Letter: The editors will decide as before whether to also publish it in a future paper issue. Providing it isn’t libellous or obscene, it can be retrieved by clicking on “eLetters” in the box at the top right hand corner. If you have a burning desire to respond to a paper published in Gut, why not make use of our “rapid response” option? Log onto our website (www.gutjnl.com), find the paper that interests you, and send your response via email by clicking on the “eLetters” option in the box at the top right hand corner. It will be posted within seven days. You can retrieve it by clicking on “read eletters” on our homepage. The editors will decide as before whether to also publish it in a future paper issue.

PostScript

**LETTERS**

Paris staging system for primary gastrointestinal lymphomas

Currently, much effort is put into the development of specific therapeutic approaches that are tailored to specific lymphoma entities. As a consequence, more specific information needs to be collected to support the choice of therapy. In gastric marginal zone B cell lymphoma of MALT-type, the need for specialisation in documentation of tumour extent in the organ wall has been stressed by the high predictive value for failure of response after eradication of the bacterium *Helicobacter pylori*. As tumour stage is one of the most important guidelines in the choice of local therapy (surgery, radiotherapy) and chemotherapy, adequate documentation of tumour localisation in the organ related lymph nodes and beyond is essential.

The Ann Arbor staging system, developed for and routinely used in nodal non-Hodgkin’s lymphoma, is not optimal for documentation of the specific relevant features of primary extranodal lymphoma in the gastrointestinal tract. Several modifications and alternatives have been proposed. However, neither differentiation of stage II (tumour extent beyond the submucosa according to Radaszkiewicz and colleagues) nor discrimination of involvement by extranodal lymphomas is sufficiently serving the demand for documenting all features of the more advanced lesion. Therefore, the Lugano system is proposing a modified TNM staging system, named after the first venue of the group in Paris. The staging system adequately records: (1) depth of tumour infiltration; (2) extent of localisation of the tumour in the organ related lymph nodes and beyond; (3) specific lymphoma spreading (table 1). It is adjusted to the gastrointestinal origin of the lymphoma, considering histopathological characteristics of extranodal B and T cell lymphomas. The use of this system in future studies will permit accurate comparison of the reported cohorts and should allow rapid accumulation of good data for proper stratification of patients for risk assessment and treatment options.

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B Dragosics
Gesundheitszentrum, Vienna, Austria

A Morgen
Medical Department 1, Technical University Hospital, Dresden, Germany

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**Table 1 Paris staging system for primary gastrointestinal lymphomas**

<table>
<thead>
<tr>
<th>TX</th>
<th>Lymphoma extent not specified</th>
</tr>
</thead>
<tbody>
<tr>
<td>TO</td>
<td>No evidence of lymphoma</td>
</tr>
<tr>
<td>T1</td>
<td>Lymphoma confined to the mucosa/submucosa</td>
</tr>
<tr>
<td>T1m</td>
<td>Lymphoma confined to mucosa</td>
</tr>
<tr>
<td>T2</td>
<td>Lymphoma confined to submucosa</td>
</tr>
<tr>
<td>T2m</td>
<td>Lymphoma infiltrates muscularis propria or subserosa</td>
</tr>
<tr>
<td>T3</td>
<td>Lymphoma penetrates serosa [visceral peritoneum] without invasion of adjacent structures</td>
</tr>
<tr>
<td>T4</td>
<td>Lymphoma invades adjacent structures or organs</td>
</tr>
<tr>
<td>NX</td>
<td>Involvement of lymph nodes not assessed</td>
</tr>
<tr>
<td>NO</td>
<td>No evidence of lymph node involvement</td>
</tr>
<tr>
<td>N1</td>
<td>Involvement of regional lymph nodes</td>
</tr>
<tr>
<td>N2</td>
<td>Involvement of intra-abdominal lymph nodes beyond the regional area</td>
</tr>
<tr>
<td>N3</td>
<td>Spread to extra-abdominal lymph nodes</td>
</tr>
<tr>
<td>MX</td>
<td>Dissemination of lymphoma not assessed</td>
</tr>
<tr>
<td>MO</td>
<td>No evidence of extranodal dissemination</td>
</tr>
<tr>
<td>M1</td>
<td>Non-continuous involvement of separate site in gastrointestinal tract [eg, stomach and rectum]</td>
</tr>
<tr>
<td>M2</td>
<td>Non-continuous involvement of other tissues [eg, peritoneum, pleura] in organs [eg, tonsils, parotid gland, ocular adnexa, lung, liver, spleen, kidney, breast etc.]</td>
</tr>
</tbody>
</table>

