Endoscopic findings and clinical patterns are not useful for distinguishing low from high grade gastric 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 male patient 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 pit-point stenosis of the pylorus, with a large mass ulcerated at the angulus and weight loss (6 kg in two months). Although H pylori was present, the patient was treated successfully with surgery (gastrectomy), followed by polichemotherapy. Both these data show that the clinical and endoscopic patterns are very similar in low and high grade MALT lymphoma, and that 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 complete remission 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 IIa.

This case shows clearly that the endoscopic pattern of low grade MALT lymphoma can change greatly even in the same 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.1 There are three main patterns: a tumour-like appearance with a polypoid mass (exophytic type); ulceration or multiple small erosions (infil- trative type); or thickened, giant folds such as those seen in limitis plastica. Unfortunately, these descriptions are not specific for gastric 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 malignancy: the endoscopic diagnosis in relation to clinical or histological recognition of gastric NHL is difficult; a high index of suspicion may help, but we feel that it is of utmost importance to take routine biopsy specimens in as many patients as possible, preferably all patients seen in the GI endoscopy unit. So we do not agree with Tursi et al who reject our conclusions (based on the experience in 114 stage I patients) with a report of four cases only, which cannot be considered as an impressive number of cases or even a series of patients.

The issue of treatment of gastric NHL by eradication of H pylori was not the subject of our study. Information on this topic is increasing and eradication treatment may induce complete regression in non-bulky, low grade MALT lymphoma stage I in approximately 70% of cases.2 3 The value of H pylori eradication in bulky disease as mentioned by Tursi et al is still a matter of much debate.

In general, it is important to detect gastric lymphoma at an early stage when treatment might be less toxic and most effective. Therefore, we would like to stress that a diagnosis of low grade gastric NHL should be kept in mind when encountering benign gastritis, and that high grade NHL NHL is often depicted with carcinoma-like features. Multiple biopsy specimens are an absolute requirement for an optimal diagnosis and treatment choice.

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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 polyps are benign, although one of the 109 patients was found to have gall bladder cancer histologically, and this ties in with previous reports.1 2 Koga et al found carcinoma of the gall bladder presenting as polypoid lesions in three of four cases, although one of the 109 patients was found to have gall bladder cancer histologically, and this ties in with previous reports.1 3 In addition, the spread and size of tumour are correlated.1 4 The patients who survive more than five years are those in whom the cancer is diagnosed histo-

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logically at cholecystectomy for presumed benign disease—that is early, with disease confined to the mucosa or submucosa. Disease beyond these limits is accompanied by a drastic drop in survival rates at five years from 64 to 44% and the overall five year prognosis is quoted as less than 5%. Gallstones have also been found commonly in association with both gall bladder polyps and gall bladder malignancy. In the light of these findings, early cholecystectomy in suspected patients could reduce long term mortality. Preliminary histology at surgery could permit dissection of portal hepatic nodes, excision of the gall bladder bed, and right hepatic lobectomy at initial cholecystectomy, thus improving survival and reducing the costs of repeat operation.

What remains controversial and undecided, however, is how to select those patients who should have early cholecystectomy from those who should have six monthly follow up ultrasound. Certainly, rapidly enlarging lesions or those with areas of gall bladder wall thickening should be removed, including benign lesions in women, especially those over 60, and in patients with gallstones, even asymptomatic ones. In the study by Moriguchi et al. the size of the polyps was not related to whether malignancy developed, extrapolating from the adenoma–carcinoma sequence that large gall bladder adenomas are premalignant lesions. Indeed in Moriguchi et al's study it is interesting that the larger polyps were found in the younger age group. Clearly, logistic reasons dictate that not every patient with polyps should have an instant cholecystectomy if asymptomatic, and there does have to be a process of sifting and selection, but, in my view, this should err towards the side of more frequent surgery in the fit patient for the reasons discussed above.

E KYRIACOU
London


Reply

EDITORS.—We are grateful to Dr Johnson for his interest in our recent paper. We agree with him that there is a small number of patients who are diagnosed with gall bladder carcinoma and that early detection of carcinoma would improve their prognosis. The study by Koga and Koga et al cited by Dr Kyriacou, however, describes the prevalence of gall bladder carcinoma in patients who had undergone cholecystectomy. Therefore, it is difficult to draw 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.

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Polypoid lesions of the gall bladder

EDITORS.—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 their solitary 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 al1 studied 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 patients with right upper quadrant symptoms. Moriguchi et al do not indicate how their patients were selected, nor whether they were asymptomatic. 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% vs 19.8%) and of malignant tumours (1.8% vs 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.1 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.


Bone mineral density in Crohn’s disease

EDITORS.—We were interested to read the recent article by Jahnsen 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),1 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 Jahnsen 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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Reply

EDITORS.—We were 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 prevalence 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 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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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 deter-
mens, CA 19.9, has become an additional diagnostic test for adenocarci-
noma of the upper gastrointestinal tract. 1-4 According to Benamouzig et al, 5 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%. 6 A very high concentration of serum CA 19.9 is therefore currently the “gold standard” marker for malignant biliary obstruc-
tion resulting from pancreatic cancer. 7

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 concen-
tration 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, eprazinohydrochlo-
ride, and prior to admission clarithromycin for a respiratory infection and obstructive lung disease. The patient had been a heavy drinker for 25 years. He stopped drinking a day. For his ischiatric pain, he also took naloxonhydrochloride and tramadolhydro-
chloride. In addition he consumed approxi-
mately 10 pints of beer a day for many years and for the past two years also a quarter bot-
tle 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 ami-
cotransferase (1398 mg/dl, normal <200 mg/dl) and cholesterol (1700 U/l, normal <200 U/l), and γ-glutamyltranspeptidase (1700 U/l, normal <60 U/l). The patient also had raised concentrations of triglycerides (1398 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 nega-
tive. CA 19.9 was extremely high at 10.981 U/ml (confirmed in a retrospective examination), while carcinoembryonic anti-
gen (CEA; normal <5 μg/l) was 14.4 μg/l.

