Paris staging system for primary gastrointestinal lymphomas

Currently, much effort is put into the development of specific therapeutical 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 specialised documentation of tumour extent in the organ wall has been stressed by the high predictive value for failure of response after *Helicobacter pylori* eradication of infiltration beyond the mucosa, a predictive parameter irrespective of the presence of the t(11;18) translocation.‡ With new techniques such as endosonography, this infiltration depth can be assessed with improving accuracy. However, the information cannot be translated into the current staging systems. The dissemination patterns of extranodal lymphomas are also essentially different from primary nodal lymphomas.‡

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 I, (confined to the mucosa and submucosa) from stage I, (tumour extension beyond the submucosa according to Radoszkiewicz and colleagues’) nor discrimination of stages I, (nodal involvement beyond the regional area, as assessed in the Musshoff modification) is sufficiently serving as a collaborative effort. As a result of discussions on staging protocols and reporting systems over the past years, we would like to propose 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 nodal involvement; as well as (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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Table 1 Paris staging system for primary gastrointestinal lymphomas*†

<table>
<thead>
<tr>
<th>TNM</th>
<th>pT/NMB</th>
<th>pN</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical staging: status of tumour, node, metastasis, bone marrow</td>
<td>Histopathological staging: status of tumour, node, metastasis, bone marrow</td>
<td>The histological examination will ordinarily include 6 or more lymph nodes</td>
</tr>
<tr>
<td>B1</td>
<td>Lymphomatous infiltration of bone marrow</td>
<td></td>
</tr>
<tr>
<td>B0</td>
<td>No evidence of bone marrow involvement</td>
<td></td>
</tr>
<tr>
<td>BX</td>
<td>Involvement of bone marrow not assessed</td>
<td></td>
</tr>
<tr>
<td>NO</td>
<td>No evidence of lymph node involvement</td>
<td></td>
</tr>
<tr>
<td>NX</td>
<td>Involvement of lymph nodes not assessed</td>
<td></td>
</tr>
<tr>
<td>NO</td>
<td>No evidence of lymph node involvement</td>
<td></td>
</tr>
<tr>
<td>N1</td>
<td>Involvement of regional lymph nodes</td>
<td></td>
</tr>
<tr>
<td>N2</td>
<td>Involvement of intra-abdominal lymph nodes beyond the regional area</td>
<td></td>
</tr>
<tr>
<td>N3</td>
<td>Spread to extra-abdominal lymph nodes</td>
<td></td>
</tr>
<tr>
<td>MX</td>
<td>Dissemination of lymphoma not assessed</td>
<td></td>
</tr>
<tr>
<td>MO</td>
<td>No evidence of extranodal dissemination</td>
<td></td>
</tr>
<tr>
<td>M1</td>
<td>Non-continuous involvement of separate site in gastrointestinal tract (eg, stomach and rectum)</td>
<td></td>
</tr>
<tr>
<td>M2</td>
<td>Non-continuous involvement of other tissues (eg, peritoneum, pleura) or organs (eg, tonsils, parotid gland, ocular adnexa, lung, liver, spleen, kidney, breast etc.)</td>
<td></td>
</tr>
</tbody>
</table>

*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 identical histomorphological structure.

‡Anatomical designation of lymph nodes as "regional" according to site: (a) stomach: parigastric 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 Research Society for Gastric Cancer (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 coecal nodes; (d) colorectum: pericolic and perirectal nodes and those located along the ileocolic, right, middle, and left colic, inferior mesenteric, superior rectal, and internal iliac arteries.
A new cause 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 medication intake but remarked on overconsumption of warm black tea for several months (1.5–2 litres/day). Physical examination was normal.

Laboratory data demonstrated normal ranges for electrolytes, liver function tests, blood count, urea, creatinin, C reactive protein, amylase, and lipase. Fasting blood glucose, triglycerides, cholesterol, and thyroid function tests were normal. Autoantibodies were negative. Serum CA19.9 was 1432 U/ml (normal <37), and CEA was 2 ng/ml (normal <5).

Upper endoscopy, colonoscopy, and barium study of the small bowel showed normal results. Abdominal ultrasonography and computed tomography scan were normal.

Four weeks later she became jaundiced, with heavy tea consumption. Four weeks later CA19.9 increased to 1432 U/ml (normal <37), and CEA was 2 ng/ml (normal <5).

