LETTERS

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Long term follow up of Helicobacter pylori induced gastric diffuse large B cell MALT lymphoma following eradication treatment alone

I was interested to read the article by Alsolaiman and colleagues on the long term follow up of gastric diffuse large B cell MALT lymphoma after eradication of Helicobacter pylori. I was encouraged to read this message in as a rare situation of being faced with a high grade gastric MALT lymphoma, one would feel confident to try antibiotic eradication of H. pylori alone with careful endoscopic surveillance, as is often employed in the case of low grade gastric MALT lymphoma.

R Sinharay
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References

Is hepatobiliary scintigraphy insensitive for the diagnosis of sphincter of Oddi dysfunction?

I was very pleased to read the letter by Dr Madacsi in response to our article “Scintigraphy versus manometry in patients with suspected sphincter of Oddi dysfunction” (Gut 2000;47:1191–202) and its findings which showed abnormal results of QHBS and endoscopy in patients with sphincter of Oddi dysfunction.

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References

PostScript

Gut 2003;52:1385–1390

Table 1

<table>
<thead>
<tr>
<th>T1/2 parameter of common bile duct emptying, measured by scintigraphy</th>
<th>QBHS (min)</th>
<th>QBHS+CKC (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>43</td>
<td>18</td>
</tr>
<tr>
<td>SD</td>
<td>23</td>
<td>16</td>
</tr>
</tbody>
</table>

QHBS, quantitative hepatobiliary scintigraphy; CCK, cholecystokinin.
Table 2  Sensitivity and specificity of scintigraphic results as compared with manometry

<table>
<thead>
<tr>
<th></th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>PPV (%)</th>
<th>NPV (%)</th>
<th>Accuracy (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>QHBS positive</td>
<td>79</td>
<td>71</td>
<td>88</td>
<td>55</td>
<td>77</td>
</tr>
<tr>
<td>CBD=12 mm</td>
<td>42</td>
<td>86</td>
<td>89</td>
<td>35</td>
<td>54</td>
</tr>
<tr>
<td>Abnormal LFT</td>
<td>26</td>
<td>71</td>
<td>71</td>
<td>23</td>
<td>38</td>
</tr>
</tbody>
</table>

QHBS, quantitative hepatobiliary scintigraphy; CBD, common bile duct; LFT, liver function test; PPV, positive predictive value; NPV, negative predictive value.


Worsening of steatosis and fibrosis progression

We read with great interest the article by Castera and colleagues (Gut 2003;52:288–92) and acknowledge that worsening of steatosis in chronic hepatitis C is associated with fibrosis progression. However, in our view there are no data supporting a causal role for this association and any specific relation of this finding to chronic hepatitis C.

Firstly, the authors provide no explanation as to why steatosis worsened in patients under consideration. Overweight, diabetes, and alcohol consumption are the main causes of steatosis in Western countries and major causes of fibrotic liver disease. There are no data throughout the study indicating whether patients in whom steatosis worsened simply gained weight or developed any of the complications associated with insulin resistance. The latter can develop within the course of liver injury and be present or be epidemiologically linked to infection by hepatitis C virus (HCV) for reasons that have yet to be determined. High serum glucose, 1 as well as diabetes, 2 are associated with liver fibrosis progression 3 and might contribute to enhanced fibrogenesis. 4,5 For alcohol consumption, a thorough evaluation is needed before ruling out the possibility of even slight increases in daily alcohol consumption transiting over the course of several years, to enhanced steatosis. There is a theoretical possibility that progression of steatosis reflects the natural course of HCV infection if steatosis were to occur later than the necroinflammatory lesions defining chronic hepatitis. However, as current knowledge stands, this is purely speculative and also, there is no indication in this study that patients in whom steatosis progressed had a longer duration of infection than those in whom it did not. Hence there appears to be no data in this study suggesting that progression of steatosis had a longer duration of infection than those in whom it did not. Therefore, in our study, progression of steatosis was associated with progression of fibrosis.

The role for this statistical association or any speculation as to why steatosis worsened in patients with chronic hepatitis C remains poorly understood.

References


Colorectal screening guidelines in acromegaly

We write with concern regarding the recent guidelines published for colorectal screening in acromegaly.

Colorectal screening guidelines in acromegaly

We write with concern regarding the recent screening guidelines published for colorectal cancer in acromegaly. While the risks of colorectal cancer in patients with acromegaly have an increased risk of developing colorectal cancer, this does not necessarily mean that colorectal screening in acromegaly is warranted.

