and did all of them undergo sphincterotomy? Were there any differences in symptoms between patients with or without sphincterotomy? Was ERCP not mentioned in the analysis of preoperative investigations (Table IV)? Was there symptomatic relief of heartburn in patients from the uncomplicated group? I would be grateful if the authors would answer some of these questions.

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An exceptional high concentration of serum CA 19.9 in a patient with alcoholic liver disease

EDITOR,—Since the discovery by Koprowski and co-workers in 1979, raised serum concentrations of carbohydrate antigenic determinants, CA 19.9, has been used as an additional diagnostic test for adenocarcinoma of the upper gastrointestinal tract. According to Benamouzig et al, at the usual 37 U/ml cut off value, CA 19.9 had a sensitivity of 90% and a specificity of 45% for malignant biliary obstruction due to pancreatic or biliary neoplasms. For a more increased cut off value of 200 U/ml, its sensitivity was 65% and specificity was 91%. If higher cut off values are used, specificity rises so that at levels greater than 1000 U/ml, it approaches 100%.

A very high concentration of serum CA 19.9 is therefore currently the “gold standard” marker for malignant biliary obstruction resulting from pancreatic cancer.

Here, we report an exceptional high concentration of serum CA 19.9 in a patient with alcoholic liver disease. When the patient ceased drinking, the CA 19.9 serum concentration returned to normal within few months.

CASE REPORT
A 58 year man was admitted to our hospital because of jaundice and weight loss. He also complained of fatigue and pruritus of several weeks duration. His medical history was, except for psoriasis, unremarkable. However, he took acetylcysteine, eprazinochlorohydride, and prior to admission clarithromycin for a respiratory infection and obstructive lung disease. The patient had been a practicing business man who had been drinking 15 pints of beer a day for many years and for the past two years also a quarter bottle of gin a day.

Physical examination showed jaundice, firm hepatomegaly and psoriatic skin lesions.

Laboratory investigations revealed increased concentrations of plasma bilirubin (total 6.26 mg/dl, conjugated 3.92 mg/dl, normal <1 mg/dl), alkaline phosphatase (1071 U/l, normal <240 U/l), aspartate aminotransferase (197 U/l, normal <20 U/l), alanine aminotransferase (101 U/l, normal <20 U/l), and y-glutamyltransferase (1700 U/l, normal <60 U/l). The patient also had raised concentrations of triglycerides (1968 mg/dl, normal <200 mg/dl) and cholesterol (667 mg/dl, normal <240 mg/dl).

Serological markers for hepatitis A, B, and C did not indicate a recent infection or active viral replication. Autoantibodies were negative. The serum CA 19.9 was extremely high at 10.981 U/ml (confirmed in a retrospective examination), while carcinoembryonic antigen (CEA; normal <5µg/l) was 14.4 µg/l.

An ultrasonography and a computed tomography scan indicated hepatomegaly and fatty change without focal lesions or dilated biliary ducts. Endoscopic retrograde cholangiopancreatography showed no abnormalities, in particular no signs of primary sclerosing cholangitis or other biliary and pancreatic duct abnormalities.

A liver biopsy revealed micro- and macrovesicular steatosis, periportal fibrosis, Mallory’s bodies, in addition to the presence of biliary microabscesses. Liver histopathology was very suggestive of alcoholic liver changes.
Although the use of medical drugs, especially clarithromycin, may contribute to this clinical syndrome, we believe that alcohol misuse was the most attributable factor. We therefore advised the patient to stop drinking. Once alcohol consumption ceased, there was a dramatic improvement in clinical and biochemical abnormalities. During a follow up visit three months after presentation, biochemistry values had almost returned to normal.

The serum concentration of CA 19.9 may be found in patients with alcoholic liver disease.

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NOTES

Sir Francis Avery Jones BSG Research Award 1998

Applications are invited by the Education Committee of the British Society of Gastroenterology who will recommend to Council the recipient of the 1998 Award. Applications (TWENTY 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 or less on 31 December 1997 but need not be a member of the BSG. The recipient will be required to deliver a 40 minute lecture at the Annual Meeting of the Society in March 1998. Applications (TWENTY COPIES) should be made to the Honorary Secretary, BSG, 3 St Andrews Place, London NW1 4LB, by 1 December 1997.

Course in Postgraduate Gastroenterology

A Course in Postgraduate Gastroenterology will be held in Oxford, UK, on 4–7 January 1998. This course has been designed for consultants and registrars, including those who do not specialise in gastroenterology. Topics will include:

- Liver disease
- Colonic neoplasia
- Acute pancreatitis
- Osteoporosis, arthritis and GI disease
- Food allergy and intolerance.

Course fee £200 ($330). Board and accommodation are available at Wadham College at extra cost. Six bursaries will be available for applicants training in gastroenterology or in research posts at British hospitals. Further information from: Dr DP Jewell, Gastroenterology Unit, Radcliffe Infirmary, Woodstock Road, Oxford OX2 6HE.

9th International Symposium on Cells of the Hepatic Sinusoid

The 9th International Symposium on Cells of the Hepatic Sinusoid will be held in Christchurch, New Zealand, from 27 September to 1 October 1998. Further information from: Professor Robin Fraser, I.S.C.H.S., Christchurch School of Medicine, PO Box 4345, Christchurch 8001, New Zealand. Tel: +64 3 3640 387; Fax: +64 3 3640 593; email: grogers@chmeds.ac.nz.
Endoscopic findings and clinical patterns are not useful for distinguishing low from high grade gastric MALT lymphoma

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