Anecdotally reports: hydronephrosis,1 endometriosis,14 splenic cyst,16 bronchogenic cyst,16 sigmoid diverticulitis,16 and hypothyroidism.16

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

<table>
<thead>
<tr>
<th>Aetiology</th>
<th>Serum CA19.9 level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild elevation (&lt;200 U/ml)</td>
<td>High levels (&gt;1000 U/ml)</td>
</tr>
<tr>
<td>Obstructive jaundice</td>
<td>+</td>
</tr>
<tr>
<td>Acute liver failure and acute hepatitis</td>
<td>+</td>
</tr>
<tr>
<td>Chronic liver disease</td>
<td>+</td>
</tr>
<tr>
<td>Alcoholic liver disease</td>
<td>+</td>
</tr>
<tr>
<td>Non-alcoholic liver disease</td>
<td>+</td>
</tr>
<tr>
<td>Pancreatitis</td>
<td>+</td>
</tr>
<tr>
<td>Acute</td>
<td>+</td>
</tr>
<tr>
<td>Chronic</td>
<td>+</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>+</td>
</tr>
<tr>
<td>Intestinal pulmonary disease</td>
<td>+</td>
</tr>
<tr>
<td>Collagen vascular diseases</td>
<td>+</td>
</tr>
</tbody>
</table>

Our patient was advised to stop tea consumption.

Discussion
CA 19.9 (sialyl lew-is-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.1

Physiologically elevated concentrations are present in many secretions of healthy individuals with the Lewis a positive phenotype in contrast with low serum levels of CA19.9 in Lewis a negative individuals (7–10%).1

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.2 Normal biliary epithelial cells secrete mucins carrying the epitope of CA 19.9. Unspecific elevation of CA 19.9 in serum reflects both inflammatory hypersecretion and leakage of biliary mucins into serum.3

In addition, there is a strong correlation between serum CA19.9 concentration and standard parameters of cholastasis: alkaline phosphatase and bilirubin during acute liver failure, acute hepatitis, and chronic liver diseases of any aetiology.4

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 associated with a wide range of molecular targets that influence cell growth and pathways of angiogenesis.5

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 symptom free, 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 overuse is unknown. Individual susceptibility to abnormal CA19.9 secretion triggered by tea overconsumption could not be excluded.

References

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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.1 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).

Table 1

<table>
<thead>
<tr>
<th>Deficiency</th>
<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>
<td>0.035</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>21.29 (1.2)</td>
<td>23.54 (2.21)</td>
<td>0.35</td>
</tr>
<tr>
<td>TG (mg/dl)</td>
<td>135.8 (25)</td>
<td>95.4 (10)</td>
<td>0.30</td>
</tr>
<tr>
<td>T-ch (mg/dl)</td>
<td>202.5 (18)</td>
<td>180 (6.0)</td>
<td>0.44</td>
</tr>
</tbody>
</table>

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

Figure 1: Histology of the liver biopsy. Macrovesicular steatosis, pericellular and centrilobular fibrosis, and ballooned hepatocytes are all observed in this section. (Method: needle biopsy; haematoxylin-eosin staining.)

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) by dehydrogenation of its expression. Overexpression of TPMT 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 homozygous and 10% heterozygous deficient in TPMT.1 One study
suggests that the frequency of individuals with the mutant TPMT allele, TPMT*3A, is lower in Southwest Asians than in Caucasian; the mutant allele causes reduced enzyme activity in vitro. 1 However, there are no data on the phenotypic expression of TPMT in South Asian populations.

We have compared expression of TPMT in South Asian and Caucasian patients attending gastroenterology, renal, rheumatology, and dermatology clinics who were being considered for treatment with thiopurines. TPMT activity was assayed by tandem mass spectrometry for 6-methylMP (assays kindly provided by Purine Research Laboratory, Guy's Hospital, London SE1 9RT, UK). Homozygous deficiency of TPMT was defined as a level <10, heterozygous as 10–25, and normal as >25 pmol/h/mg haemoglobin. Of 83 Caucasian patients, one (1.3%) was homozgyous deficient, 10 (12%) were heterozygous, and 72 (86.7%) had normal expression of TPMT. The corresponding values in 77 South Asian patients were 0.7 (9%), and 70 (91%), respectively.

The prevalence of deficiency of expression of TPMT in South Asian patients resembles that in Caucasians. Use and monitoring of azathioprine and 6-MP should therefore follow similar principles in both ethnic groups.

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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 leptin levels to fatigue in chronic hepatitis C patients. On the surface, their observation that serum leptin levels correlate with the fatigue impact scale are very intriguing. However, leptin shows a gender based difference and women have circulating plasma leptin concentrations that are at least three times higher than men, even when matched for body fat mass. 1 Thus sex needs to be considered when investigating any association with leptin levels, and multivariate analysis is necessary to reduce the bias caused by this confounding variable. Indeed, in the study by Piche et al the fatigue impact scale was found to be significantly higher in females. However, without a multivariate analysis the authors cannot affirm that serum leptin levels are implicated in fatigue development in patients with chronic hepatitis C.