The endocrine literature has witnessed a significant debate, polarising into two separate camps. In 2002, Colorectal screening guidelines were published in a follow-up study. The endocrine literature has witnessed a significant debate, polarising into two separate camps. In 2002, Colorectal screening guidelines were published in a follow-up study.

The risks of colorectal cancer in patients with acromegaly have been studied in various case series. The risks of colorectal cancer in patients with acromegaly have been studied in various case series. For example, in a study by Perry and colleagues, they detected 10 asymptomatic cancers, as well as numerous adenomas. In this same 500 examinations, we experienced. In this same 500 examinations, we experienced. In this same 500 examinations, we experienced.

Atrophic gastritis: pathology and endoscopy in the reversibility assessment

We read with interest the paper by Walker et al. We agree that histology remains the most suitable test for both detecting and assessing reversion of atrophic gastritis. Such a view elicits two basic questions, however: (1) how extensively to biopsy, to correctly evaluate any presence/absence of gastric atrophy? (2) to amalgamate the different viewpoints and also test interobserver agreement in atrophy classification, an international group of pathologists recently published an extensive description of the different phenotypes of gastric atrophy. By merging Western and Eastern experiences, the new proposal extensively describes the diagnostic categories that should be adopted to enable acceptable comparisons between clinicopathological studies involving gastric atrophy (both non-metaplastic and metaplastic). The proposed classification also introduces a new diagnostic category (that is, indefinite for atrophy) which permits the evaluation of atrophy when high grade inflammation—mostly related to Helicobacter pylori infection—interferes with a reliable assessment of the “loss of appropriate glands”.

As for the number and location of biopsies for atrophy assessment, the recommendations of the updated Sydney system seem to be a suitable compromise between the excessive pathologists’ demands and the limiting limits of routine practice. The question of “where to biopsy” is more intriguing. No doubt both the oxyntic and antral mucosa need to be tested, but endoscopists too often neglect the recommendations for the updated Sydney system.

Atrophic gastritis: pathology and endoscopy in the reversibility assessment

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Acknowledgements

The results of this study were discussed with Pelayo Correa; the authors thank Professor Correa for his valuable advice.

References

2. Genta RM. "We used the Sydney System...". Am J Gastroenterol 1997;92:1960-1.

Diversion colitis with a mucosal tear on endoscopic insufflation

I read with interest the report of Cruz-Correa and colleagues (Gut 2002;51:600). They described three cases of collagenous colitis with mucosal tears on endoscopic insufflation and stated that as far as they were aware there were no reports in other gastrointestinal diseases. We would like to present the case of a similar mucosal tear on endoscopic insufflation in a patient with diversion colitis.

A 46 year old Japanese man presented with an acute abdomen caused by ascending colon diverticular perforation. He underwent drainage of the abdominal cavity with loop colostomy. He had been suffering from systemic lupus erythematosus and chronic renal failure for 25 years. He had received more than 90 g of oral steroid at the time of referral and was taking 10 mg/day. After operation, he was free from symptoms and gave no history of haematemesis or blood in stools. On surveillance colonoscopy, the dysfunctional colon mucosa, which was 10 cm away from the loop colostomy, was torn with slight bleeding, and the muscularis mucosal was exposed on endoscopic insufflation with air (fig 1). The lumen of the colon was narrowed and the remaining colon mucosa showed mild colitis with a decreased vascular pattern and oedema. The post endoscopic course was uneventful without any treatment. Routine laboratory investigations revealed: white blood cell count 10600/µl (normal range 4000–9000/µl), haemoglobin 12.2 g/dl (normal range 14–18 g/dl), haematocrit 36.1% (normal range 40–48%), blood urea nitrogen 76.4 mg/dl (normal range 9–21 mg/dl), and serum creatinine 1.14 mg/dl (normal range 0.6–1.2 mg/dl). Cultures for stool pathogens were negative.

Diversion colitis may occur in a part of the bowel that was previously healthy and which has been placed outside the faecal stream because of a proximal stoma. The mechanism of diversion colitis remains unclear but may be associated with changes in the intestinal bacterial flora, absence of essential nutrients, or intestinal toxins. In most cases, there are no symptoms, as in our case. Frisbie et al reported that mucosal erythema and friability were seen in most patients who had undergone diverting colostomy for neuropathic large bowel. Continuous high doses of steroids make human tissue fragile, including the colon mucosa. Taken together, these results

Table 1  By site prevalence of atrophic gastritis (distinguishing between atrophy with and without IM) in 504 consecutive Helicobacter pylori positive patients. Patients with phenotype "indeterminate for atrophy" and/or "foveolar restricted IM" were included among cases of non-atrophic gastritis.