**TX** Clinical staging: status of tumour, node, metastasis, bone marrow

**TX** Histopathological staging: status of tumour, node, metastasis, bone marrow

**TX** pTNM: Clinical staging: status of tumour, node, metastasis, bone marrow

**TX** pN: Histopathological staging: status of tumour, node, metastasis, bone marrow

**TX** Clinical staging: status of tumour, node, metastasis, bone marrow

**TX** Histopathological staging: status of tumour, node, metastasis, bone marrow

**TX** The histological examination will ordinarily include 6 or more lymph nodes.

*Valid for lymphomas originating from the gastro-oesophageal junction to the anus (as defined by identical histomorphological structure)*

†In case of more than one visible lesion synchronously originating in the gastrointestinal tract, give the characteristics of the more advanced lesion.

‡Anatomical designation of lymph nodes as “regional” according to site:

(a) stomach: perigastric nodes and those located along the ramifications of the coeliac artery (that is, left gastric artery, common hepatic artery, splenic artery) in accordance with compartments I and II of the Japanese Staging of Regional Lymph nodes (1995).

(b) duodenum: pancreaticoduodenal, pyloric, hepatic, and superior mesenteric nodes;

(c) jejunum/ileum: mesenteric nodes and, for the terminal ileum only, the ileocolic as well as the posterior cecal nodes;

(d) colorectum: pericolic and perirectal nodes and those located along the ileocolic, right, middle, and left colic, inferior mesentric, superior rectal, and internal iliac arteries.

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**Medical Department 1, Technical University Hospital, Dresden, Germany**
A new case for CA19.9 elevation: heavy tea consumption

Serum carbohydrate associated antigen (CA19.9) is a reliable tumour marker of biliopancreatic malignancies. A number of benign diseases are also known to be related to CA19.9 elevation. Here we report a case of markedly raised levels of CA19.9 associated with heavy tea consumption.

Case report

A 52 year old woman was referred to our unit for epigastric pain and anorexia of two months’ duration. She also complained of nausea and a 2 kg weight loss. Her past medical history was unremarkable. She was a non-smoker and denied alcohol abuse or medical history was unremarkable. She was a non-smoker and denied alcohol abuse or medic consumption could not be excluded. In addition, the epithelial tissue target involved in CA19.9 secretion by tea overuse is unknown. Individual susceptibility to abnormal CA19.9 secretion triggered by tea overconsumption could not be excluded.

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References


Table 1 Non-malignant causes of CA19.9 elevation (medline research)

<table>
<thead>
<tr>
<th>Aetiology</th>
<th>Mild elevation (&lt;200 U/ml)</th>
<th>High levels (&gt;1000 U/ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Obstructive jaundice</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Acute liver failure and acute hepatitis</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Chronic liver disease</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Alcoholic liver disease</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Non-alcoholic liver disease</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Pancreatitis</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Acute</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Chronic</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Intestinal pulmonary disease</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Collagen vascular diseases</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>

Anecdotally reports: hydrenephrosis, endometriosis, splenic cyst, cholangenic cyst, sigmoid diverticulitis, and hypothyroidism.

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D de Jong
The Netherlands Cancer Institute, Amsterdam, the Netherlands, on behalf of the European Gastro-Intestinal Lymphoma Study Group (EGILS)

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References


9 Rahatiner A, D’Amore F, Coiffier B, et al. A rechallenge test was then attempted. The patient restarted tea consumption as previously. Four weeks later, CA19.9 increased to 745 U/ml followed by a fall to 25 U/ml one month after withdrawal. Follow up one year later revealed no clinical abnormalities. Abdominal and chest computed tomography scan were normal.