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Reference

Mutant K-ras 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) studies 1-3 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 a 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. However, as a collaborative group, therefore, we felt it was generally unhelpful to consider Kirsten ras mutations collectively, rather than the effect of each individual mutation separately when considering the prognosis of patients with colorectal cancer. With this in mind, we believe that two crucial questions are left unanswered by the study of Ryan et al. Without definitive answers to these, it is unlikely that serum detection of mutated Kirsten ras gene will ever be clinically useful.

Firstly, the effect of any new mutation in the serum of a patient who has had colorectal cancer raises the possibility of tumour recurrence, although the presence of a Kirsten ras mutation is not a colonic specific. However, more than one third of patients in Ryan’s study did not clinically relapse despite the presence of a serum mutation for very long periods. Others have also detected tumour DNA in patients for long periods without evidence of overt clinical recurrence. Therefore, screening for the presence of Kirsten ras mutations per se is unlikely to be clinically helpful very often. However, it would have been very interesting to know whether the patients who did not relapse in this study were those who had the RASCAL group defined as “benign” rather than “aggressive” mutations.

Secondly, the authors do not say whether each postoperative serum mutation which they detected corresponded with the genotype of the primary tumour. While a number of studies have suggested that occasional metastases may carry a different mutation to the primary, this is very uncommon. If the mutation is not the same as the primary tumour, this raises many issues, not least that despite the very careful use of controls, nested polymerase chain reaction techniques may be too sensitive for clinical practice.

For an individual with colorectal cancer, a multitude of factors may shape the clinical course. To improve our knowledge, we must seek to study the most subtle variation in molecular profiles possible, as small differences may be very important. While rigorous small prognostic clinical studies may be intriguing, they may have significant limitations. Some of us have argued previously that there is only one way forward: 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 do not carry the least risk of methodological flaws.

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R Radiological 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 organ based (that is, small bowel) tumours which reflect technique for or those which reflect pathological conditions. In this case the editor has gone for both and while this results in a superlative volume, there is bound to be some repetition. For example, reference to Crohn’s disease or small bowel tumours will appear both in their own section as well as under CT/MR, etc. However, as all of these chapters are written by different authors with their own experiences, this can prove to be an advantage enabling the reader a “second bite of the cherry”. Is there competition in the market place and how does this book hold up? The inevitable comparison has to be made to Clinical Imaging of the Small Intestine by Heilig, Maglinte, and Birnbaum, interestingly also published by Springer. This is the more mature book, now in its second edition, having been originally published in 1989. Its authorship is primarily North American with two European contributors whereas Radiological Imaging is primarily a European work with some distinguished North American contributors. Indeed, two of the editors of Clinical Imaging are authors. Both books go for the same approach in colorectal cancer (editorial). J Clin Oncol 2001; 19: 286–8.

BOOK REVIEW

Radiological Imaging of the Small Intestine

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References

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 Tatibouët, IN-SERM M10103, Service Hospitalier Frédéric Joliot, CEA Direction des Sciences du Vivant, Direction de la Recherche Médicale, 4 place du Général Leclerc, 91401 Orsay Cedex, France. Tel: +33 169 867 779; fax: +33 169 867 739; email: tatibouet@shlj.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 suggesions on pathophysiology, diagnostics, and therapy of liver diseases in ten programmes. Further details: Ms Veronika Revicka. Tel: +420 241 445 759; fax: +420 241 445 806; email: veronika@congressprague.cz

Falk Symposia—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 Division, PO Box 6529, 15704 Berlin, Germany. Tel: +49 30 684 15 140; fax: +49 30 684 15 14 359; email: symposia@falkfoundation.de; website: www.falkfoundation.de

Gastroenterology and Endotherapy: XXIst European Workshop

This will be held on 16–18 June 2003 in Brussels, Belgium. Further details: Nancy Beaufrez, Administrative Secretariat of the Workshop, 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: beaufrez@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 ACCGIB 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-directory@asgb.org.uk; website: www.acgb.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 Wadström, President- EHSG, Lund University, Department of Infectious Diseases & Medical Microbiology, Division of Bacteriology, Solvagatan 23, SE-223 62 Lund, Sweden. Tel: +46 46 173 241; fax: +46 46 152 364; email: torkel.wadstrom@mmub.lu.se; website: www.helicobacter.org
Fatigue is associated with high circulating leptin levels in chronic hepatitis C

M Romero-Gómez, D Sánchez-Muñoz and M Cruz

Gut 2003 52: 915
doi: 10.1136/gut.52.6.915

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