<table>
<thead>
<tr>
<th></th>
<th>IM absent</th>
<th>IM present</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Corpus only</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Antrum only</td>
<td>3</td>
<td>10</td>
<td>13</td>
</tr>
<tr>
<td>Antrum and incisura angularis</td>
<td>6</td>
<td>14</td>
<td>20</td>
</tr>
<tr>
<td>Incisura angularis only</td>
<td>9</td>
<td>20</td>
<td>29</td>
</tr>
<tr>
<td>Total</td>
<td>18</td>
<td>45</td>
<td>63</td>
</tr>
</tbody>
</table>

IM, intestinal metaplasia.

Incisura angularis, number of patients whose gastric atrophy (with or without IM) was detected only in the incisura biopsy samples.

Antrum, number of patients whose gastric atrophy (with or without IM) was detected only in the antral biopsy samples.

Antrum and incisura angularis, number of patients whose gastric atrophy (with or without IM) was detected in both the antral and the incisura angularis biopsy samples.

Corpus, number of patients whose gastric atrophy (with IM) was detected only in the corpus biopsy samples.

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Acknowledgements

The results of this study were discussed with Pelayo Correa; the authors thank Professor Correa for his valuable advice.

References

2. Genta RM. "We used the Sydney System...". Am J Gastroenterol 1997;92:1960-1.
suggest that the mucosal tear in our case may have been attributable to diversion colitis with fragile mucosa.

PH10A

PH10A

Edited by T Tsuji, T Higashi, M Zeniya, Advances in the Treatment of Gastroenterology alike in the 21st century. They have made a creditable attempt especially viral hepatitis and autoimmune diseases. They have informed us of the varied characteristics of these diseases and developed novel therapies. Can this textbook of IBS help their plight? The authors (both basic scientists at heart) will be most welcome in the next edition. The synopsis claims this text to be an “updated and comprehensive description of the most relevant features of colorectal cancer . . . ”—I beg to differ.

The image contains a page from a document discussing recent advances in immunology. The text highlights the importance of emerging immunology, mentioning that the complexity of the science involved makes it difficult for clinicians and scientists working predominantly in other areas to acquire an introduction to liver immunology. It also notes that the field of immunology has evolved, and that recent developments in immunology have been influential in understanding liver diseases and the molecular signals involved in their regulation. The text further discusses the role of dysregulated immune responses in various conditions and the importance of understanding the immune system's role in disease management.

References

BOOK REVIEWS
Molecular Biology and Immunology in Hepatology. Advances in the Treatment of Intractable Liver Diseases

Dysregulated immune responses underlie the pathogenesis of many liver disorders including not only autoimmune diseases but also viral hepatitis and the chronic inflammatory responses stimulated by alcohol. Thus understanding liver immunology and the molecular signals involved in its regulation are critical if we are to gain insights into the pathogenesis of these diseases and develop novel therapies. Recent advances in immunology have been phenomenal and it is not surprising that many clinicians find it difficult to integrate and understand the importance of emerging immunology research. The editors of this book are to be commended for providing a summary of our knowledge of immunology of the liver and how this informs liver diseases, especially viral hepatitis and autoimmune diseases. They have made a creditable attempt to demystify the molecular and immune complexities involved and to distil the field into one concise volume.

Chronic hepatitis C infection is one of the greatest challenges facing hepatologists and gastroenterologists alike in the 21st century and it is now clear that both viral and host immune factors determine the outcome of infection. It is thus not surprising that viral hepatitis accounts for a substantial part of the book, which covers issues from viral genetics and host responses to gene therapy of viral hepatitis and transgenic mouse models of viral progression and hepatocellular carcinoma. Potential mechanisms of autoimmune liver diseases and the clinical features of the various liver syndromes are thus also extensively reviewed. This section of the book demonstrates particularly well how an immunological understanding can provide direct insights into a fundamental disease. The book includes chapters dedicated to the clinical management of liver disease, including viral hepatitis, hepatocellular carcinoma, acute liver failure, and living related liver transplantation. There is no doubt that the authors’ extensive experience in the field of living related liver transplantation will appeal to physicians and surgeons alike but the insights brought to these areas by immunologists are less clearly stated.