Discussion

CA 19.9 (sialyl Lewis-a) is a monosialoganglioside with small increases in a number of benign diseases, with highest levels in pancreatic adenocarcinoma, hepatocellular, and cholangiocellular cancer, and also in gastric, colorectal, and occasionally other cancers. Physiologically elevated concentrations are present in many secretions of healthy individuals with the Lewis a positive phenotype in contrast to low serum levels of CA19.9 in Lewis a negative individuals (7–10%). Among non-malignant causes, obstructive jaundice is frequently associated with increases in CA19.9. Relief of jaundice is often associated with a fall in CA19.9 in benign cases and mostly in patients with malignancy. Normal biliary epithelial cells secrete mucins carrying the epitope of CA 19.9. Unspecific elevation of CA 19.9 serum reflects both inflammatory hypersecretion and leakage of biliary mucins into serum.

In addition, there is a strong correlation between serum CA19.9 concentration and standard parameters of cholestasis; alkaline phosphatase and bilirubin during acute liver failure, acute hepatitis, and chronic liver diseases of any aetiology.

Other benign causes of CA19.9 increases are shown in table 1. The common underlying mechanism for each is probably inflammatory hypersecretion of CA19.9 by normal epithelial cells.

The constituent flavonoids of tea beverage are known to be potent antioxidants. It appears that the flavonoids in tea can inhibit a wide range of molecular targets that influence cell growth and pathways of angiogenesis.

In this case, the usual causes of CA19.9 elevation were ruled out before a link with the tea beverage was suspected. After tea consumption withdrawal, a dramatic improvement occurred and the patient became asymptomatic, suggesting the diagnosis of tea intoxication. At the same time, serum CA19.9 levels dropped and a positive rechallenge test proved the relationship between tea overconsumption and raised levels of CA19.9.

To our knowledge, this is the first reported case of markedly raised levels of CA19.9 associated with heavy tea consumption. The mechanism of this relation remains unclear. In addition, the epithelial tissue target involved in CA19.9 secretion by tea overdose is unknown. Individual susceptibility to abnormal CA19.9 secretion triggered by tea overconsumption could not be excluded.
Non-alcoholic steatohepatitis and hepatic steatosis in patients with adult onset growth hormone deficiency

Adult onset growth hormone (GH) deficiency closely resembles syndrome X. Patients with syndrome X often suffer from obesity, dyslipidemia, insulin resistance, and hypertension, and hepatic steatosis and non-alcoholic steatohepatitis (NASH) have also been characterised as symptoms of syndrome X. However, the relationship of GH deficiency to hepatic steatosis and NASH remains unclear.

We looked for the presence of hepatic steatosis using computer tomography (CT) in a study of 18 patients with adult onset anterior lobe pituitary hormone deficiency, with or without GH deficiency. We also performed a liver biopsy in one patient with adult onset GH deficiency. None of the patients was positive for hepatitis B surface antigen, hepatitis C virus antibody, antinuclear antibody, or antimitochondrial antibody. The criterion used for diagnosing hepatic steatosis was a ratio for the liver/spleen CT value of less than 0.9. In 15 patients with GH deficiency, seven showed hepatic steatosis (53.8%) while in five patients without GH deficiency no hepatic steatosis was present (p=0.035). Body mass index (BMI), TG, and T-ch were analysed using an unpaired Student’s t-test. BMI, TG, and T-ch were analysed using an unpaired Student’s t-test. The male patient on whom we performed a biopsy was diagnosed with hepatic steatosis at 58 years old. He showed hepatic steatosis, measured by CT, at 64 years. Serum aspartate aminotransferase level fluctuated from 20 to 80 U/l. The patient did not have a history of alcohol abuse but was slightly obese (BMI 30 kg/m²). Liver biopsy was performed when the patient was 72 years old, and this showed the presence of NASH (fig 1).

We have demonstrated that hepatic steatosis is more frequently observed in patients with GH deficiency than in those without GH deficiency. Furthermore, by liver biopsy, we have shown that a patient with GH deficiency also had NASH. These results indicate that adult onset GH deficiency is a possible risk factor for hepatic steatosis and NASH.