An ultrasonography and a computed tomo-
graphy scan indicated hepaticomegaly and fatty change without focal lesions or dilated biliary ducts. Endoscopic retrograde cholan-
giopancreatography showed no abnormali-
ties, in particular no signs of primary scleros-
ing cholangitis or other biliary or pancreatic duct abnormalities.

A liver biopsy revealed micro- and macro-
vesicular steatosis, periforal fibrosis, Ma-
lory’s bodies, in addition to the presence of bilirubinostasis. Liver histology was very sug-
gestive of alcoholic liver changes.

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2 Nathanson LK, Shimi S, Cushieri A. Laparo-
scopic cholecystectomy: the Dundee tech-

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Incidence of persistent symptoms after laparoscopic cholecystectomy

EDITOR,—I read with great interest the article by Luman et al (Gut 1994; 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 pro-
spectively symptoms before and after laparo-
scopic cholecystectomy is not quite true. Although in a much smaller patient popula-
tion, we published a paper on the same subject in 1995. 1 In our study, we found that cholecystectomy significantly improved qual-
ity 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 cholecs-
tectomy.

Furthermore, I would like to stress that, although Luman et al’s study provides us with lots of interesting data and recommen-
dations, it also leaves us with many unan-
swered 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 cholangiopancreatog-
omy (ERCP) and did all of them undergo sphincterotomy? Were there any differences in symptoms between patients with or without sphincterotomy? How was ERCP not mentioned in the analysis of preoperative investiga-
tions (Table IV)? Was there symptoma-
tic 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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1 Plaisier PW, van der Hul RL, Nijss HGT, den Hertoghe L, Terstra OT, Brunt EH. Quality of life and the course of biliary and gastrointesti-
nal symptoms after laparoscopic and conven-

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Reply

EDITOR,—We are grateful to Drs Cowan, Gregory and Jenkins for their interest and comments on our study. Crohn’s disease is obviously a more severe systemic disease than ulcerative colitis and surprisingly most stud-
ies have failed to demonstrate a difference in bone mineral density between the two patient groups.

The adverse effects of corticosteroids on bone density are well recognised. However, published studies provide conflicting results on the association between steroid treatment and bone mineral density in patients with IBD. 1-7 The cumulative steroid dose is usually calculated retrospectively from medical records and indeed this method is uncertain as treatment is intermittent and of varying length and dose. Inaccurate estimation of the total steroid dose may have contributed to the conflicting results reported in the literature on this issue.

We fully agree that steroid use (but also total lifetime dose) might reflect other variables connected to the intestinal illness which impact on bone mineralisation, such as disease activity.

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2 Bjarnason I, Macpherson A, Macintosh C, Buiston-Thomas M, Foragu I, Moniz C. Reduced bone density in patients with inflam-
mation of the gut between the two patient

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Letters, Book reviews, Notes
The time will come, one supposes, when all the brain’s mysteries will be disclosed. Then, the combined forces of information technology and neurosciences will come up with methods to introduce and store knowledge—conveniently when we are asleep—so that existing methods of learning will not suffice. The lecture, seminar and written word will become redundant.

What will then become a blockbuster textbook such as the present offering? Is it to be replaced by a chip downloaded directly into our brains? Maybe so, but until that time we shall just have to make do with the good, old-fashioned read—and try and retain what we can.

If we are to continue with books for the time being, then please let them be as agreeable as the latest edition of Inflammatory Bowel Diseases. The greatest care has gone into the choice of chapters (112) and authors (155) to produce a definitive text for our time.

This third edition, appearing some seven years after its predecessor, reflects the steady progress that has been made in our knowledge of this most enigmatic of areas within clinical gastroenterology. The addition of three new editors has led to the production of a volume that is, in many ways, very much more than just an update of the previous version. There are nearly 200 pages on pathogenesis which at least enable the clinician to feel a little more confident about answering patients’ most invariable question, at some time or other, as to the cause of their condition. Knowledge may provide comfort even if we all know the best short answer to that highly pertinent query! The clinical section offers comprehensive coverage of diagnostics (incorporating new imaging modalities), the treatment section has been updated and, in the case of surgery, the text has been more than minimally invaded by completely new material.

This book is 1000 pages long, and one perhaps should be a little wary of highlighting omissions. The field is fairly fast-moving, but this reviewer would like to have seen a little more about host-bacterial interaction in the gut. Presumably, the deadline was a little too late to cover all of the important recent progress in genetics. Similarly, targeted therapy has not received the attention that it might do in, for example, an annual update. It is somewhat of an old canard to say that textbooks are out-of-date as soon as they hit the bookstalls. I certainly heard that said when I was a medical student some 25 years ago, but surely it must be ever-tamer today and in the future as online journals become available as the latest edition of Inflammatory Bowel Diseases. The greatest care has gone into the choice of chapters (112) and authors (155) to produce a definitive text for our time.

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

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