The book is aimed at both clinicians and scientists, and provides much needed background reading in the rapidly evolving field of hepatology. However, it would be hard going for anyone without a background understanding of basic immunology/molecular biology given the complexity of the science involved. One problem with such a book is assessing the target audience. The rapid evolution of the immunological accounts for the fact that parts of this book will be out of date by the time it is published and therefore of less relevance to people working directly in the field. It is perhaps most useful for clinicians or scientists working predominantly in other areas who need an introduction to liver immunology. In this context it would have been helpful to include more explanatory diagrams and a rather more “user friendly” style. However, overall this is a useful book and a good introduction to liver immunology.

Irritable Bowel Syndrome: Diagnosis and Treatment

Gastroenterologists derive job satisfaction from performing endoscopic procedures, establishing diagnoses, and explaining and treating symptoms. Patients with irritable bowel syndrome (IBS) are not usually regular endoscopic procedures, diagnosis is often uncertain, symptoms cannot easily be explained, and there is no effective treatment. The health service will pay for its not surprising that therefore few gastroenterologists relish the prospect of seeing a patient with IBS. Can this textbook of IBS help their plight? The answer is undoubtedly yes; however, success with IBS patients depends critically on good communication, a skill that cannot be gained from reading a textbook. The authors (both basic scientists at heart) are to be congratulated on assembling a holistic collection of contributors and supplying lip service to the different profiles of IBS in varied clinical settings. Equal weight is given to psychological aspects, serotonergic receptors, physiology, causative factors, the concept of consultation behaviour, and many other factors. Thus the book is much more than a text-book on “diagnosis and treatment”. However, I support the concept that this wider view of IBS is mandatory for effective diagnosis and treatment.

The book is an excellent resource for all health professionals dealing with IBS and it will be a vital starting point for those wishing to research IBS. The chapters are extensively referenced and many questions in areas of uncertainty are left refreshingly open. My only regret is that the various chapters are not all of the same standard. Perhaps the most disappointing aspect of this book is the failure to put IBS in the context of other unexplained gastrointestinal symptoms. Grant Thompson provides a list of other functional gut disorders. This very medical approach to unexplained symptoms is driven by the need for clean entry criteria for drug trials and physiological research studies. It presumes that it must be possible to define distinct pathological entities and produce drugs to correct them. The real world is not like this: pure, typical, or textbook IBS is unusual. Patients present with a variety of symptoms, originating from many systems, some of which may have features of IBS. Clinicians reading this book might, quite reasonably, ask themselves whether they can apply all of this IBS information to the patients they see in clinics. I suspect that the underlying issues are similar, regardless of the underlying symptoms, but this remains to be proven. A chapter placing IBS in the context of other unexplained bodily sensations (IBS) will be most welcome in the next edition.

Colorectal Cancer

My initial reaction to a single author text on colorectal cancer was that the author was either a brave or a foolish man to attempt such an onerous task single handed. New information about aetiology, screening, pathogenesis, and all aspects of treatment have changed considerably in the last 10 years and the literature abounds with new information, only some of which is important, but all of it needs sifting to distil a worthwhile and up to date text.

The synopsis claims this text to be an “updated and comprehensive description of the most relevant features of colorectal cancer . . . ”—I beg to differ. In the preface, Dr de Leon states that he hopes the volume captures his spirit of the times. He is a constructive and critical attitude which may help a new generation of investigators, and to some extent the volume has achieved this aim. However, he also recognises his own limitations in taking on this daunting task. The author is not explicit as to who the book is aimed at but refers to a “new generation of investigators”—if this means that the
book is aimed at giving an overview of colorectal cancer to people working in basic science on colorectal cancer then the book is short enough to be digestible. As an overview of colorectal cancer in the 21st century, a single author could not be expected to do justice to the whole topic and this text is not a comprehensive overview of colorectal cancer.

The best sections of the book are not surprisingly those areas which Dr de Leon has written and published on himself, namely the genetics of colorectal cancer and chemoprevention of colorectal cancer. In many respects the excellent description of the state of the art in these areas highlights the inadequacies in other areas such as pathology, surgical technique, mesorectal excision, adjuvant chemotherapy, and the role of radiotherapy, which are covered in a superficial manner. With the exception of the genetics of colorectal cancer, the reviews of the literature are brief and highly selected. The section on adjuvant chemotherapy and the data presented on faecal occult blood screening are far too brief to do them justice given the current interest worldwide in these aspects of the disease. The section on screening by endoscopic means makes no mention of the potential complications of this modality, and surely deserves at least a mention. Unfortunately, there are also some inaccuracies in the book—for example, the section on screening by CT colography.