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References


Expression of thiopurine methyltransferase in South Asians

Azathioprine and its metabolite 6-mercaptopurine (6-MP) are widely used in the treatment of Crohn’s disease, ulcerative colitis, autoimmune hepatitis, and a range of other inflammatory and autoimmune diseases as well as for prevention of transplant rejection. Thiopurine methyltransferase (TPMT) converts 6-MP to 6-mercaptopurine (6-MP) and hypoxanthine-guanine phosphoribosyltransferase (HGPRT) enzyme and its expression predisposes treated patients to bone marrow depression; it may also enhance the efficacy of thiopurines by increasing their metabolism to 6-thioguanine nucleotides. 1 Previous reports in Caucasians have shown that approximately 0.3% are homozygously and 10% heterozygously deficient in TPMT. 2 One study

Table 1

<table>
<thead>
<tr>
<th>Without GH deficiency</th>
<th>With GH deficiency</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hepatic steatosis (+/-)</td>
<td>0/5</td>
<td>7/6</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>21.29 (2.1)</td>
<td>23.54 (2.21)</td>
</tr>
<tr>
<td>TG (mg/dl)</td>
<td>135.8 (25)</td>
<td>95.4 (10)</td>
</tr>
<tr>
<td>T-ch (mg/dl)</td>
<td>202.5 (18)</td>
<td>180 (6.0)</td>
</tr>
</tbody>
</table>

Values are mean (SD). BMI, body mass index; TG, serum triglyceride level; T-ch, serum total cholesterol.
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Mutant K-ras2 in serum

Ryan and colleagues’ careful and well conducted study (Gut 2003;52:101–8) raises once again the interesting issue of whether molecular analysis and knowledge of mutations of the Kirsten ras gene in particular, have a role in the management of patients with colorectal cancer.

The two RASCAL (Kirsten Ras in Colorectal Cancer Collaborative group) studies1 2 which eventually enrolled data from 4266 patients from 42 centres in 21 countries showed that although the frequency of Kirsten ras mutations at codons 12 and 13 may vary a little between populations, overall they are present in just over one third of patients. This is significantly less frequent than quoted by Ryan et al. In addition, the RASCAL study also showed that Kirsten ras mutations are not associated with sex, age, tumour site, or Dukes’ stage.

Much more importantly, however, they also showed that of the 12 different possible specific point mutations at codons 12 or 13, only one guanine to thymidine mutation, which are found in less than 10% of all patients, is an independent variable for relapse and death. Indeed, the most common mutation, guanine to adenine, exerted an effect on survival barely different from wild-type ras. Others have shown there is a reasonable biological basis for these findings and work.

As a collaborative group, therefore, we felt it was generally unhelpful to consider Kirsten ras mutations collectively, rather than the effect of each individual mutational separately when considering the prognosis of patients with colorectal cancer. With this in mind, we believe that two crucial questions are left when considering the prognosis of patients with colorectal cancer.

1. How do we determine which Kirsten ras gene will play a role in disease progression?

2. How do we assess the clinical significance of the Kirsten ras mutations in the management of patients with colorectal cancer?

As Kirsten ras mutations are found in certain types of patients, such as those with right sided tumours,1 2 3 the non-invasive method of detecting ras mutations in serum may be helpful in determining which patients will benefit from adjuvant therapy. In our experience of over 100 patients who have undergone surgery at the Royal Marsden hospital, the mutation was present in just over one third of patients. Therefore, with the RASCAL results that Kirsten ras mutations are not associated with sex, age, tumour site, or Dukes’ stage, we believe that the frequency of mutations will not differ between populations. However, further studies are required in other centres to confirm this hypothesis.

References


Fatigue is associated with high circulating leptin levels in chronic hepatitis C

We read with great interest the recent paper from Piche and colleagues (Gut 2002;51:434–9) relating serum leptin levels to fatigue in chronic hepatitis C patients. On the surface, their findings are very intriguing, they may have significant limitations. Some of us have argued previously that there is only one way forward.1 All bodies funding prospective therapeutic studies in colorectal cancer should insist that a panel of molecular markers are measured and recorded in all patients using a standardised technique, even if they are not included as an end point, and make these results readily available for subsequent analysis. Studies which are performed prospectively carry the least risk of methodological flaws.