The book is written in a very readable style but with very few illustrations and the quality of the illustrations included is adequate. I found the lack of detail and lack of inclusion of some of the most relevant literature (the last five years) irritating and frustrating. Given the size of the task, I imagine such a book was several years in gestation and this may account for some recent important publications being omitted. It is certainly not a reference book but might provide useful background reading for investigators who are new to the area.

J H Scholefield

NOTICES

Sir Francis Avery Jones British Society of Gastroenterology Research Award 2004

Applications are invited by the Education Committee of the British Society of Gastroenterology who will recommend to Council the recipient of the 2004 Award. Applications (TWENTY COPIES) should include:

- A written statement confirming that all or a substantial part of the work has been personally conducted in the UK or Eire.
- 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

Applications are invited by the Endoscopy Committee of the British Society of Gastroenterology who will recommend to the Council the recipient of the 2004 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.

An applicant need not be a member of the Society. The recipient will be required to deliver a 30 minute lecture at the Annual meeting of the Society in September 2004.

Further details: Torkel Wadstrom, President- EHSG, Lund University, Department of Infectious Diseases & Medical Microbiology, Division of Bacteriology, Sövegatan 23, SE-223 62 Lund, Sweden. Tel: +46 46 173 241; fax: +46 46 152 564; email: torkel.wadstrom@mmb.lu.se; website: www.helicobacter.org

European Helicobacter Study Group (EHSG)

This meeting, on Helicobacter infections and gastroduodenal pathology, will be held on 3–6 September 2003 in Stockholm, Sweden. Further details: Professor Torkel Wadstrom, President- EHSG, Lund University, Department of Infectious Diseases & Medical Microbiology, Division of Bacteriology, Sövegatan 23, SE-223 62 Lund, Sweden. Tel: +46 46 173 241; fax: +46 46 152 564; email: torkel.wadstrom@mmb.lu.se; website: www.helicobacter.org

Falk Symposium 135—Immunological Diseases of Liver and Gut

This symposium will be held on 12–13 September 2003 in Prague, Czech Republic. Further details: Falk Foundation c.v., Congress Division, PO Box 6529, Leinwenberstr. 5, 79041 Freiburg/Br, Germany. Tel: +49 761 15 140; fax: +49 761 15 14 359; email: symposia@falkfoundation.de; website: www.falkfoundation.de

The European Society of Parenteral and Enteral Nutrition (ESPEN)

ESPEN will celebrate its silver anniversary at the time of the annual congress, which is to be held on 20–23 September 2003 in Cannes, France. Further details: www.espen.org

XII Falk Liver Week

The XII Falk Liver Week, in honour of Hans Popper’s 100th birthday, will be held on 15–22 October 2003 in Freiburg, Germany. Further details - see Falk Symposia above.

3rd Congress of the European Chapter of the American College of Nutrition

This meeting will be held on 14–15 November 2003 in Göttingen, Germany. Abstract deadline: 01 October 2003. Main topics: Metabolic Syndrome, Plant-genomics, Treatment of Obesity, Hormonal Regulation of the Body Weight, Pediatric Nutrition, Malnutrition, Food-induced Diseases, Food and Allergy. Further details: G Schickedanz, Congress Secretary, Department of Gastroenterology and Endocrinology, University of Göttingen, Robert-Koch-Str. 40, 37075 Göttingen, Germany. Tel: +49 551 396326; fax: +49 551 3919125; email: nutrition2003@med.uni-goettingen.de; website: www.nutrition-europe.org

European Course on Laparoscopic Endoscopy

This course will be held on 18–21 November 2003 in Brussels, Belgium. Further details: Secretary to Professor Cadière, Service de Chirurgie Digestive, Rue Haute 322, Brussels 1000, Belgium. Tel: +32 (0)2 648 07 60; fax: +32 (0)2 647 86 94; email: straeb.asmb@proximedia.be; website: www.straeb-asmb.com

Hong Kong-Shanghai International Liver Congress 2004

This conference will be held on 14–17 February 2004 in Hong Kong. The topic of the conference is “Liver Diseases in the Post-Genomic Era”. Further details: Ms Kristie Leung, Room 102–105 School of General Nursing, Queen Mary Hospital, 102 Pokfulam Road, Hong Kong. Tel: +852 2818 4300/8101 2442; fax: +852 2818 4030; email: kristieleung@hepa2004.org; website: www.hepa2004.org