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PostScript 915

Downloaded from http://gut.bmj.com/ on April 10, 2017 - Published by group.bmj.com
Radioimaging Imaging of the Small Intestine


Springer Verlag are producing an extensive range of Medical Radiology books under the general editorship of Professor AL Baert, and this volume on the small intestine is the latest in the line. Professor NC Gourtsoyiannis is its distinguished editor. This is now a very well established series with topics which vary from organ based (that is, pancreas) to technique based (that is, spiral computed tomography (CT)) of the abdomen. As with all Springer publications, the illustrations are outstanding and the more than 800 in this book are no exception.

Imaging of the small intestine remains the last redoubt for the gastrointestinal radiologist due to the remorseless advance of endoscopy from both ends of the alimentary tract; a point noted in the foreword by Professor Baert. Virtual colonoscopy has redeemed some interest but this is strictly a polyp/tumour detection exercise and the more subtle and interesting colonic abnormalities elude it. Study of the small bowel however retains that interest, both because of the myriad of processes which may affect it as well as the fact that it is not so easy to initiate the “knee jerk” response of biopsy it. Therefore remains more of a deductive radiological approach in colorectal cancer (editorial) for any radiologist with an interest in the gastrointestinal system.

A H Freeman

NOTICES

New In Vivo Imaging Modalities for Molecular Biology, Cell Biology and Physiology

This Jacques Monod conference will be held on 31 May–4 June 2003 in Roscoff, France. Further information: Bertrand Tavitian, IN- SERM M10103, Service Hospitalier Frédéric Joliot, CEA Direction des Sciences du Vivant, Direction de la Recherche Médicale, 4 place du Génral Leclerc, 91401 Orsay Cedex, France. Tel: +33 1 696 8779; fax: +33 1 696 8739; email: tavitian@ihl.cea.fr

Prague Hepatology Meeting

To be held on 5–7 June 2003 in Prague, Czech Republic. Leading speakers from Europe and the USA will present new ideas and suggestions on pathophysiology, diagnostics, and therapy of liver diseases in ten programmes. Further details: Mrs Veronika Revicka. Tel: +420 241 445 759; fax: +420 241 445 806; email: veronika@congressprague.cz

8th International Liver Symposium

This symposium will be held on 13 June 2003 in Manchester, UK. Leading speakers from the USA and Europe will present state of the art reviews on recent advances in hepatology. Further details: Professor TW Warnes, Department of Gastroenterology, Manchester Royal Infirmary, Oxford Road, Manchester M13 9WL, UK. Tel: +44 (0)161 276 4316; fax: +44 (0)161 276 8779; email: judith.harrop@cmmc.nhs.uk

Falk Symposium—New Findings on Pathogenesis and Progress in Management of IBD

Two symposia and a workshop will be held on 10–14 June 2003 in Berlin, Germany. Further details: Falk Foundation e.V., Congress Divi- sion, PO Box 629, Lievenweberstr. 5, 79041 Freiburg/Br, Germany. Tel: +49 761 15 140; fax: +49 761 15 14 359; email: falksymposia@falkfoundation.de; website: www.falkfoundation.de

Gastroenterology and Endotherapy: XX1st European Workshop

This will be held on 16–18 June 2003 in Brussels, Belgium. Further details: Nancy Beaupré, Administrative Secretariat of the Work- shop, Gastroenterology Department, Erasme Hospital, Route de Lennik 808, B-1070 Brussels, Belgium. Tel: +32 (0)2 555 49 00; fax: +32 (0)2 555 49 01; email: beaupre@ulb.ac.be

The Association of Coloproctology of Great Britain & Ireland

This annual meeting will be held on 7–10 July 2003 in Edinburgh, UK. Further details: Conference Secretariat, The ACGBI at the Royal College of Surgeons of England, 35–43 Lincoln’s Inn Fields, London WC2A 3PE. Tel: +44 (0)20 7973 0307; fax: +44 (0)20 7430 9235; email: acgb@asgbi.org.uk; website: www.acgbi.org.uk

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, Solvegatan 23, SE-223 62 Lund, Sweden. Tel: +46 46 173 241; fax: +46 46 152 564; email: torkel.wadstrom@mmibi.lu.se; website: www.helicobacter.org
Non-alcoholic steatohepatitis and hepatic steatosis in patients with adult onset growth hormone deficiency
T Ichikawa, K Hamasaki, H Ishikawa, E Ejima, K Eguchi and K Nakao

Gut 2003 52: 914
doi: 10.1136/gut.52.6.914

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